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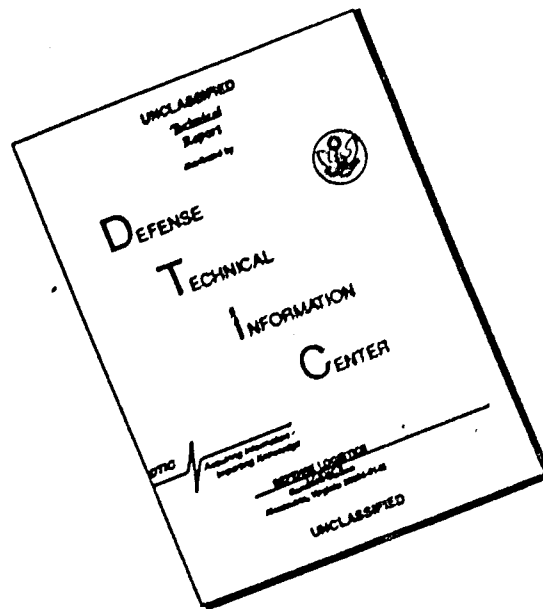
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TECHNICAL REPORT

PROCEEDINGS OF THIRD ANNUAL
TRI-SERVICE CONFERENCE ON
BIOLOGICAL EFFECTS OF MICRO-
WAVE RADIATING EQUIPMENTS

25, 26, 27 AUGUST 1959

Rome Air Development Center
Air Research & Development Command
United States Air Force
Griffiss Air Force Base, New York

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Compiled by
Dr. Charles Susskind, University of California

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P R E F A C E

Annual Tri-Service Conferences were initiated in 1957 by the Rome Air Development Center as the means for annually reporting to the military services in fulfillment of tri-service responsibility for determining the biological effects of microwave radiation. The 1957 conference proceedings were published in ASTIA Document No. AD 115 603; the 1958 conference proceedings in ASTIA Document No. AD 131 477.

These conferences bring together key researchers in the biological effects area and related scientific disciplines with representatives of industry, universities, and various government agencies to discuss and exchange knowledge made available by rapid advances during the past year. In furthering these objectives, unclassified papers were solicited. The papers covered the conference subjects or related areas which might contribute to a better understanding of the biological effects of microwave energy.

At the invitation of the University of California, the third annual conference was held on the University campus in Berkeley under the joint sponsorship of the Rome Air Development Center and the Tri-Service Ad Hoc Committee on the Biological Effects of Microwave Energy, Colonel George M. Knauf, Chairman.

The Tri-Service Ad Hoc Committee was formed in July 1958 by mutual agreement between representative of the U. S. Army Medical Research Laboratory, Office of the Surgeon General, U. S. Army, and the U. S. Army Environmental Health Laboratory; the Bureau of Medicine and Surgery and the Office of Naval Research, U. S. Navy; the Office of the Surgeon General, the Air Research and Development Command, Office of Scientific Research and the Rome Air Development Center of the U. S. Air Force.

Members representing each of the referenced activities meet twice each year to review the tri-service biological programs conducted by the Rome Air Development Center to insure that requirements of their individual services are adequately covered.

The Committee is supported by a group of consultants in Physics, Physiology, Ophthalmology, Microwave Engineering, Biophysics and Pathology, whose functions are to critically analyze the experimental results of the program and provide the Rome Air Development Center with recommendations for insuring the validity of developed data and sound approaches to meeting the more novel requirements of the military services.

The sponsoring activities wish to express appreciation to Professor Charles Susskind and to the staff of the University of California for their hospitality and skillful handling of conference arrangements; to the industrial and educational organizations represented on the program who gave so freely of their time and talents; and to the representatives of the Army, Navy, Air Force and of the National Institute of Neurological Diseases and Blindness whose cooperation made possible the success of this conference.

HERBERT S. BROWNSTEIN (RADC)
Secretariat
Tri-Service Ad Hoc Committee on
Biological Effects of Microwave
Energy

BIOLOGICAL EFFECTS OF MICROWAVE ENERGY
AT 200 MEGACYCLES UPON THE
EYES OF SELECTED MAMMALS

Drs. C.H. Addington, C. Osborn, G. Swartz, F. Fischer, and Y. Sarkees
Departments of Biology and Electrical Engineering
The University of Buffalo

SUMMARY

No ocular changes have been noted in guinea pigs, dogs, sheep and mice when exposed to microwave energy at 200 megacycles. However, the Horn antenna, yielding polarizing radiation, is producing much greater temperature ranges and field intensities than the previously employed helical antenna.

INTRODUCTION

The present report deals with the results of experiments conducted since our last meeting in January of this year. You will recall that we are working in the 200 megacycle band. Originally we had at our disposal a low power pulsed type apparatus giving peak emanations of 0.04 watt/cm^2 , with an average level of about $1/250$ th of this power. In these experiments mice and guinea pigs were employed and no ocular changes were found. We were particularly interested in the possibility of delayed damage for reasons presented at the previous conference. One guinea pig which had logged the longest exposure record for some 27,000 minutes at 4 ft. from the antenna, still does not show any ocular changes, other than aging, one year later.

In January we reported results of experiments conducted with a more powerful CW transmitter employing a Helical type antenna. A number of guinea pigs, a large dog, and a sheep were exposed in acute and chronic experiments at various power densities. None of these animals exhibited any ocular changes other than those associated with aging.

RECENT EXPERIMENTS

Experiments with Helical Antenna

Guinea Pigs. A group of 7 guinea pigs, averaging 1 lb. 12 oz., were exposed at distances of 5 ft., and 3 ft. facing the antenna. The field strengths at these positions were 220 and 350 mw/cm^2 respectively. Exposure time was 60 min/day at 3, 5 and 7 days per week. The total exposure time is indicated in the table as well as the average temperature elevation. The longest experiment has run some 45 weeks for a total of 332 hours. The average elevation of rectal temperature was less than 2° F. for the entire group. None of these animals have so far shown any ocular changes (TABLE I).

Dog and Sheep. The dog and sheep (TABLE II) originally reported

TABLE I - GUINEA PIGS* - CHRONIC EXPOSURES (200 MEGACYCLES-CW) HELICAL ANTENNA (NON-POLARIZED) - FACING

| Fig | Distance | Field mw/cm ² | Exposure | Total Exposure | Av. Temp. El. |
|-------------|----------|--------------------------|------------------|-------------------|---------------|
| 1. T. & W | 5' | 220 | 60 min. 3 day/wk | 55 hr. - 12 wks. | 1° F. |
| 2. Tri. cl. | 5' | 220 | 60 min 3 day/wk | 187 hr. - 42 wks. | 1° F. |
| 3. B. & W | 5' | 220 | 60 min. 3 day/wk | 181 hr. - 42 wks. | 1.2° F. |
| 4. Br & W | 5' | 220 | 60 min. everyday | 332 hr. - 45 wks. | 1.5° F. |
| 5. Br & W | 5' | 220 | 60 min. everyday | 332 hr. - 45 wks. | 1.9° F. |
| 6. Tri. cl. | 3' | 350 | 60 min. 5 day/wk | 173 hr. - 39 wks. | 2.0° F. |
| 7. T. & W | 3' | 350 | 60 min. 5 day/wk | 171 hr. - 39 wks. | 1.6° F. |

* Av. Wt. - 28 oz.

TABLE II - DOG AND SHEEP OPHTHALMOLOGICAL STUDIES (200 MEGACYCLES-CW)

| Animal | Position | Exposure Distance | Exposure mm/cm ² | Duration Hours | Duration Weeks | Rectal Temp. Av. el. | Result |
|--------------------------------------|-----------|-------------------|-----------------------------|----------------|----------------|----------------------|--------|
| <u>Chronic - Non-polarized Field</u> | | | | | | | |
| Dog 47# | Broadside | 5' | 220 | 70 | 24 | 1.6° F | Neg. |
| Sheep 55-100# | Head on | 3' | 350 | 90 | 30 | 1.1° F | Neg. |
| <u>Acute and Polarized Field</u> | | | | | | | |
| Sheep | X Axis | 5' | 165 | 40 min | single | 5.8° F | Neg. |
| Sheep | X Axis | 3-1/2' | 207 | 45 min | single | 5.6° F | Neg. |

at the last meeting, have been continued to be exposed 3 times a week at 60 and 90 minute intervals respectively. The dog has been positioned 5 ft. from the antenna in a field of 220 mw/cm². He has now logged 60 hours. The sheep, placed at 3 ft. from the antenna in a field of 350 mw/cm², has accumulated 90 hours. You will note that the original weight of this animal was 58 lbs. and that now it weighs slightly more than 100 lbs. Neither of these animals has shown any ocular changes

Mice. Several litters of mice irradiated continuously in the chamber at field intensities of 50 to 200 mw/cm², successfully passed through 4 generations. During this period the eyes were observed at regular intervals and no ocular changes other than associated with normal aging were seen.

Experiments with Horn Antenna

Recently the Helical antenna has been replaced by a Horn creating a polarized field and subsequently field intensities and temperature responses have increased a good deal.

Guinea Pigs. TABLE III is a composite of the results obtained with 7 guinea pigs in chronic and acute exposures. The first 2 animals have so far endured 19 exposures of 20 minutes each for a total of 365 minutes. They were placed in plastic cages facing the antenna (Z axis). The average elevation of rectal temperature for pig I at 2 ft. was 4.81° F., and pig II at 4 ft. was 2.43° F. The fields at these positions were 390 and 194 mw/cm². The remaining five animals were given single exposures at the various field intensities and distances as indicated. None of these animals have as yet shown any ocular changes.

TABLE IV shows a series of 3 guinea pigs of the same size and weight irradiated at different positions in the polarized field. The animals were again placed in plastic cages and either faced the antenna (Z axis) or were broad side to it (X axis). Although the temperature elevations and the field intensities were relatively high, no ocular changes have so far been observed.

Dogs. TABLE V indicates our experience to date with dogs irradiated in a polarized field. All of these animals except one, weighs 50 lbs. or more. The data on the chart indicate the distance from the antenna, intensity of field at that point, orientation of the animal in respect to the field, total exposure time, and the average elevation of the rectal temperature. Again we can find no change in the ocular system despite rather marked elevation of temperature. In all fairness one should point out that it is much more difficult to accurately examine large animals. It is impossible to use the major slit lamp. We have employed the B & L hand slit lamp powered by a transformer (not the smaller battery handle model). This instrument throws a very powerful slit beam and the lens can be observed very well with the aid of a loupe.

TABLE III - GUINEA PIGS - POLARIZED FIELD (200 MEGACYCLES-CW)

| Animal | Wt. oz. | Distance | Field mw/cm ² | Exposure Min. | Total Exp. | Av. Temp. el. |
|------------|---------|----------|--------------------------|---------------|------------|---------------|
| 1. Tri.cl. | 27 | 2' | 390 | 30 min./day | 365z* | 4.81° F. |
| 2. T. & W | 27 | 4 | 194 | 20 min./day | 365z* | 2.43° F. |
| 3. B. & W | 28 | 18" | 432 | Single | 22x | 7.0° F. |
| 7. | 25 | 33" | 305 | Single | 23x | 7.8° F. |
| 4. | 25 | 4' | 194 | Single | 20z | 5.1° F. |
| 5. Red | 25 | 7' | 105 | Single | 31z | 3.2° F. |
| 6. | 25 | 11' | 035 | Single | 50x | 1.8° F. |

* Total 19 Exposures

TABLE IV - GUINEA PIGS - POLARIZED FIELD (200 MEGACYCLES-CW)

| Animal | Wt. oz. | Distance | Field mm/cm ⁻² | Exposure Min. | Temp. Elevation |
|--------|---------|----------|---------------------------|---------------|-----------------|
| 8 | 24 | 5" | 500 | 30z | 9.7° F. |
| 2 | 24 | 5" | 500 | 20x | 7.8° F. |
| 7 | 24 | 2' | 390 | 16z | 7.4° F. |
| 6 | 24 | 2'6" | 305 | 26x | 6.1° F. |
| 5 | 24 | 2'6" | .05 | 20x | 5.1° F. |
| 1 | 24 | 3' | 220 | 28x | 7.5° F. |
| 4 | 26 | 4' " | 173 | 24x | 5.3° F. |
| 3 | 24 | 5' | 165 | 30x | 4.9° F. |

TABLE V - DOGS - POLARIZED FIELD (200 MEGACYCLES-CW)

| Animal | Wt. lbs. | Distance | Axis | Field mw/cm ² | Total Exp. | Temp. Range | Av. Temp. el. |
|--------|----------|----------|------|-----------------------------|------------|-------------|---------------|
| 1. M | 53 | 5' | 2x | 165 | 225 | 2.2-8.6 | 5.6° F. |
| | | 6' | 1x | 135 | | | |
| | | 7' | 2y | 105 | | | |
| | | 18' | 1y | 9 | | | |
| 3. M | 50 | 7' | 3x | 105 | 215 | 2.5-5.7 | 4.4° F. |
| | | 7' | 1y | 105 | | | |
| | | 10' | 1y | 38 | | | |
| 4. M | 55 | 4' | 1x | 194 | 66 | 4.5-5.8 | 5.1° F. |
| | | 5' | 2x | 165 | | | |
| 6. M | 50 | 10' | 2xy | 38 | 55 | 5.7-7.2 | 6.5° F. |
| 5. F | 70 | 18' | 2xy | 9 | 122 | 0.0-2.0 | 1.0° F. |
| 7. F | 35 | 18' | 2xy | 9 | 120 | 0.8-1.6 | 1.2° F. |

An acute experiment was performed in which a thermocouple was placed in the posterior chamber of a dogs eye. This was done in the anesthetized animal by making an incision at the upper border of the lateral rectus muscle down through the sclera to the choroid. A mattress suture was then placed across this wound. The thermocouple was securely tied to a 4 "0" silk suture. A long slightly curved needle was then passed through the scleral wound and out the nasal side at the equator. The suture then pulled the thermocouple into position into the posterior chamber where it could be observed through the pupil. The mattress suture was then tied and the thermocouple securely anchored in place by additional sutures in the sclera. The eye tolerated this foreign body rather well for about 2 weeks but the wires protruding from the eye were too stiff and irritated the animal when manipulated. It was therefore necessary to again anesthetize the dog in order to obtain temperature readings in the chamber. The animal was placed 6 ft. 8 in. from the antenna and facing it. The intensity at this point was 115 mw/cm². In 40 minutes the rectal temperature rose 1.9° F. and the ocular temperature 1.7° F. Eventually the eye developed an endophthalmitis and glaucoma and had to be enucleated.

The animal was then run for the same length of time in the same position but not under anesthesia. The rectal temperature rise was almost twice that of the anesthetized animal. (TABLE VI).

We have also inserted thermocouples and thermistors in isolated beef eyes suspended by nylon threads and kept moist by a continuous drip bottle. No temperature rise has been noted.

TABLE VI - DOG (RODNEY): THERMO-COUPLES IN LEFT EYE - POLARIZED FIELD (200 MEGACYCLES-CW)

| | Wt. | Distance | Axis | Field mw/cm^2 | Exp. | Temp. e.l. |
|-------------------------|-----|----------|------|-------------------------------|------|-----------------------|
| <u>Anesthetized</u> | 23# | 6.8m | Z | 115 | 40 | Rectal Eye 1.9 1.7 |
| <u>Not Anesthetized</u> | 23# | 6.8m | Z | 115 | 40 | 3.7° F. |

THERMAL EFFECTS OF 200 MEGACYCLE(CW) IRRADIATION
AS RELATED TO SHAPE, LOCATION AND
ORIENTATION IN THE FIELD

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Departments of Biology and Electrical Engineering
The University of Buffalo

BACKGROUND

Workers in electromagnetic irradiation have known for some time that field intensities are increased or concentrated midway along elongated, uniformly shaped objects oriented in the plane of polarization. It is not unlikely that such an "antenna effect" may be significant in producing localized heating in man and test animals if they are illuminated critically in such a field. There is the further likelihood that parts of the body such as extremities or tubular or hollow viscera may concentrate energy in a similar or related manner.

EXPERIMENTS

Heating of Models

My colleague, Mr. Fischer has already indicated the nature of our physical setup and he has explained the rationale of some of our experiments using tubular models of various shapes filled with egg albumen as well as other natural tissues and solutions of electrolytes. Albumen proved to be an especially useful indicator because it changes visibly (coagulates to a cloudy and later white opaque) and because this response occurs at relatively low temperatures at which most tissues are seriously injured. It provides an accurate indication of where concentrated heating occurs and when a centigrate value of +65° to +70° is reached.

Accordingly, plastic tubular models were made in various shapes and sizes, filled with albumen or other related solutions and exposed to electromagnetic irradiation (200 megacycles-cw) at several field intensities and for various periods of time.

(At this point several 2" x 2" Kodachromes were projected to illustrate the results.)

Of the many observations made, the following may have relevance to problems of local heating in man exposed to microwave energy.

1. Uniformly cylindrical tubes of albumens heated (as shown by coagulation) fastest midway between the ends when exposed in the axis of polarization. Some coagulations occurred in a few seconds while others took several minutes depending on starting temperature, size and field intensity.

2. Similar models exposed at 90° to the plane of polarization did not exhibit heating.

3. Similar models exposed at intermediate angles between the two planes mentioned above heated only slightly, with the reaction increasing as the plane of polarization is approached.

4. Cylinders having an end gradually tapering to a point exhibited local heating in the tip of the taper as well as midway along the cylinder as described above.

5. Wherever tubes of different sizes were adjoined in a straight line, localized heat appeared first in the smaller tube near the junction.

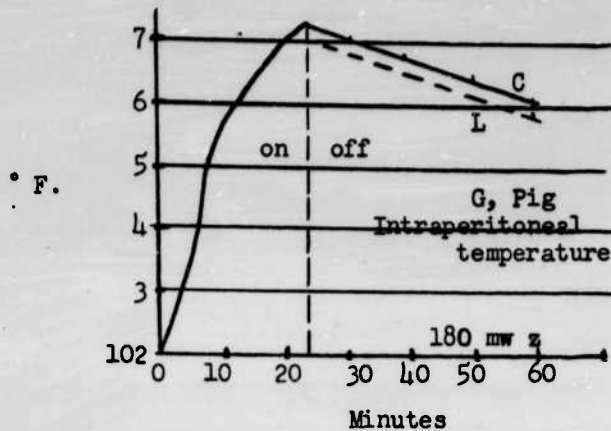
6. Where multiple tubes adjoined along radial lines to a central cylinder or sphere were irradiated, only those aligning in the plane of polarization heated disproportionately and then at the point of junction with the central mass.

The question is raised whether these observations may have important implications for local heating at the distal tips of extremities, the area of junction of the proximal end of extremities with the torso and perhaps along segments of tubular or hollow visera.

Intra-peritoneal Temperatures - localized

Data obtained with albumen models stimulated the initiation of experiments designed to give information on localization of heating in mammals. Whereupon, plastic fistulas were located by surgical means in the lateral abdominal wall of guinea pigs. Each fistula was designed with a minute screw cap which could be removed to allow insertion of pickup devices. Leads following minimum r.f. plans were run to externally located telethermometers of the Yellow Springs type.

This preliminary report includes findings in only three experiments of this type completed to date. The guinea pigs were totally exposed in the Z axis (plane of propagation) to 390, 180 and 105 milliwatts per phase (power on) which required from 12 to 28 minutes to cause a 5° F elevation. The thermister probe was inserted so that its tip was in the mid-longitudinal plane in the peritoneal cavity. In the cooling phase (power-off) the probe was alternately located in mid-line or one-half to three-quarters of an inch lateral to mid-line in order to measure the temperature with accuracy at these two locations. All possibility of error due to pickup was avoided in-as-much-as the power was off during this phase of recording. The results were so similar in the three experiments, that the curve of but one will suffice as typical for illustrative purposes.



Graph 1. Intraperitoneal Temperatures

In graph 1, solid line C indicates intraperitoneal temperatures existing in the central (mid-plane) axis of the animal. Rectal temperatures were consistently $1/2$ to 1 degree lower but otherwise the slope of the curve was similar to line C. The broken line, L, represents the intraperitoneal temperature taken in a lateral position as indicated above. Here, it is seen that the temperature is consistently at least one-half degree lower.

In themselves, these data are of considerable interest, but the technique is promising and should, with appropriate modifications, allow us to record more reliable data on local internal temperatures than have been feasible here-to-for.

Burns, Lethal Dosages etc.

As an observation which is quite incidental to the objectives of our experiments we can report two integumentary burns to date. One of these was on the lateral thorax of a 70 lb. dog which received total irradiation at 210 milliwatts for 22 minutes. By the end of this exposure the rectal temperature had elevated 8.8° F and the dog exhibited ataxia and digestive upset. The animal acted listless and lethargic and required 3 or 4 days to return to normal behavior. The subcutaneous injury did not ulcerate until the 7th day after exposure. The appearance and healing program followed closely that described by the University of Rochester investigators.

The second burns appeared on a 100 lb. sheep which had been totally exposed for 40 minutes where the incident intensity was 165 milliwatts per square cm. At the end of the exposure rectal temperature had elevated 5.8° F. No specific symptoms manifested themselves immediately although the animal showed little interest in its surroundings, was slow to respond when food was offered and ate little for almost a week. Hard lumpy masses were palpated subcutaneously 6 to 7 days after irradiation on the right side and top of the neck and on both sides of the thorax.

These hard areas ranged between the size of a dime and a half-dollar. Three spots ulcerated at 10 to 12 days and healed uneventfully thereafter. Some generalized shedding of hair became quite prominent 7 or 8 days after irradiation. Hair loss was extreme, ending in bareness in the areas coinciding closely with the subcutaneous heat injury. Some six weeks later, the hair is growing in again indicating no permanent damage to the hair follicles.

In recent months since being equipped with a horn antenna, producing a polarized field of higher uniform intensity, we have lost only four animals (one dog and three guinea pigs) from over exposure. Field strengths are sufficiently high that lethal doses for such animals could be reached in a short time (only a few minutes) if placed near the mouth of the horn where incident field strengths are approaching 500 milliwatts per cm^2 .

The dog which expired has been exposed to about 200 milliwatts/ cm^2 for 31 minutes. Rectal temperature reached 112°F . The animal weighed 47 lbs.

The guinea pigs weighed approximately 28 to 30 ounces each and were exposed to 432, 500 and 680 mw/cm^2 for 28, 40 and 18 minutes respectively. Rectal temperatures elevated 7 to 9°F in all cases. In each instance it seems that the heating is adequate to account for the death and that non-thermal factors are probably not indicated.

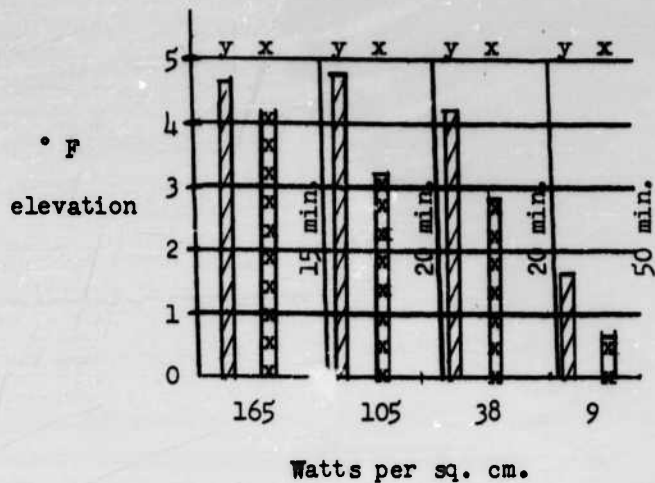
No significance should be attached to the number of deaths we have recorded in our experimental work since we have not tried to determine LD 50s nor maximum dosages which experimental animals could tolerate. Being more interested in the effects of chronic exposure we have usually not chosen dosages which would cause more than a 5°F rise in rectal temperature. Such treatment programs are tolerated quite well. More observations on such animals will be described by Dr. Addington.

Effect of Position in Polarized 200 Megacycle Field

Man, a biped, when in the erect position is potentially a long cylindrical antenna. Quadriped mammals such as dogs likewise may be expected to act more or less like antennas depending upon their size, length of head, tail and body and the manner in which the stretch out in a polarized electromagnetic field.

To gather data on the role of body position as it may relate to plane of polarity in the field during exposure, eleven dogs were exposed at 4 different field intensities in the Y axis (plane of polarization) and at 90° in the X axis.

Data showing relative rises in rectal temperature are seen in the accompanying diagram (Graph 2.).



Graph 2. Body Heating in Relation to Position in Polarized Field

While, as may be seen, exposure duration is not the same in all cases, the trend seems to be clear and consistent. When the longitudinal axis of the body corresponds in its position more or less to the plane of polarization, heating occurs more rapidly and more extensively than when orientation is 90° away from the plane of polarity.

Qualitative differences regarding shape of head, length of tail etc. seem to influence the heating response but too few data on this point are yet available to allow a dependable conclusion in this regard.

SUMMARY

1. Plastic albumen filled models are valuable indicators of heat localization. Observations made thereon may aid in the design of animal experiments.
2. Two subcutaneous "burns", one in a dog and one in a sheep have been recorded and described.
3. Lethal exposures to 200 megacycles-cw have been described.
4. Orientation of the long axis of the body parallel to the plane of polarization of the r.f. field results in more and faster heating than other positions.
5. Convincing evidence of non-thermal effects has not been demonstrated to date.

ELECTRICAL INSTRUMENTATION OF BIOELECTRIC
HAZARDS AT 200 MC AND THE DEVELOPMENT OF
A MINIATURE HAZARD METER

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The University of Buffalo

SUMMARY

A miniature r-f field strength meter and a miniature thermistor bridge are described. More and more information is needed at an almost point locations and microminiaturization of measuring instruments seems to provide the answers. The problems of measuring field strength and temperature with regard to biologic specimens are outlined. Some studies on models indicate that field strengths in the x- and y- coordinate directions may be important at 200 mc where the z- direction is the direction of propagation. Also, the volume power density may be more significant in large specimens than the total power absorbed.

INTRODUCTION

A method for measuring high electric or magnetic field intensity in air (or an equivalent power density) at almost point locations in the vicinity of an antenna, operating at 200 mc has been reported by Fischer, Neubauer and Sarkees.¹

Such a method required a small dipole or loop and a twinax shielded cable to serve as an r-f transmission link to a more remote attenuator, detecting system, and d-c readout microammeter (or oscilloscope if a pulsed system, requiring a peak reading). A relatively simple field-strength meter resulted and the principal development complication to a group like ours at the University of Buffalo was the calibration of this substitute instrument at 200 mc. An elaborate method for calibration against a Stoddart RI-FI Meter was described in the article referred to.

It may be possible to employ a much simpler means of calibration for this field-strength meter when measuring the electric field (this field is considered the pertinent one in biological hazards) by the utilization of an appropriate parallel-plane condenser system energized by voltage at 200 mc. The sensing dipole is to be oriented midway between the plates with its transmission link aligned at right angles to the field. If the geometry is right, one may reasonably assume a uniform electric gradient between the plates which is derived from the overall voltage and spacing of the plates. The plates are to be driven in a balanced-to-ground manner.

The foregoing introduces a basic limitation of the use of field-strength meters - the transmission link ought to be oriented along a line of zero electric field intensity so that the link does not significantly distort the field to be measured. This is, of course, the condition that exists when the instrument is calibrated. A generalized field does not possess a direction of zero electric intensity although a minimum field direction usually may be found. The sensing antenna must be very small with respect to a wavelength. Mutual effects between transmitting and sensing antennas, if any, should be recognized.

Temperatures have been measured by standard means of thermocouples or thermistors by introducing the leads along minimum pickup directions and employing suppressor chokes when necessary. If pickup could not be sufficiently suppressed to yield "power on" readings, then "power off" readings were resorted to.

NEW TRENDS

In order to minimize the effect of the minimum electric field component at least two other recourses are open: (1) Develop an almost neutral link. (2) Miniaturize the r-f field-strength meter with no high conductivity transmission link present (telemetry or remote sensing instead). Both of these methods have been under study and the work is far from completed. Nevertheless some trends of field strength measurements can be reported as new items and will be discussed in this paper just a little later on, along with a miniaturized thermometer. There is no doubt that the basic trend for instruments in this hazard field is toward microminiaturization with associated telemetry of some kind (no leads).

USE OF AN ELECTROMAGNETIC HORN

At this point it would be well to state that many of the electrical and thermal measurement problems encountered in the project were due to the helical beam antenna which possessed an approximately circularly polarized field. It was chosen initially to get us into business rapidly since the antenna was easy to construct. However, since June 1959, a large electromagnetic horn has been in operation which produces a simply polarized field which is quite uniform over a rather large aperture (horn mouth is 6 ft x 6 ft square) with a power density of 0.52 watt/cm² at the mouth diminishing nonuniformly to 0.5 mw/cm², along an axial line, at the end of the 42-ft anechoic chamber. This point is 25.5 ft distant from the mouth of the horn. The horn and launcher were designed experimentally from smaller models operating at 5 times frequency and then the scaling technique was used to preserve the desired antenna impedance and propagation pattern. Both the wave guide cross-section (33.6" x 33.6") and the linearly flared taper are square with the launcher polarization adjustable over 90°. The horn assembly is made of galvanized sheet steel. The launcher is a partial loop terminating on horn ground. The horn is fed by a 50-ohm coaxial line through a two-stub adjustable matching section which was designed locally. Six kilowatts of r-f power are delivered to the horn with a typical standing-wave ratio averaging about 1.05.

With the use of the electromagnetic horn and a simply polarized field, quite accurate field-strength readings are possible in the University of Buffalo anechoic chamber because a direction of almost zero electric field can be found. The chamber has been calibrated for a fixed and reproducible transmitter power with distance as the variable vs. electric field intensity. "Power-on" temperature measurements employing either thermistor or thermocouple instruments with readout exterior to the anechoic chamber also can be made accurately on non-moving small animals for the same reason as for field strengths. This has to be qualified somewhat because long animals like snakes, aligned in the direction of polarization, have capability of changing the direction of polarization at their end points and hence can cause severe pickup on thermometer leads terminating in these locations; such leads are oriented 90° from the original axis of polarization. Leads in the central region are unaffected. The presence of pickup on thermal measurements always can be detected reliably by noting the existence of the short thermal transient of the probe upon "power on" or "power off."

It would seem that no new measuring techniques for electric field or temperature beyond that reported by us in the past would be necessary. This is not quite the case. For field measurement at 200 mc by the original method, the twinax transmission link proved stiff, heavy, and awkward with no intrinsic possibility of becoming partially neutral with all field components present. For temperature measurements on large animals, only partially restricted by a cage, material transmission links exterior to the anechoic chamber do not seem feasible. Also, animals that are irregularly shaped can modify the polarization in some locations.

AN IMPROVED LONG LEAD FIELD-STRENGTH METER

From work done during the summer of 1958 on open wire thermocouples with "power on", it was gleaned that direct current could be a reasonably good transmission link without benefit of shielding. A very flexible lead accrues. A dipole simply leads to a closely positioned shielded detector system with a highly twisted pair of, say, #32 wire leading to a remote d-c readout. This works as well as the original method when the wires are oriented properly, and possesses great lead flexibility. A mild disadvantage is that the instrument is not subject to multi-range with a single scale and a "multiply" dial, which is possible when a-c attenuation is used in the original method.

Enough work was done with this new method to show that a considerable degree of transmission link neutrality could be achieved by suitably placed twin chokes in this twisted pair and by very high resistance in the twisted pair composition, both in the vicinity of the sensing device. Work on this link neutrality was halted when it was decided to build a portable miniature field-strength meter without any sensible transmission link. It must be mentioned that the high resistance leads reduce the output sensitivity.

MINIATURE FIELD-STRENGTH METER OR HAZARD METER

Two research models of portable instruments without leads were constructed to demonstrate the feasibility of such devices at 200 mc. It is believed much smaller instruments can be made with application over a wide frequency range since small non-resonant dipoles are not very frequency sensitive.

The larger, more sophisticated instrument (hazard meter) consists of a 2 1/2-inch, ruggedized, 0-100 microampere, d-c meter-relay (made by Assembly Products, Inc.) with an adjustable upper limit relay contact, operating an audible local buzzer circuit powered by a 1 1/2-volt battery. An averaging shunt rectifier (IN54A) is the solitary component used in the signal circuit for conversion. Pulses or cw can be handled by the averaging detector. A simple dipole or a "bow tie" type antenna (the latter being more omni-directional) is employed over which is placed a rotatable Faraday screen (with circular graduations). This screen effect is analogous to the variable transmissivity of light through two sheets of polaroid material, one rotated with respect to the other. Non-metallic parts of antenna are machined polystyrene. The Faraday screen serves as a full scale set and in one other position, if desired, as a two-step attenuator, all determined when the instrument is calibrated. A detent mechanism could locate the steps. The microammeter which is now merely marked in microamperes could have two calibrations, directly in far-field power density, for the two steps.

The research model, however, has been calibrated as a single step instrument for 200 mc testing with a dynamic range to 100 mw/cm² with the present Tri-Service tentative standard at 10 mw/cm². This standard yields about a 1/3-scale reading. The instrument is properly calibrated in the absence of any human being or foreign object so that it can serve as a true field-strength meter when neutrally supported and viewed from a little distance (like being positioned on the end of an insulating wand which might be held and maneuvered by the hand). It may also be viewed by a telescope at greater distances. The meter-relay can be adjusted to alarm when the field-strength meter reads any pre-selected arbitrary value. It is anticipated that with a limited number of separate instruments, or recalibration "adjust" of the same instruments by different antennas, one can cover a high frequency range at least up to 1000 mc. The instruments are light so more than one unit is not a liability. The authors of this paper could only work at the single frequency available to them.

A smaller field-strength meter with a 1-inch, 0-100 microammeter d-c readout (International Instruments) which can employ an antenna similar as above, but without benefit of alarm, has also been constructed.

It should be recognized that the hazard meter aspect was a by-product of basic research into field-strength meters and much better "second generation" instruments are possible. The heavier research

prototype weighs 1 1/2 pounds; the lighter one weighs 8 oz. A wand and meter scale magnifying lens, if used, will add to the weight.

Since detection is more nearly linear except for the very low field strengths, the scale of these instruments for power density will be nonlinear, since far-field power density will be considered solely proportional to the square of the electric field intensity.

ELECTRICAL FIELD INTENSITY vs. POWER DENSITY

The group would like to point out that electric field intensity, especially at the lower frequencies, is a more meaningful parameter in electromagnetic hazard studies than "far-field" power density since a great deal of hazard occurs in the "near field" where direct measurements of electric field intensity can be made.

This matter seems to be resolved if one regards the meter reading in these studies as solely a measure of electric field intensity squared rather than the classical concept of net power per unit area streaming through a medium. The magnetic field is purposely neglected and search loops are not suggested for hazard meters.

A field-strength meter as described in this paper would possess a reasonably linear scale in the electric field intensity ie. volts per meter.

MINIATURE THERMISTOR BRIDGE AND CLINICAL THERMOMETER

A miniature thermistor bridge, power supply and variable sensitivity rheostat were constructed with a 1-inch readout meter to read about 35°F above bridge-balance temperature. The entire assembly was housed in a metal mini-box 2 3/4" x 2" x 1 1/2". A short thermistor probe was attached. The entire unit is strapped to the animal with the probe in proper position. Long leads are dispensed with and the animal can turn around. The thermistor circuit can easily be calibrated. The instrument in present form only can be read with power momentarily off. Chokes and high resistance thermistor leads should help the "power on" condition where variable pickup can occur on the probe lead as the animal turns. This pickup results in a temperature reading that is too high. A 20-second "power off" interval gives ample time for the probe thermal transient to subside so that a good reading can be obtained.

It is to be noted that a very small meter can be employed when this instrument is to be used as a clinical thermometer since only small temperature rises on live animals are permissible.

SOME EXPERIMENTAL STUDIES ON EGG-ALBUMIN MODELS

The Biology group at the University of Buffalo embarked on a

provocative study of simple geometric shapes of polystyrene tubing filled with egg albumin and exposed to an r-f field.

The tubes were somewhat long compared to their diameter and could be aligned with the direction of polarization or otherwise. The albumin served as a good "hot-spot" index (high temperature) since when it coagulated, it whitened.

It soon became apparent to the Electrical group that these shapes were behaving like lossy dielectric parasitic antennas. Voltage and current distributions could be found along the shapes in the direction of polarization and at right angles to the direction of propagation.

The following important thermal observations were made on tubes aligned in the direction of polarization which seem to be confirmed by electromagnetic field theory when considering lossy media:

(1) Any single uniform tube, with symmetrical load terminations, suffers the highest temperature in the central region (coagulation first) with the ends cool, regardless of the length of tube (within the limits of the horn's effective uniform illumination); except as the tube shortens, a point is reached where no coagulation temperature occurs, although the trend of central heating still persists.

(2) Unequal end terminations or a non-uniform horn field will shift the first impact of coagulation somewhat off center.

(3) If a small diameter tube is junctioned longitudinally to a large tube with the albumin continuous, the first impact of coagulation always appears in the small tube at or near the junction and in an extremely short interval of time considering either the heating time (time for first coagulation) of the small tube alone or the large one alone in a length equivalent to the composite.

(4) All tubes at right angles to the direction of polarization suffer negligible heating compared to the same tubes in the polarized direction.

The foregoing suggests that the electric field distribution in man, caused by electromagnetic radiation, say, in the 200 - 500 mc range (where man's vertical dimension is commensurate with wavelength) should be reappraised in the x- and y- coordinate directions where z is in the direction of propagation of the wave. Variations of the electric field intensity in z-direction, especially at microwave frequencies, has been much discussed in hyperthermia but the boundary value problem with field distributions in the other coordinate directions has been somewhat lost when considering the lower frequencies. The importance of polarization is also apparent.

The suggestion also arises again as to the possibility of a

sudden hot spot in some anatomical substructure without a significant rise of general body temperature. In this case the total power absorbed by a specimen would have less meaning than the volume power density which is proportional to the electric field intensity squared within the small volume.

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PHYSICAL ASPECTS OF MICROWAVE RADIATION

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The purpose of this introductory contribution is to provide some background in microwave electronics that is particularly relevant to the subject of the conference. The potential hazard of microwave radiation to biological systems is a research problem in the interaction between radiation and matter. We should, therefore, consider in a broad way the several aspects of what characterizes microwave radiation, the processes of interaction between radiation and matter, and the measurement of the quantities that are important.

Microwave radiation is a certain spectral region of electromagnetic waves. We are all familiar with wave phenomena in one form or another. A wave may be defined loosely as a propagating disturbance--loosely, because we must differentiate between progressive or traveling waves and standing waves. We shall first speak in terms of traveling waves. In a wave we have some physical quantity that varies in space and time. For example, in the case of surface waves in a body of water the physical quantity is the displacement of the surface from its equilibrium position. At a fixed point in space the surface displacement varies with time and at a given instant the surface displacement varies from point to point. It is not necessary to belabor the point since everyone is familiar with water waves and waves along strings and other structures. What is perhaps not so generally appreciated is that wave phenomena always have associated with them a transport of momentum and energy. That is, in fact, one of the most important aspects of waves.

In the case of electromagnetic waves the physically varying quantity is actually a set of quantities: electric and magnetic field vectors. What we have is an electric field \vec{E} and a magnetic field \vec{H} in space such that at any point they vary with time, and the time variation progresses from point to point. Our measure of an electric field is the force that is exerted on a concentrated electric charge placed in the field. If we should hold a charge in an electromagnetic field, we would observe that the force exerted on the charge varies with time. Our measure of magnetic field is the force exerted on a small electric current element and, again, if we should try to hold a small current element in a fixed position while the wave passes by, we would experience a time-varying force on the element. Although the electric and magnetic fields are characterized by, and accordingly measured by, forces exerted on two different things, they are not independent entities. One cannot have an electric field without the magnetic field in an electromagnetic wave.

Figure 1 illustrates a very simple type of electromagnetic wave and the spatial relationships to which I have referred. The figure pictures the electric and magnetic field intensities along a line of propagation of the wave at some instant of time. The electric and magnetic field vectors are perpendicular to one another and are both perpendicular to the direction of propagation. We call this type of wave a TEM type denoting the transversality of both \vec{E} and \vec{H} with respect to the direction of propagation.

A basic type of TEM wave is one in which \vec{E} and \vec{H} vary sinusoidally in both time and space. As we go from point P_1 to point P_2 in Fig. 1 we note that the vectors \vec{E} and \vec{H} go from a crest value to zero, and from P_2 to P_3 they increase in magnitude but are reversed in direction. P_3 corresponds to a trough in a water wave where the displacement from the equilibrium surface is greater in magnitude than that for neighboring points but is opposite in direction to what it is at a crest. From P_3 to P_4 the magnitude decreases and finally from P_4 to P_5 we have a buildup to the magnitude and direction corresponding to that which exists at P_1 . This pattern repeats itself periodically along the line of propagation. If x measures distance from a maximum point such as P_1 , the spatial variation of E at this instant is

$$E = E_0 \cos \frac{2 \pi x}{\lambda} \quad (1)$$

where E_0 is the value of E at P_1 and λ is the distance P_1P_5 . λ is the spatial period or, as it is better known, the wavelength. If, on the other hand, we should stand at a point such a P_1 and measure the field vectors as they vary in time, we should find that the electric vector, say, fluctuates in time in a sinusoidal fashion

$$E = E_0 \cos 2\pi \nu t \quad (2)$$

if we take as our time reference $t = 0$ an instant such as the one when the conditions are as shown in Fig. 1. The quantity ν is the frequency, the number of cyclic changes in E per unit time. The velocity of propagation, the so-called phase velocity v_p , is related to the wavelength and frequency by

$$\nu \lambda = v_p \quad (3)$$

The phase velocity is determined by the constitutive properties of the medium in which the wave phenomenon occurs. In the case of free space the velocity of propagation v_p is the velocity of light 3×10^8 m/sec. The following table gives values of frequency and wavelength.

Table I. -- Frequency ranges of interest.

| Operational Region | ν (cps) | λ (m) |
|-------------------------|---|---|
| Broadcast | 500 - 1500 x 10 ³ | 600 - 200 |
| TV | 25 - 50 x 10 ⁶ | 12 - 6 |
| FM | 100 x 10 ⁶ | 3 |
| Microwave communication | 4000 - 7000 x 10 ⁶ | 0.075 - 0.043 |
| Microwave radar | 3000 - 30,000 x 10 ⁶ | 0.1 - 0.01 |
| Infrared | 3 x 10 ¹² - 3 x 10 ¹⁴ | 10 ⁻⁴ - 10 ⁻⁶ |
| Visible light | 4 x 10 ¹⁴ - 7.5 x 10 ¹⁴ | 7.5 x 10 ⁻¹¹ - 4 x 10 ⁻¹¹ |

We noted earlier that an important aspect of wave propagation is the transfer of energy. The flow of energy in a wave is described in several ways. One important quantity is the intensity of power flow along the direction of propagation, that is, the energy crossing (per unit time) a unit of area normal to the direction of propagation. This quantity is known as the Poynting vector of the field in the case of electromagnetic waves. In the case of harmonic time variation the quantity of interest is the average value of the flow vector over a time cycle of the variation of the field vectors. Quite generally it turns out to be

$$S = \frac{1}{2} \eta |\mathbf{E}_0|^2 \text{ watts/meter}^2 \quad (4)$$

where E_0 is measured in volts per meter. The quantity η depends upon the medium in which the propagation takes place and the particular form of the wave.

The TEM type of wave which I have described is the type that is associated with propagation along a pair of wires, or within a coaxial line, or at large distances from an antenna (Fig. 2). More complicated types of waves result from the process of combining or superposing TEM waves traveling in various directions. To illustrate, consider a wave type that can be propagated freely in a rectangular waveguide (Fig. 3); it results from the superposition of a pair of TEM waves bouncing back and forth from one wall to the other as they progress along. The composite waveform is one in which the electric vector \mathbf{E} is vertical and the magnetic vector \mathbf{H} has one component parallel to the y direction and one component in the direction of propagation.

So far we have spoken primarily of traveling waves. When a wave encounters a change in the properties of the medium it suffers, in general, partial reflection and transmission. The reflected wave is superimposed on the incident wave and gives rise to a partial standing wave. In the case of total reflection (Fig. 4) the standing wave is complete when the reflected wave is along the same line as the incident wave. The pattern is such that

there are fixed points at which the resultant disturbance is zero at all times, and other fixed points at which the disturbance is a maximum at all times. (This situation is to be contrasted with the traveling wave considered in Fig. 1. In the traveling wave as time progresses, the zero points, and crest and trough points, move along in the direction of propagation. A vibrating string with fixed ends is the result of superposition of waves traveling back and forth along the string.

Reflection of waves is of importance in several connections. First, it gives us a way of measuring the properties of material. A study of the constitutive properties of matter often is made by studying the fractional reflection and transmission of electromagnetic waves through matter. The reflectivity depends on the angle at which the wave strikes the boundary between the two media. Thus, when a material is studied by the measurement of reflector coefficients in waveguide, allowance must be made for the way the component TEM waves are traveling before one can make a comparison with similar measurements made in a coaxial line. This notion is extremely important. For the sake of brevity, we shall merely refer to a very extensive literature on waveguides for details.

The second aspect of reflection that concerns us is the standing-wave pattern in which the field intensity is greater than or less than that in either separate traveling wave. A field with an intensity just at the threshold of safety in the absence of reflections can become hazardous at the antinodes (points A in the standing-wave pattern of Fig. 4) when reflection occurs. Reflection is associated with dimensional resonance phenomena where the field amplitude builds up because the dimensions are such that successive reflections from walls or boundaries just overlay one another properly in space and time. Such dimensional resonances can occur in structures like the eyeball; the determining factor is the ratio of the wavelength (in the medium constituting the eyeball) to the dimensions of the eyeball. The seriousness of the effect depends on the reflecting properties of the walls.

A third important aspect of reflection is its bearing on any discussion of dosage. Consider a body, say a mouse, placed in a field whose intensity is known in the absence of the mouse. The free-space value may have little relevance to establishing a tolerance level for other animals, for what is important is the field within the mouse. To determine the latter we must determine the reflectivity of the fur and skin.

Another aspect of radiation that often comes into consideration is the quantum structure. From the quantum-theory standpoint all electromagnetic radiation has a discrete structure made up of energy elements

$$\epsilon = h\nu \quad (5)$$

where $h = 6.625 \times 10^{-34}$ joule-sec is Planck's constant and ν , as before, is the frequency. From the quantum standpoint the power intensity measures the mean value of the number of quanta per unit volume flowing along in the beam. The quantum aspect is important in considering processes of interaction between radiation and matter. When the interaction is at the atomic

and micromolecular level we must consider whether or not the quantum energy associated with the frequency ν corresponds to the energy state difference of the atoms. If it does, there is a possibility of resonance absorption at the atomic level and the possibility of dissociation. This is the essential basis of radiation hazards in the visible, ultraviolet, and X-ray regions, for example, and in the interaction between high-energy particles and matter.

A convenient way of delineating the quantum effect is in the terms of a temperature T_ν , given by

$$h\nu = kT_\nu \quad (6)$$

If T_ν is very small compared with ordinary temperatures, quantum effects are of minor, and indeed of negligible, significance in the interaction between the radiation and matter. For if resonance absorption and emission were possible at energies corresponding to the low value of T the system would be in a continuous state of spontaneous transitions merely by virtue of the thermal excitation processes. And if the processes in question were dissociative and were to lead to irreversible changes in the system, the dissociation and change would take place without the introduction of radiation.

If T_ν is comparable with ordinary temperatures or greater than ordinary temperatures the effects must be given due consideration with reference to resonance phenomena associated with interactions between the radiation and matter at the atomic and micromolecular level. Table II gives the equivalent temperature T_ν for various spectral regions.

Table II--Equivalent temperatures over the electromagnetic spectrum.

| | ν (cps) | T_ν (°K) |
|--------------------------------------|-------------------|----------------------|
| Broadcast | 1.5×10^6 | 7.2×10^{-5} |
| FM | 10^8 | 4.8×10^{-3} |
| Microwave communication and radar | 10^{10} | 0.48 |
| Infrared | 10^{12} | 48 |
| Visible light | 10^{14} | 4800 |

So far we have mentioned only continuous waves. Any power source is limited by its structure as to the continuous power it can deliver and, therefore, the amplitude of field vector that can be obtained under cw. operation is limited. The distribution of energy in time can be varied in many ways without affecting the average power. A generator can be pulse-modulated to emit energy over a very small time interval and to be inactive over a comparably long interval. During the active interval the power attained is many times greater than the average power. This situation is possible since by virtue of the heat capacity and other thermal constants of the generator the power does not reach the danger point of destruction that it would reach if the same power were delivered over a long period of time. Corresponding to the peak power the field amplitude in the wave attains an enormously

high value during the active period. One special advantage of microwave oscillators is their susceptibility to pulsing operations. This feature may result in the misinterpretation of the frequency dependence of radiation hazards. The hazard may be associated with the field intensity rather than with the frequency in any direct way. In comparing results obtained at different frequencies under pulsed conditions one must be very sure to determine that the field intensities are the same and the active periods are comparable.

To explain some of these points we must consider in at least a qualitative way how the electromagnetic field interacts with matter other than by quantum resonance interactions. Atoms and molecules are complexes of charge and when placed in an electric field, their charge distribution becomes polarized. Some systems, such as many molecules in living tissue, already have charge distributions which are polar, that is, the center of gravity of the negative charge is displaced from that of the positive charge. All membranes have polar structure. In a time-varying field the polarization vibrates and by virtue of interaction between different elements of the molecular system the electrical energy is transformed into heat. This is the dominant phenomenon in what may be termed the low-amplitude region. The hazard aspect of radiation of this regime is a purely thermal one. The destruction of tissue is a secondary process resulting from the generation of heat. If the intensity of radiation is low so that the rate of generation of heat can be handled by the distribution processes in the organism, the result is only discomfort. When the intensity of radiation increases not only does heat generation increase but another effect comes into play. The enforced redistribution of electric charge under the applied field can be so great that a complete reorganization results. This is the so-called process of field-induced transitions. It is particularly possible in systems comprising unsaturated bands and relatively free electrons and comprising loose bonding such as hydrogen bonds. The interaction is only incidentally associated with the frequency of the wave; rather, it is one of a field effect and could be produced by static fields if static fields of comparable intensity could be produced. The magnetic field vector in the electromagnetic waves generally plays a secondary role in this process. Magnetic forces are considerably smaller in magnitude. But they are not entirely negligible and in the case of large magnetic field intensities the forces can build up to a point at which mechanical disruption can take place.

It becomes apparent quickly that what must be measured are the electric and magnetic field vectors within the structure under study and the frequency and power flow. Measurements within the system at a microscopic level are actually impossible and the values must be inferred. It is necessary first to measure the values in "free space" regions. The electric and magnetic vectors can be measured by inserting probes and loops into the waveguide or the space inside a coaxial line. The conversion to absolute level is effected by measuring total power flow by terminating the line in a so-called matched load or nonreflecting load. The absorption results in heating of the load and by calorimetric methods the absolute amount of energy transferred to the load is measured.

The measurement of the field intensity in free-space is actually more difficult. We generally divide the space with respect to the field produced by an antenna into two major regions: the near zone and the far zone. In the far zone, the dominant structure of the field can be described as a TEM wave

as was indicated in Fig. 2. In this region the power flow takes place as though it is emanating from a point source, that is, in any radial direction the intensity varies inversely as the square of the distance from the antenna. At a fixed radius the intensity is found to be a function of the direction of observation, that is, of the angular positions (θ, ϕ) (Fig. 2). It is convenient to describe the power flow in terms of the power per unit solid angle in the direction (θ, ϕ) and to describe the directivity by a gain function $g(\theta, \phi)$. The gain function relates the power $P(\theta, \phi)$ radiated per unit solid angle in the direction (θ, ϕ) to the total power P_T radiated by

$$P(\theta, \phi) = \frac{P_T}{4\pi} g(\theta, \phi) \quad (7)$$

The electric field intensity at a distance R and in direction (θ, ϕ) is given in terms of $P(\theta, \phi)$ by

$$|E_0|^2 = \frac{2 P(\theta, \phi)}{\eta_0 R^2} \quad (8)$$

where η_0 , the constant of Eq. 4, for free space is $1/377$ mhos. The gain function can be measured by various methods without reference to the total power radiated. The power radiated is the difference between the power flowing to the antenna on the transmission line and the power reflected by the antenna to the line. One method of determining P_T is by substitution of an absorptive load which presents the same mismatch to the line as does the antenna and measuring the heat absorbed by calorimetric methods. The validity of the technique depends upon the absence of any appreciable reflections from walls or other bodies outside the antenna and negligible losses in the transmission line feeding the antenna.

In the near zone region the field has a rather complex structure and any measurement is complicated by interactions between the measuring device and the antennas. While in principle one can measure fields in the near zone, the measurement of the field within a body placed in the near zone becomes exceedingly difficult.

This brief survey in no way covers all of the fundamental problems in microwave physics and technology that are relevant to the investigation of radiation hazards. We have tried to point out some of the aspects of the field which enter into all phases of studying the interaction between radio-frequency radiation and matter and to call attention to where the interpretation of experimental results may go astray.

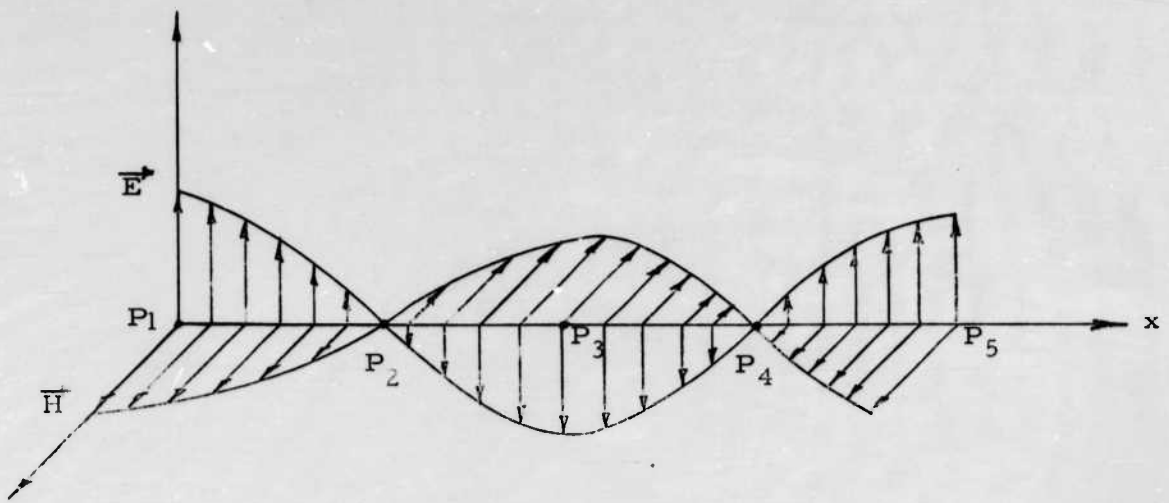
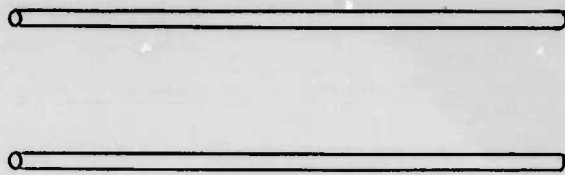
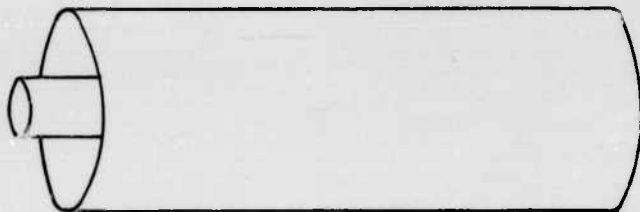
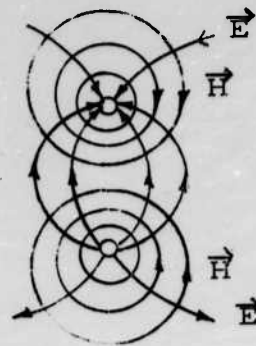


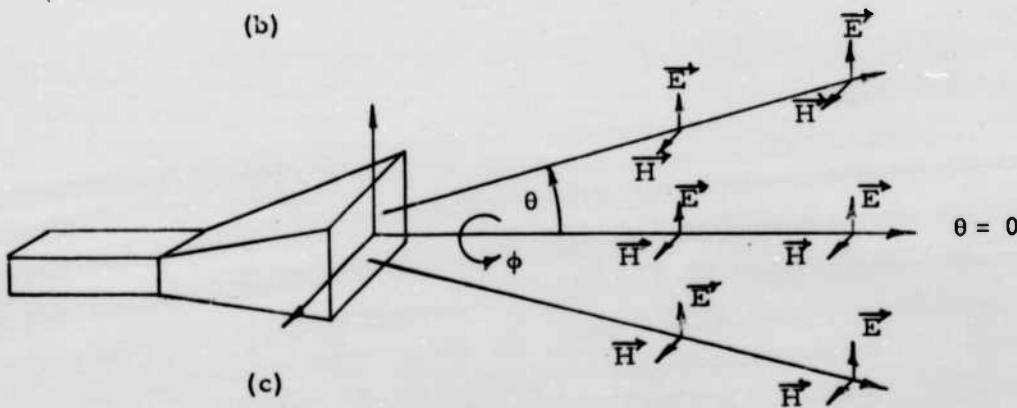
Fig. 1. -- Spatial variation of the electric vector \vec{E} and magnetic vector \vec{H} in a simple TEM wave.



(a)



(b)



(c)

Fig. 2. --TEM waves: (a) Wave guided along by a pair of parallel wires; (b) wave guided along between a pair of coaxial conductors; (c) wave at large distances from a horn-type antenna: at large distances the field spreads along radial lines as though the source were a point, and the source is directive in that the intensity of the field at a given radius vanes with angular position.

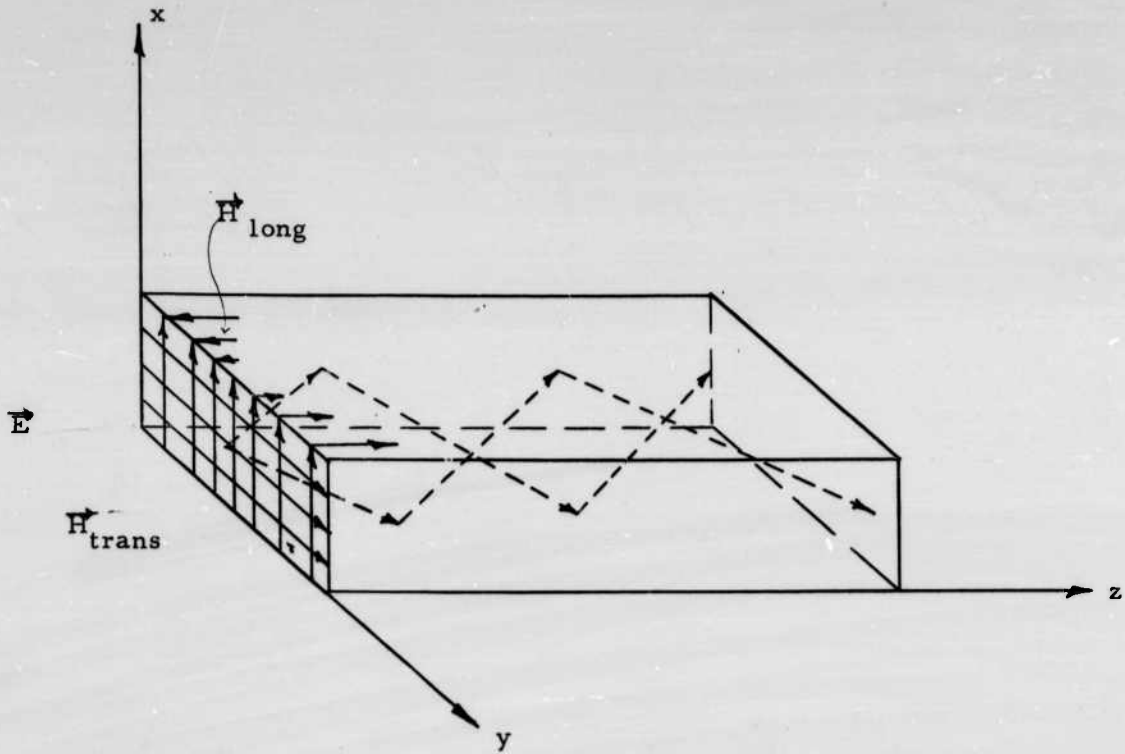


Fig. 3. --Superposition of two TEM waves to form a TE wave in a rectangular waveguide. The arrows indicate qualitatively the relationships between \vec{E} and \vec{H} at the plane $z = 0$; distribution of \vec{H}_{long} on the bottom is opposite in sense to that on the top wall of the waveguide.

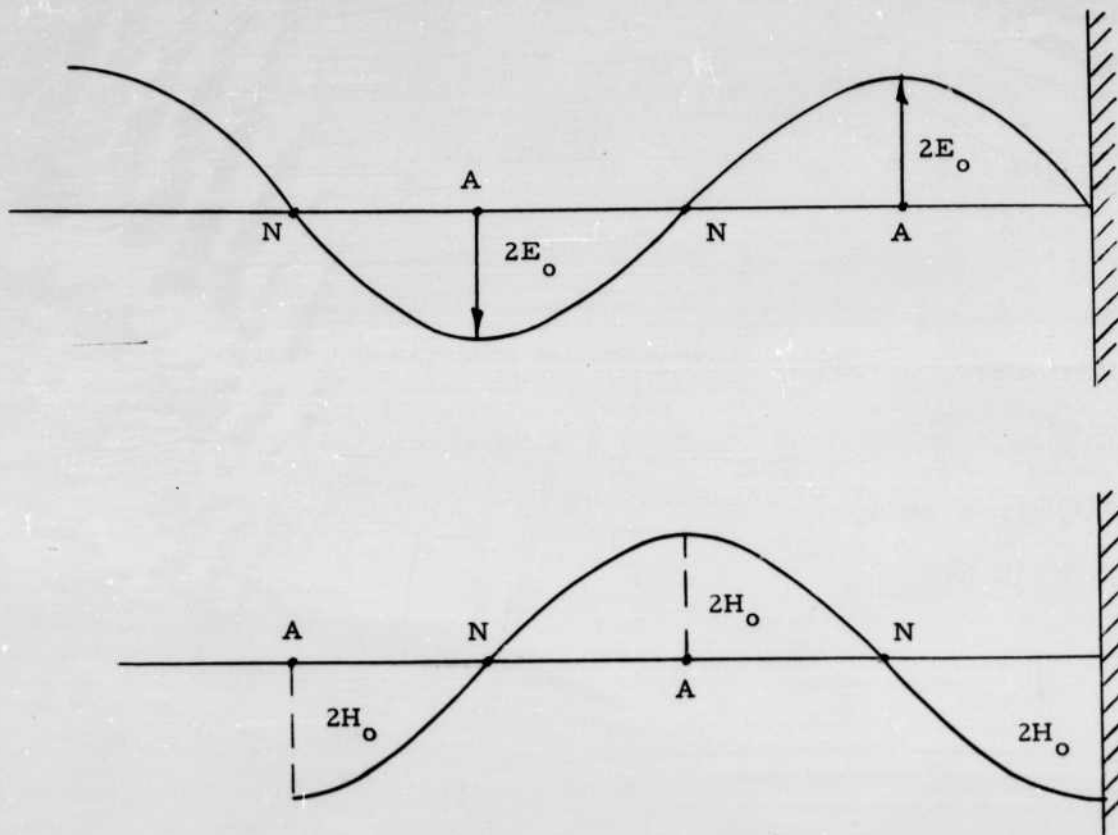


Fig. 4. --Relative spatial distributions of electric and magnetic intensities when a plane wave undergoes total reflection under normal incidence on a perfectly inducting plane boundary. The field intensities at antinodes (A) are in each case twice the maximum value in a single traveling wave. Nodal points of \vec{E} coincide with antinodes of \vec{H} .

TEMPERATURE REGULATION IN LABORATORY ANIMALS IRRADIATED WITH 3-CM MICROWAVES

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Investigations carried on in Berkeley in which the thermal response of small animals to microwave irradiation was studied have consisted of two different types of exposure. The first is long-term, low power-density, continuous irradiation; the second is intermittent exposure to power densities varying between 0.009 and 0.438 w/cm². Temperature recovery was also studied in an effort to determine what factors influence the rate of cooling.

Twelve-week old Swiss Albino mice weighing approximately 35 g and 3-week old Sprague-Dawley rats of approximately 80 g were irradiated with pulsed 3-cm radiation from an AN/TPS-10D radar transmitter. Temperatures were recorded using a thermistor inserted rectally.

In the first series groups of mice were irradiated at low power densities until death or for 4-6 hr apiece. It was found that below 0.060 w/cm² the temperature rise graph reaches a steady state that is below lethal temperatures. The temperature at which this steady state occurs is a function of the power density.

Close analysis of individual temperature recordings of mice exposed to low power densities indicated an interesting phenomenon. Figure 1 shows that instead of a uniform temperature rise and subsequent balance during irradiation, a periodic short-term rise and fall in temperature occurs. (This phenomenon was also seen in the data presented by the Rochester group.) The experiment was repeated, both with normal and with hypophysectomized rats. The normal rats exhibited the same behavior as did the mice described above, whereas the hypophysectomized rats gave smooth temperature curves that rose and subsequently leveled off. In the latter case there was no periodic rise and fall in body temperatures.

This behavior is suggestive of a homeostatic process. When the animal's environment is changed to raise the animal's body temperature outside the normal limits, its thermal system tends to become unstable and alters its activity to correct for the environmental change. It is suspected that the hypothalamus is the thermoregulatory mechanism, but the function of this mechanism is not fully understood. The data indicate that at low power densities a stabilizing plateau is reached periodically after a certain amount of overshoot. If the environment provides too much heat the hypothalamus is unsuccessful in stabilizing the thermal system of the animal and the animal subsequently dies.

A series of tests was made to determine whether the thermal response to microwave irradiation is in any way affected by previous exposures.

In the first test the mice were exposed to 0.156 w/cm^2 for 5.25 min (3/4 of the LD50). The mice were then divided into five subgroups, each receiving a rest period of different length before the power was again turned on until death. The rest periods were 5, 10, 15, 20, and 60 min. It was found that when the power was turned on after a rest period between 5 and 20 min, the initial average slope of the temperature rise curve was flatter than that during the first irradiation, but after 2-3 min the slope changed to the original rate for the corresponding body-temperature elevation. This phenomenon is shown in Fig. 2 for cases where a 10-min rest was given. This result was observed both when the temperature had not yet reached the original baseline, as in the 5- and 10-min rest periods, and when it had, as in the 15- and 20-min rest periods. Exposure after a 60-min rest period produced a temperature rise curve similar to that obtained during the first irradiation.

In the second test the mice were irradiated with power densities between 0.234 and 0.438 w/cm^2 so that 1-min exposures were alternated with rest periods of 1, 2, 3, and 5 min, till death occurred. Figure 3 plots the results for mice irradiated at 0.438 w/cm^2 for 1 min at a time at intervals of 1, 2, 3, and 5 min. The temperature curves show that continuous exposure to this power density for 2.5 min raises the body temperature at approximately the same rate as half or even a third of this dose of microwave energy per unit time; during the exposure period a quarter of the continuous energy (1:3) slowed down the temperature rise rate to approximately 70 per cent of the original. This part of the data is similar to some of those reported previous to this meeting by Deichmann and co-workers.

A more detailed plot of the temperature-rise curves is shown in Fig. 4. This plot indicates what happens while the power is off and offers an explanation of why half the continuous, or control, dose of microwave energy per unit time results in a net temperature rise during the exposure period that is roughly equivalent to that occurring during continuous exposure. It was reported at last year's conference that body temperature continues to rise for a short time after the power is turned off at power densities above 0.2 w/cm^2 . Apparently at this power density rest periods of 1 or 2 min are not long enough to permit the mouse's body temperature to cool below the maximum reached during the previous exposure. Furthermore, the slope of the temperature curve during exposure is the same in each case. Therefore, by dropping out the rest periods a temperature-rise rate results that is very similar for continuous irradiation and for 1-min exposures alternated with 1- or 2-min rest periods.

There are two factors at play when the rest period is longer than 2 min. First, the body temperature drops below the maximum reached during the previous exposure. Second, there is an initial lag in the temperature-rise curve in all irradiations after the first one, as can be very clearly seen in Fig. 2; it is also evident in Fig. 4. As a result of these two factors the net rate of temperature rise is slower than that of mice irradiated continuously.

Figure 5 indicates what occurs when a lower power density of 0.234 w/cm^2 is used. In this case half the control dose of microwave energy per unit time takes 1.5 times as long to achieve the same temperature rise as during continuous exposure, largely because there is a temperature drop

during most of the off periods.

It may be of interest to note the variation with time in body-temperature reaction during the off periods. It appears, from Figs. 4 and 5, that cooling becomes more efficient with time.

It was also found that for a given power density there is a ratio of time on to time off that produces a net balance or lowering of the body temperature.

These results seem to suggest that there is a time delay before the cooling mechanism begins to operate, as can be seen in Fig. 2, where a 10-min rest is given between exposures to radiation at relatively low power density; and again in Fig. 4, where the rest period is 3 min or longer. The initial lag is evident even when a high power density of 0.438 w/cm² is used. A 60-min rest period seems to provide too long a time lapse between the initial stimulation and the repeated thermal exposure, so that the temperature curves of the first and second exposures are identical.

In summary, then, a second exposure may be affected by a previous one if it was given not less than 3 and not more than 20 min before, in that the initial exposure stimulates the cooling mechanism to operate more efficiently during the second exposure.

The repeated-exposure experiment was next somewhat modified by devising a set-up that would permit faster on:off cycles, thus simulating the type of exposure one would receive while standing in the field of a rotating antenna.

Microwave absorbing material suspended in a circle over the radiating horn contained a pie-shaped aperture, the angle of which was variable. Aperture angles of 30°, 60°, 90°, 120°, 150°, and 360° were used. The structure was rotated at a speed of 12 rpm. Power densities used ranged between 0.009 and 0.360 w/cm².

Figure 6 is a summary of the results, which are still quite preliminary, although general trends can be seen. From the data compiled to date it appears that the larger the angle the greater the temperature rise. This seems to be in linear proportion so that

$$\Delta T \sim P \alpha$$

where

T = steady state temperature

P = power density

α = aperture angle

Groups of mice were irradiated under varying conditions in an effort to discover what factors determine the rate of cooling. In the first phase of this experiment the mice were irradiated at power densities ranging between 0.156 and 0.438 w/cm² with 80 per cent of the LD50.

The curves in Fig. 7 show that the average maximum temperature rise was 5° C above normal. The cooling rate is the same in each case.

Three groups of mice were next irradiated at a low power density of 0.047 w/cm² for various periods of time. It was shown at last year's conference that this power density produces a steady-state temperature rise of approximately 2.3° C. Figure 8 shows the cooling curves of mice irradiated at 0.047 w/cm² for 10 min, 1 hr and 4-6 hr. Cooling from an average maximum body temperature rise of 2.3° occurred at the same rate in each case.

If a plot is made on semi-log paper of the average curves derived from Figs. 6 and 7 the straight lines in Fig. 9 result. They are described by a Newtonian cooling equation having an asymptote of -1.2° C:

$$T - T_0 + 1.2 = Ae^{-bt} \quad (1)$$

where

- 1.2 = cooling overshoot
- T = temperature
- t = duration of cooling
- A = constant related to maximum temperature
- b = cooling rate constant

The 1.2 figure represents the cooling overshoot seen in most mice (described previously).

Equation (1) yields upon differentiation

$$\frac{dT}{dt} = -Abe^{-bt} \quad (2)$$

which describes the rate of cooling. If Eq. (1) is then multiplied on both sides by b it reads

$$bAe^{-bt} = b(T - T_0 + 1.2) \quad (3)$$

which yields upon substitution into Eq. (2)

$$\frac{dT}{dt} = -b(T - T_0 + 1.2) \quad (4)$$

The significant thing about Eq. (4) is the fact that A and t drop out so that the cooling rate depends solely upon the prevailing body temperature and not upon the maximum temperature reached nor upon the length of time the animal has already been cooling. Equation (1) also indicates that in cooling, temperature is an exponential function of time.

In conclusion, then, the work done at the University of California with animals during the past year may be summarized as follows:

(1) Normal mice and rats exposed to low level microwave irradiation experience a net temperature rise and subsequent balance which is characterized by a periodic rise and fall in body temperature. When hypophysectomized rats were exposed to the same irradiation dose the resulting temperature-rise curve was smooth.

(2) Repeated exposure to microwaves indicated that the temperature curve from the second irradiation following a previous one by 3 to 20 min may be modified by more efficient cooling during the initial few minutes. If the time lag is outside these limits such modification is not apparent.

(3) Preliminary data on exposures of the rotating antenna type indicate that the change in the steady state temperature rise is roughly proportional to the aperture angle times the power density.

(4) The rate of cooling after exposure to microwaves depends solely upon the prevailing body temperature and not upon the maximum temperature nor upon the length of time the animal has been cooling. Furthermore, it was found that during cooling, temperature is an exponential function of time.

Body temperature
(deg C)

0.047 w/cm²

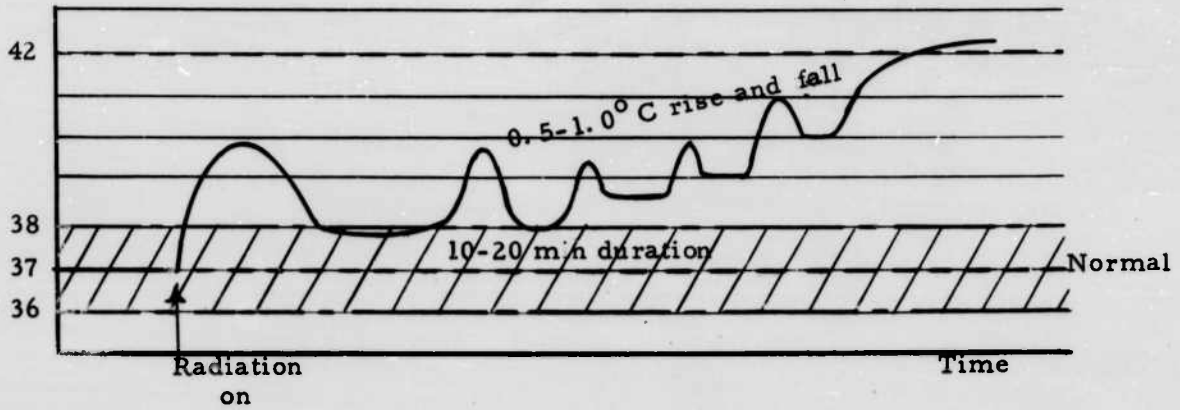


Fig. 1. -- Typical individual temperature record at low power density.

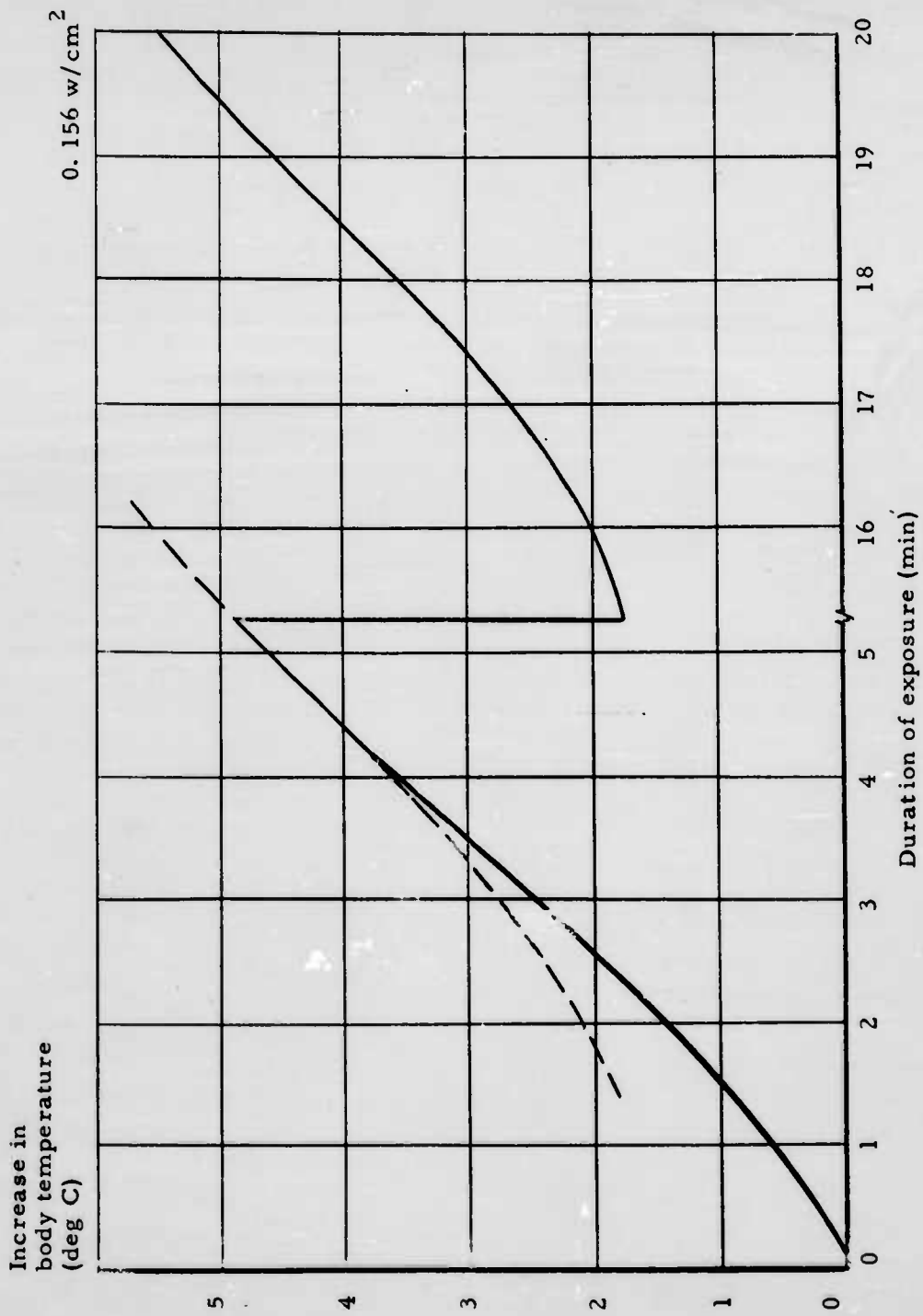


Fig. 2. ---Average temperature rise during exposure to 0.156w/cm² before and after a 10-min rest.

Increase in
body temperature
(deg C)

0.438 w/cm²

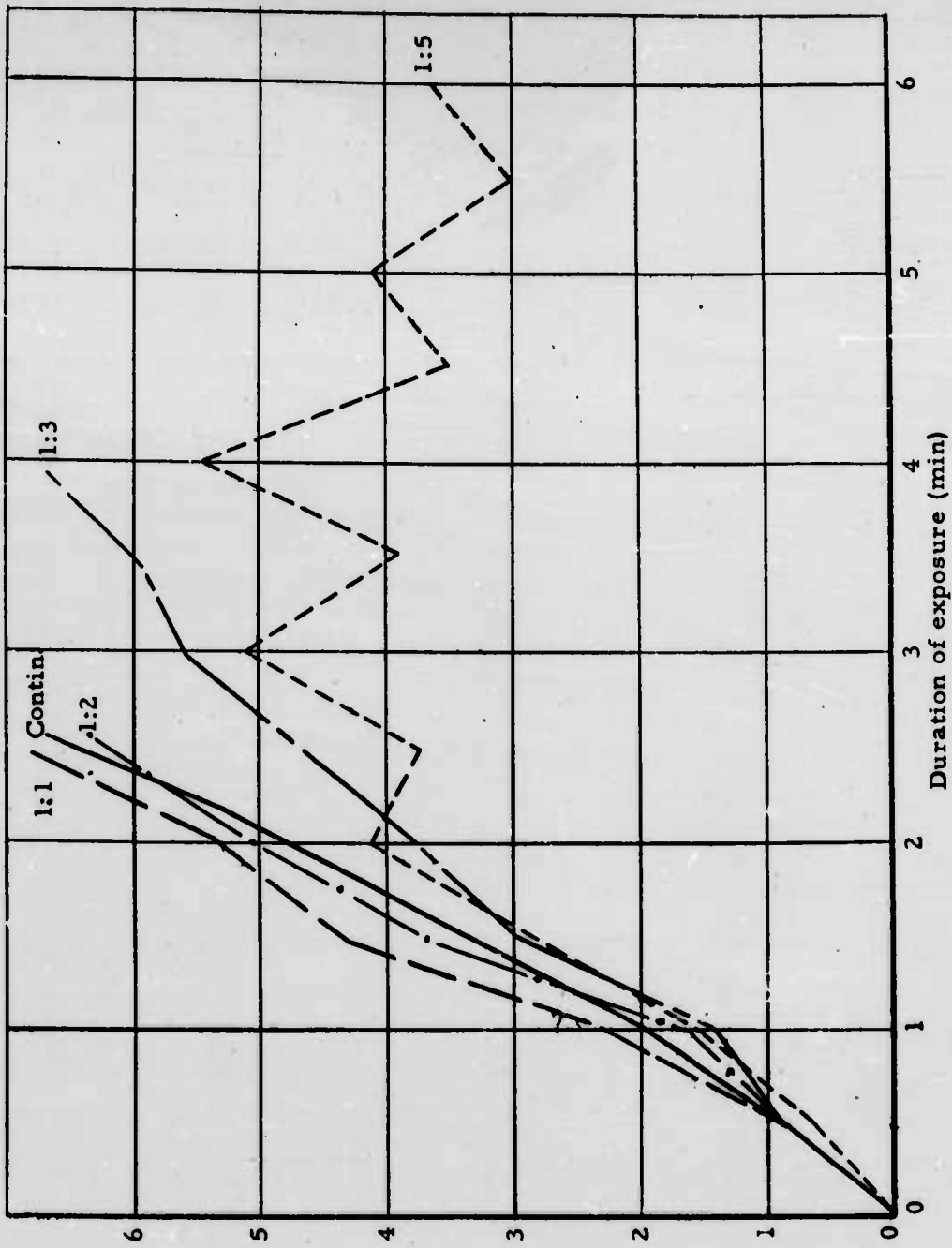


Fig. 3. -- Effects of intermittent exposure to 0.438 w/cm² on the rectal temperature of the mouse, showing thermal response during irradiation only. Rest periods are 0, 1, 2, 3, and 5 min.

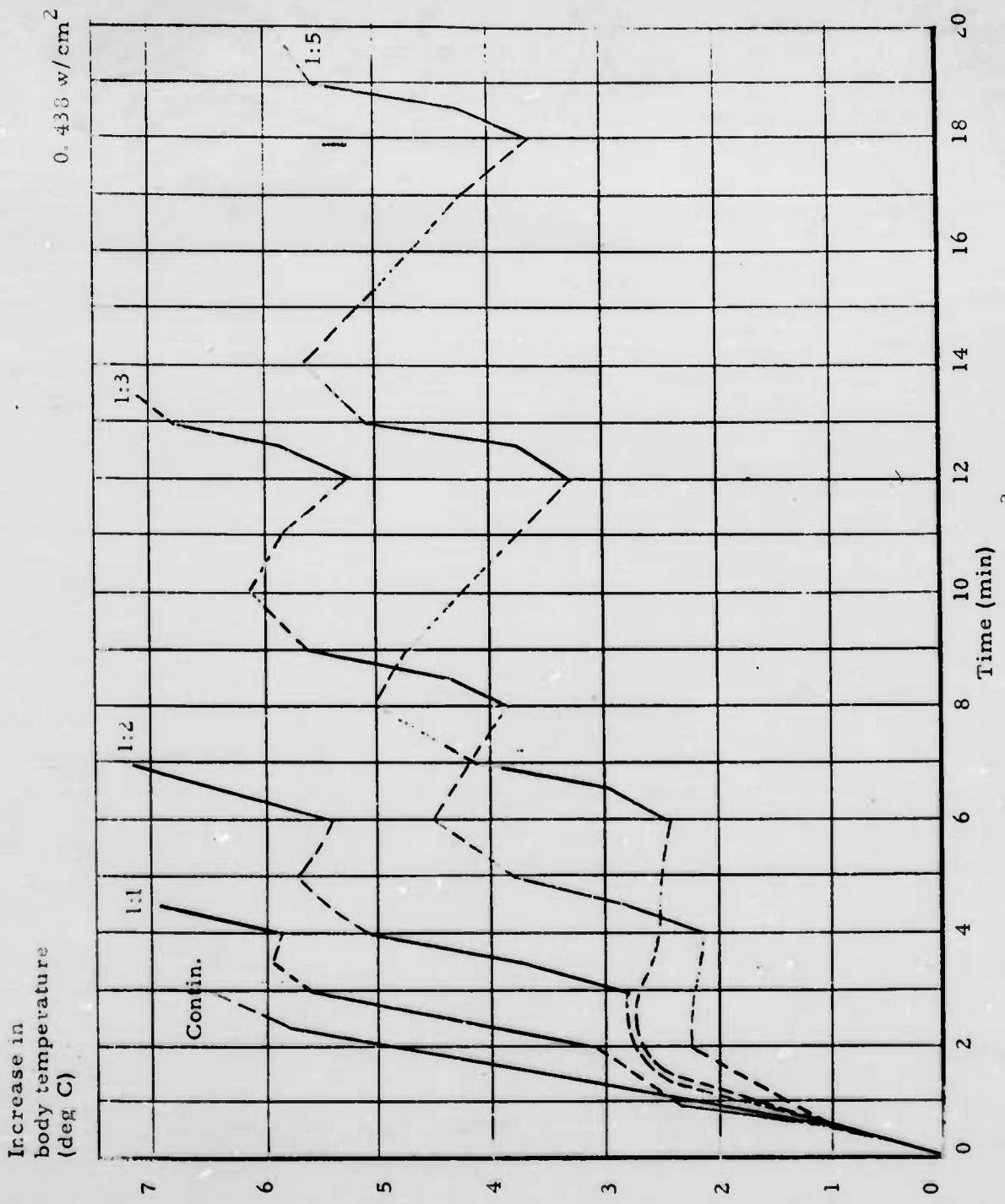


Fig. 4. -- Effects of intermittent exposure to 0.438 w/cm² on the rectal temperature of the mouse, indicating thermal response during exposure and rest periods.

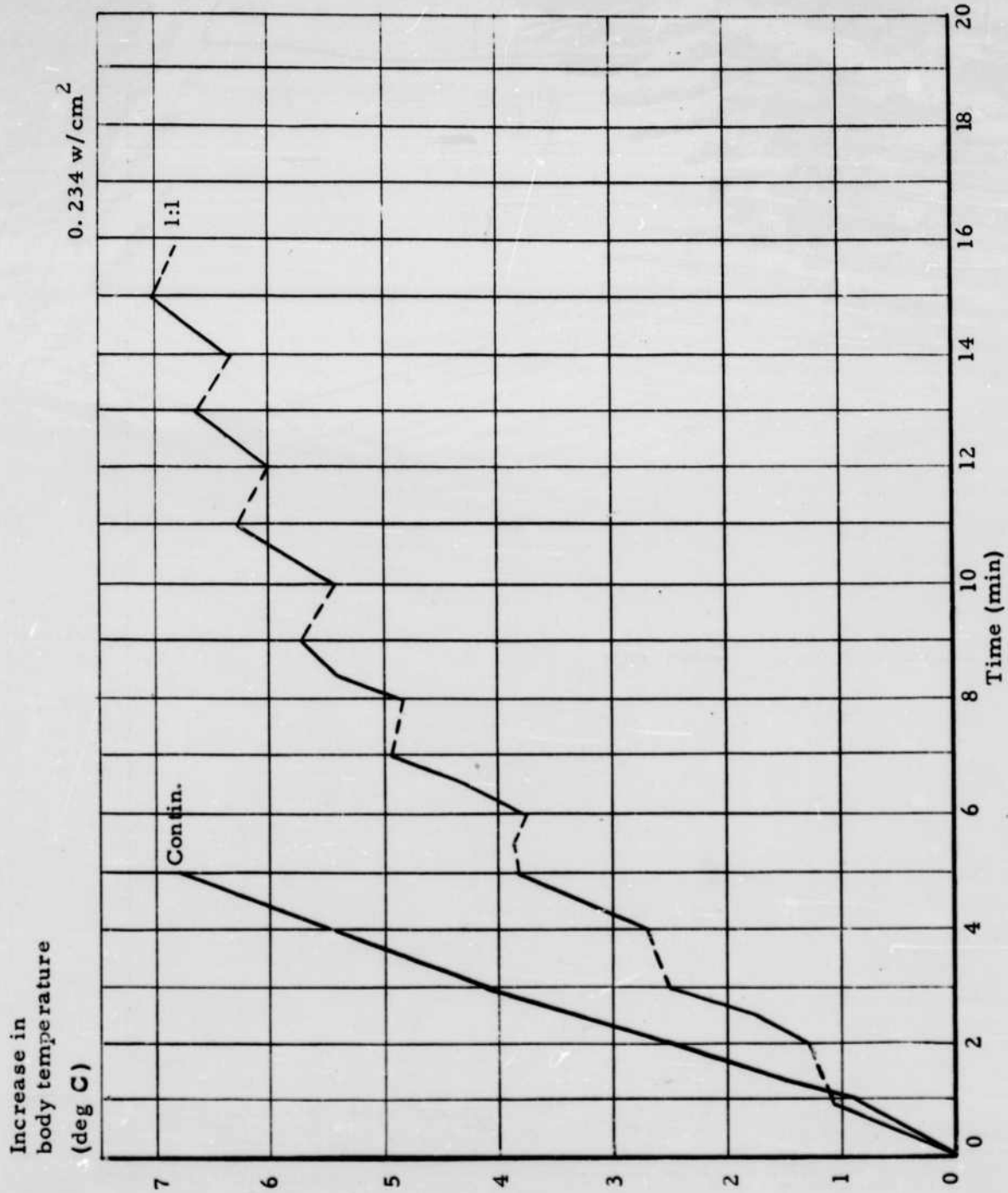


Fig. 5. -- Effects of intermittent exposure to 0.234 w/cm² on the rectal temperature of the mouse, indicating thermal response during exposure and rest periods.

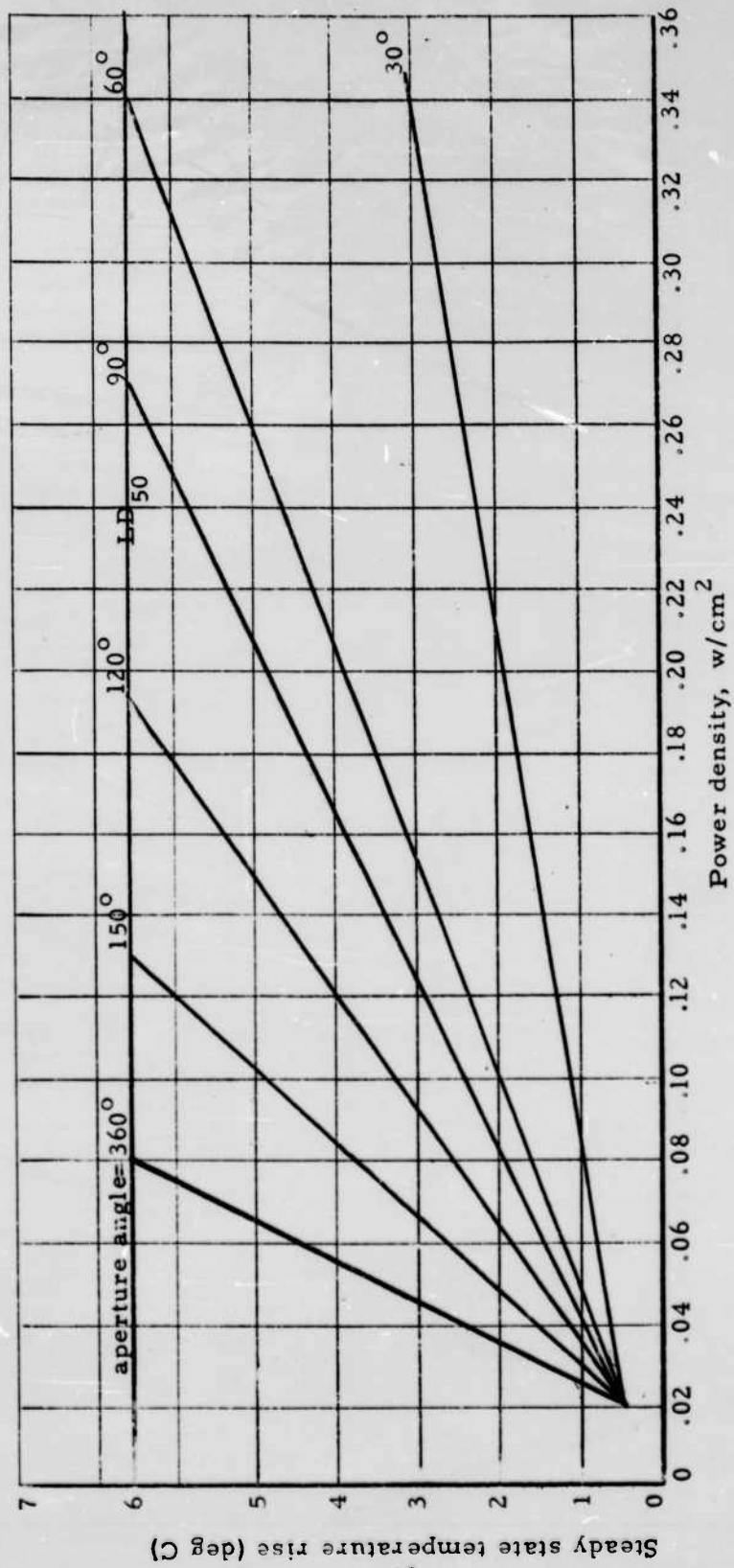


Fig. 6. -- Rotating antenna.

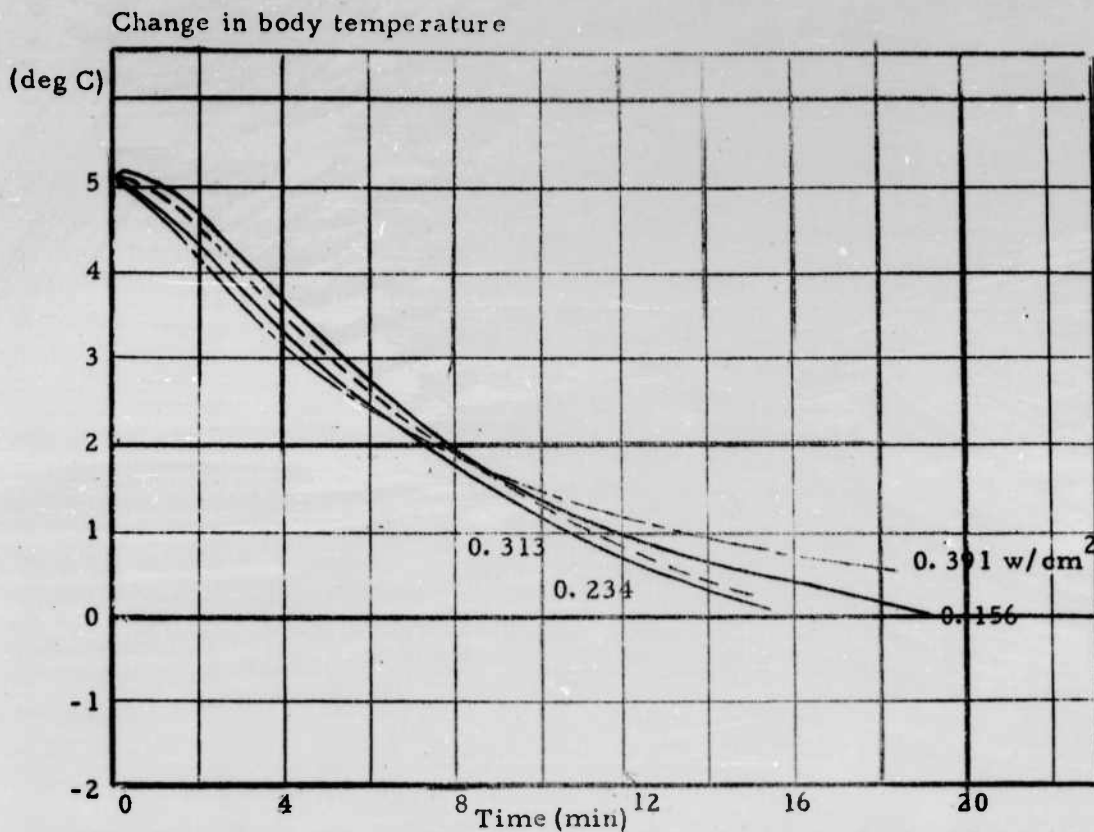


Fig. 7. -- Comparison of the average cooling curves obtained by raising the body temperature of mice 5°C above normal, at various power densities.

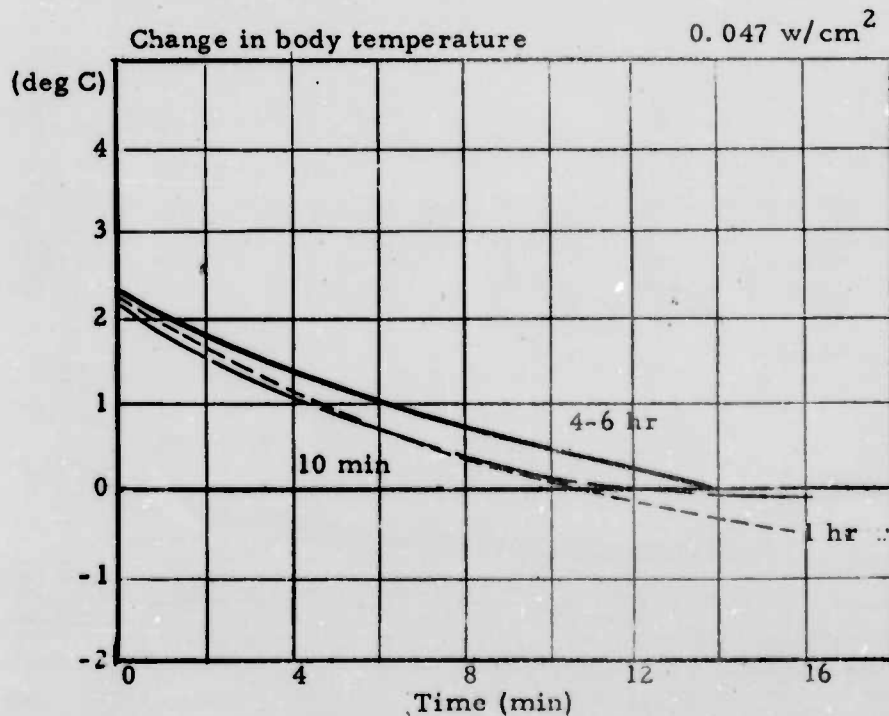


Fig. 8. -- Comparison of the average cooling curves obtained by raising the body temperature of mice 2°C above normal. All mice were irradiated at 0.047 w/cm², but exposures were of different durations.

$$T - T_b$$
$$(T_b = T_0 - 1.2^\circ\text{C})$$

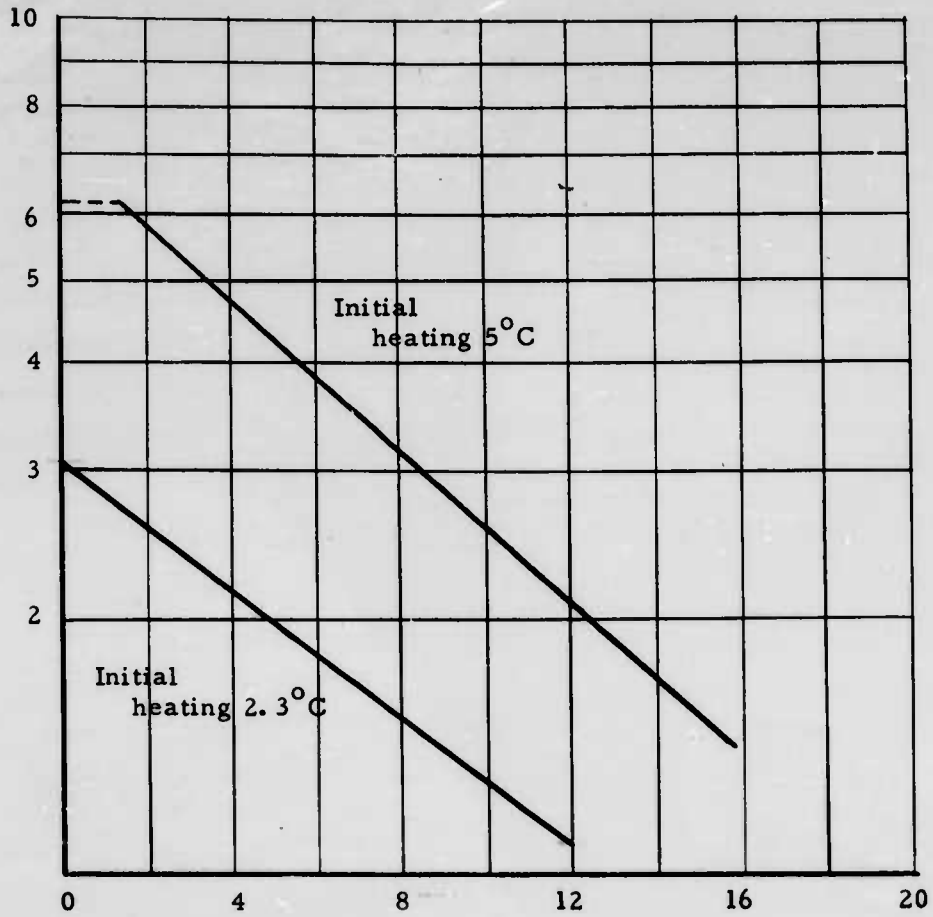


Fig. 9. --Average postirradiation cooling curves.

ANALYTICAL AND EXPERIMENTAL INVESTIGATION
OF UNICELLULAR ORGANISMS WITH 3-CM MICROWAVES

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A clear and concise statement of the problem has to precede any type of investigation, be it analytical or experimental. The question with which we are concerned is the possible production of nonthermal effects by 3-cm electromagnetic radiation. What do we mean by nonthermal effects?

Electromagnetic radiation can act on single cells basically in two ways: directly, by eliciting resonances in the molecules that constitute the organism; and indirectly, by interaction with the medium in which the organism lives. This indirect action consists of a degradation of the impinging energy to a form that increases the random motions of the molecules of the medium. In other words, the indirect action increases the temperature of the organism. Such an indirect effect of electromagnetic radiation we shall call a thermal effect. Distinct from the indirect action of electromagnetic waves is the direct effect; this is a condition of resonance with molecules in the living organism. Such radiation modifies the distribution of the quantum levels of the molecules and changes their rotational or vibrational energy distribution depending on its frequency. The basic difference between thermal and nonthermal effects is, then, the action of randomly distributed energies versus the action of discrete changes in energy levels of the substance under investigation.

As one can clearly see, it is quite difficult to differentiate between these two modes of action at microwave frequencies, since all that is possible in that frequency range is a superposition of discrete frequencies on the intrinsically random distribution of the rotational energy levels. All one can do is to enhance the randomness of the motions by cooling or heating, and to superimpose certain frequency bands on these distributions. Since the subject under investigation is quite temperature-sensitive, the intrinsic distribution of the vibrational or rotational energy levels cannot be eliminated by operations conducted at very low temperatures.

The first experimental investigations of such superpositions lead to interesting observations. De Pereira Forjaz in 1935 investigated the influence of radio waves on the kinetics of chemical systems.¹ He observed an acceleration of the reaction speed (up to 40 per cent) in the synthesis of ethyl and methyl acetate and equally in the formation of the corresponding formic acid esters. The same type of radio waves with wavelengths from 0.1 to 400 m applied to the phosphatase system shows a definite effect in accelerating the speed of hydrolysis by about 20 per cent. It cannot be said, however, whether the structure of the enzyme or the enzyme-substrate compound is influenced by the radiation.

Lepeschkin in 1945 compared the effects of heating human serum in a water bath with its irradiation by electromagnetic rays of wavelengths from 2 to 20 m by analysis of its mean molecular weight.² He observed that heating by itself brought about a decrease of the mean molecular weight of the serum proteins. A polymerization under the influence of heat was only observed on euglobulin, which amounts to less than 20 per cent of the total serum proteins, whereas serum albumin and pseudoglobulin decompose on heating. The change in the serum that is brought about by short waves is a much more stable effect than that produced by heating. The effect of heating disappears after a few minutes, as long as the point of denaturization has not been reached. Cooling of the serum enhances the polymerizing action of short waves.

These two experiments may serve as examples of the possibility of nonthermal effects in the region of short radio waves. The specific action of the electromagnetic radiation on the organic molecules can be interpreted as a superposition effect of radiation of a specific wavelength on a normal background of thermal agitation. Enzymes or proteins are highly complex molecular structures with a great degree of biological specificity encoded in them. A subtle change in their structure affects their biological activity to a variable extent, which depends on the location of the change. The usual method by which structural changes in high molecular compounds are brought about is by use of ultraviolet radiation, X rays, or particulate radiation. However, these highly energetic types of radiation do not produce subtle changes in most cases but actually break the molecular compounds into fragments by ionization of components of the molecules. X rays can also be used to obtain structural information about molecules by means of scattering or diffraction. But one aspect, which may be more or less important in the analysis of a living cell, is lost by such methods: the dynamical relationships between the constituents of high molecular compounds, their modes of vibration, of rotation, and of energy exchange in this region.

Early investigators in the field of the effects of radio waves interpreted their observations by resorting to a description of molecular processes in terms of displacement currents which rub the molecules against each other or produce mechanical stresses. Such interpretations just refer the problem of correct interpretation to a more complicated (if not impossible) one, namely, the understanding of quantum-mechanical events by classical descriptions.

With the advent of quantum-mechanical descriptions in the range of microwave frequencies, as put forward in the theory of microwave spectroscopy, a basis for interpretations of interactions of electromagnetic waves and chemical systems has become possible and an extension of such an analysis to short waves has assumed a less formidable character. In the course of the present project, the problem of investigating the action of 3-cm waves on unicellular organisms has been approached along these lines. Before undertaking such a task, it would be convenient to know that one has a definite goal, a biological effect that is produced by the exclusive action of electromagnetic waves of the frequency range under investigation. The

possibility of such an occurrence has been shown by the analytical approach that has been outlined above--the presence of absorption bands of complex molecules in the microwave region, its importance in the dynamical specificity of high molecular compounds, and its susceptibility to changes by incident radiation.

Apparent evidence that short waves and ultra-high frequencies produce nonthermal effects in unicellular organisms was found by various investigators: Fleming in the 1-20 m band,³ supported by earlier investigations by Haase and Schliephake;⁴ Nyrop;⁵ and Liebesny.⁶ They found that growth of micro-organisms increases under the stimulus of high frequency at a progressively faster rate with increase in power until a point of overstimulation is reached and lethal effects occur. This point differs widely depending on age, virulence, and extent of colonization of the organism under study. However, in this region a good analysis of the events by means of quantum mechanics seems rather far fetched and may be unnecessary in the long run, if the understanding of the chemico-physical laws comes from other types of investigations.

In the present project, the initial objective was a definite goal, a biological effect that is exclusively produced by the action of X band microwaves. A logical point to start would be to irradiate unicellular organisms at frequencies that produce resonance in the biochemical constituents of the cell; but, unfortunately, no such data were available. Instead, yeast cells in a suspension of buffer solution were irradiated and a change in the survival characteristics of the organism was sought--essentially a repetition of experiments performed at lower frequencies (with requisite methodological alterations). Because of the absorption peak of water in the 3-cm frequency range and the necessity for efficient cooling of the cells, a dilemma arose. Water was wanted, and at the same time was not wanted--but could not be avoided anyway, since drying the cells changes their dynamical or viable characteristics too much to leave them useful in a rigorous experiment. It proved possible to work out a compromise, however. The relative biological effect, i. e., survival of yeast cells, was compared in direct heating and in microwave heating. These experiments were performed under identical conditions, with the wattage per sample the same, and the temperature of the suspension, the flow rates, the water bath temperature, and many more parameters equal; and it was possible to observe an effect that appeared to be produced exclusively by the action of microwaves. The rate of survival of the cells treated with microwaves was lower than that of the cells treated with direct heat. All during the experiment a systematic error was suspected; this suspicion was confirmed by the results. Yeast cells are not killed in a water bath at 30°C but have optimum growth; how could this well-known fact be reconciled with the finding that about 60 per cent was killed at 30°C? The possibility of a point-heating effect came to mind but was found to be a false interpretation of Debye's equations, confirming the interpretation made by Higasi in a monograph published in Japan in 1950.⁷ Direct microscopic observation of the cells during exposure was the next step, and the apparently erroneous results were explained as follows. It was observed that the temperature gradient produced by dielectric absorption in the water causes the cells to coagulate along the boundaries of little vortices. The decrease in survival value is explained by

the fact that coagulated cells form the clones that are counted on the Petri dish instead of clones arising from individual single cells.

Why all this interest in the biological effects of 3-cm microwave radiation on unicellular organisms?

Since the single cell is the building material of all higher organisms, a rigorous investigation may start at this level and produce generalizations pertaining to the multitude of such cells. Another aspect presents itself for experimental investigation in the microwave range, one of much more fundamental character since it is not connected with the solving of any immediate problems. This is a better understanding of the functioning of the living cell by analysis of its mode of information transfer.

In his analytical study of the physical foundation of biology, Elsasser⁸ tackles one of the most fundamental problems in science: what is the difference between an organism and an automaton? He demonstrates that the modern theory of information has provided us with new tools for considering problems that have been set aside in the past by the serious investigator as too philosophical. He shows that the timidity of previous years must now give place to new vigor in an attack upon the fundamentals of biology.

Summarizing Elsasser's analysis, one might say that he compares two types of dynamical systems with each other: the artifacts known as automata, and living organisms. A vast area of functional behavior is found in which the theory of artifacts can indeed illuminate and explain the performance of organisms. Such an analysis was made in great detail by Rothstein.⁹ Elsasser, however, professes to recognize a radical difference. He notes the amazing lack of evidence for the existence of devices that would store information mechanistically, after the manner of storage components of electronic computers. A similar lack of evidence has been noted for the associated devices to be used in the distribution, writing in, reading out, sorting, and other operations on information, which in the view of the electronics engineer would have to be quite elaborate and conspicuous. Elsasser summarizes these experiences by enunciating the principle that organisms on the whole do not store information by mechanistic means.

Viewed as an automaton, the organism must have, in von Neumann's terminology,¹⁰ instruction for the proper break-up of environmental units, for the proper selection of the necessary components, and for their proper arrangement. From the viewpoint of information theory, the organism must have a sufficiently large information content. It must contain all the information necessary for the proper break-up of the environmental unit, the selection of proper units resulting from the break-up, and the building of a duplicate organism from those units.

Leaving aside the different (sometimes divergent) estimates of the information content of cells and organisms we can see right away that the information content must be very large, so that the organism must be sufficiently complex. These considerations put a lower limit on the complexity of even the simplest organisms.

If we consider the different kinds of atoms of which living organisms are made as elementary units, we find that this number is very limited--of the order of ten. Therefore, second-order units must be made from combinations of these elementary units. Such second-order units are the diverse kinds of molecules of which an organism is composed. This situation is quite analogous to the formation of a very large number of distinct words from a limited number of letters in the alphabet of a written language.

From the viewpoint of this analogy, the formerly mentioned problem becomes formally analogous to the following one. Given a written language, how large, in terms of the total number of words, must a book printed in that language be in order to contain complete information necessary to manufacture this book? The answer depends on the nature of the environment and on what is given for the manufacturing. If a complete linotype machine, bookbinding machine, paper, ink, etc., are available, the book need not be too large. If, however, only rough materials are available out of which one must make the paper, ink, machines, etc., then the size of the book will be beyond the practical limits of a single volume, and we shall be faced with the problem of reproducing not a single book but an entire library. In the case of the living organism, the situation is nearer to that of the second alternative, which accounts for the tremendous complexity of organisms.

The analogy between the information content of a written text and that of organic molecules has been pointed out by Branson¹¹ and Gamow.^{12, 13} The physically different atoms and their physically different aggregates play the role of letters and words.

Last year a French engineer, Polonsky, wrote an article¹⁴ in which he analyzed the functioning of a living cell by information theory, drawing on biochemical and biophysical data available. He presents the following picture: all the information necessary for adequate functioning of a living cell is stored in the DNA, the hereditary material of the cell that is the generator of information. The RNA is distributed throughout the cytoplasm of the cell and acts as an amplifier of the information, transmitting it to the effectors, which are enzymes and other proteins. The whole system is intricately coupled by feedback loops. The most important aspect for us, however, is the fact that he predicts, on the basis of biochemical and thermodynamical properties of the involved molecules, that this information transfer occurs by means of electromagnetic waves, modulated according to certain codes in various frequency ranges, depending on the desired action which is to be produced by means of this information transfer.

Elsasser has a similar idea about the functioning of the living cell. However, he treats the problem of noise obscuring the information transfer differently from Polonsky. He considers noise as a special form of input to the complex system of the functioning of the cell. If one does treat noise as an input it is indeed very difficult to explain information stability, the protection of circulating information from the progressive deleterious influence of noise. Applying these arguments to the coupling of enzymatic reactions with the substratum of protein molecules we see that over sufficient amounts of time the information corresponding to the structural details of these molecules will be communicated to the dynamics of the cell, to higher levels of

organization as it were, and may influence such dynamics. Polonsky solves this problem by assuming that the cell membrane acts as a very efficient noise filter, and that information transfer always occurs with very high redundancy. Elsasser arrives at the conclusion that the structure and behavior of organisms cannot be deductively derived from a few simple axioms or principles, as is the ideal of physics and as is asserted by Polonsky.

Stimulated by this controversy about the treatment of noise in a biological system, the attention of the present project was turned to the investigation of the cell membrane. If one can find that this structure can act as a filter for certain frequencies in the electromagnetic spectrum, a part of the argument will be resolved and a better understanding of the functioning of living cells will result.

The cell membrane is commonly assumed to consist of a dipole double layer, oriented radially between thin protein layers. The properties of such a structure have been investigated by impedance measurements. Some more or less satisfactory interpretations in terms of molecular structure have been advanced by analysis of data in the frequency range up to 10,000 Mc. The dielectric constant of the cell membrane has been estimated to be rather low--about 3. This value corresponds to a membrane thickness of about 35 Angstroms, a value considerably lower than established by other methods; however, the estimate of a dielectric constant of about 3 suggested by several authors, which would correspond to the properties of a lipid layer, is subject to criticism. The membrane is composed of lipid, protein, and water. The dielectric constant of protein at very high frequencies depends on the amount of bound water; the constant is certainly higher than 10. It may be argued that the impedance measurement detects only the lipid part of the membrane and, therefore, results in a smaller value of membrane thickness than are obtained by other techniques.

If one wants to analyze the cell membrane for its possible filtering action of electromagnetic waves, one has to proceed by assuming molecular models of the membrane structure and making an analysis of their behavior in the frequency range of interest. The filter would act by preventing certain bands of frequencies from reaching the cell interior. Since there is at present no reliable way of measuring field intensity inside a single cell, the approach to the problem by means of molecular models seems the only alternative.

Such an analysis necessitates knowledge of the behavior of proteins in aqueous solutions under the influence of electromagnetic waves, and of their interactions with each other and with other types of molecules, such as lipids.

The theory by Kirkwood and Shumaker,¹⁵ which explains the influence of dipole-moment fluctuations on the dielectric increment of proteins in solution, pertains only to static electric fields. If this theory is expanded to include dielectric dispersion and absorption, it is found that the relaxation time spectrum of an ellipsoidal molecule is determined not only by external rotatory diffusion but also by the diffusion of the mobile protons on the

surface of the molecule. This relationship means that the structural interpretation of dielectric dispersion is considerably more complex than for a molecule possessing a permanent dipole without fluctuation. Their analysis of the attractive force between proteins arising from fluctuations in charge and in configurations of mobile protons is necessarily schematic, because of lack of knowledge of the details of protein structure. Although it is a force of long range at low ionic strength, it appears to exhibit specificity only through the influence of structure on the fluctuations. In favorable orientations, steric matching of a constellation of basic groups on one molecule with a complementary constellation on the other could conceivably produce a redistribution of protons leading to a strong and specific attraction depending on the local structural details of the complementary constellations.

For an analysis of a possible filtering action of protein molecule arrangements it is important to note that except in highly acid solutions, the number of basic sites on the proteins usually exceeds the average number of protons bound to the molecule, so that many possible configurations of the protons exist, differing little in free energy, among which fluctuations may occur. An ordered arrangement of such proteins may very well act as a filter by virtue of resonances in the energy-level distribution of the protons that could be easily increased by splitting caused by the neighboring strong electric fields present in the lipid part of the membrane structure.

These speculations may point the way for disproof or proof of Polonsky's hypothesis, an objective that he has thoughtfully left to the biophysicists.

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STUDIES WITH 2450 MC-CW EXPOSURES
TO THE HEADS OF DOGS

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SUMMARY

Studies were conducted on dogs anesthetized with sodium pentobarbital and exposed to 2450 mc-CW irradiation for periods of one to seven hours employing a clinical microwave diathermy apparatus. The microwave energy was directed toward the top of the head with the antenna 5 cm from the skin and parallel to the midline of the calvarium. The field densities employed were estimated to be 0.5 to 0.8 watt/cm². Temperature measurements were made within the cranium, on the scalp, and in the rectum. The temperature of the cerebrospinal fluid in the cisterna magna appeared to rise more rapidly than the frontal or midbrain temperatures. Cerebrospinal fluid and serum transaminase levels were measured after exposure to microwave irradiation. Serum transaminase elevations were correlated with visible thermal damage to the scalp but cerebrospinal fluid transaminase levels did not suggest any appreciable brain tissue damage. Blood pressure, heart rate, cerebrospinal fluid pressure, respiratory rate, and rectal temperature measurements in dogs exposed to irradiation which had led to death after several hours in another group suggested a shock-like trend of events without evidence of central nervous motor excitation or convulsions. The primary heat-loss mechanism of panting was evident early in all exposures.

INTRODUCTION

The work with experimental exposure of dogs to microwaves in this laboratory has been oriented by two questions: (a) Are some deep-lying organs of the head or trunk relatively more vulnerable to microwave irradiation than others? and (b) What patho-physiological reactions are set up in organs and tissues as a result of the irradiation of limited areas of the head or trunk?

The results from earlier studies on microwave energy absorption in which it was found that the temperature of the contents of certain visceral organs was markedly increased¹ suggested that body tissues adjacent to such fluid-filled hollow viscera as the gall bladder, stomach, intestine, or urinary bladder might suffer thermal damage. Thus, in a recent series of investigations, the results of liver function tests were obtained following abdominal irradiation but, as previously reported,² no positive evidence of liver damage was found.

The present report gives results of head irradiation studies. The time-courses of temperature elevations in the cerebrospinal fluid of the cisterna magna, in the mid- and forebrain tissue, on the surface of the scalp directly in the path of the radiation, and in the rectum are described. A lethal head-irradiation pattern has included measurements of cerebrospinal fluid and blood pressures, heart and respiratory rates, and rectal temperatures in similarly irradiated dogs. Besides the usual visible signs referable to central nervous system involvement, a transaminase test sensitive to brain cell damage has been used in an effort to detect subtle sub-clinical brain tissue damage. Thus far the effects observed could be ascribed solely to the thermal effect of microwave irradiation.

METHODS

Healthy, mongrel dogs weighing 11 to 15 Kg were anesthetized with 35 mg/Kg of sodium pentobarbital and placed in a prone position on a light wooden frame. The head was supported by a gauze sling under the mandible. The rectangular director of a clinical microwave diathermy machine (Raytheon Microtherm, Model CDM10 with "C" director) was placed with its long axis parallel to the midline of the calvarium. In this position the antenna was 5 cm from the surface of the scalp and a near-field or, at most, cross-over field, was considered to exist at the scalp. Power densities calculated on this basis are approximations. Depending upon the percentage of the maximum power output of the diathermy unit used, the calculated field intensities at the scalp varied from 0.5 to 0.8 watt/cm².

Copper-constantin thermocouples were used for temperature measurements which were read out on a multichannel recorder (Brown Electronik). Intracranial temperatures were obtained by passing the thermocouples into the brain through small holes drilled in the skull. The cisterna magna temperature was obtained by passing a thermocouple through a hollow needle inserted between the first and second cervical vertebrae. Thus, simultaneous recording of temperature was obtained from the frontal lobe (thermocouple inserted through the frontal bone to one side of the midline), the mid-brain (thermocouple inserted from the side through the temporal bone) and the cisterna magna in close association with the medulla and roof of the fourth ventricle. Rectal temperatures were similarly obtained at a point about 6 cm inside the external anal sphincter. Skin temperatures were measured on the irradiated scalp surface. Temperature measurements were made with the microwave generator turned off.

Pressure transducers (Statham Laboratories) with amplifiers and recorders were used to measure spinal fluid pressure from the cisterna magna and arterial pressure from the femoral artery. Heart and respiratory rates were obtained from the blood pressure or spinal fluid pressure tracings.

Serum and spinal fluid transaminase levels were measured by an adaptation of the method of Reitman and Frankel.⁵ Serum and spinal fluid glutamic oxalacetic transaminase levels (GO-T) were measured using a prepared sub-

strate of alpha-ketoglutarate and L-aspartate (Sigma Chemical Company), then measuring the oxalacetate produced spectrophotometrically after development of an amber color with 2, 4- dinitrophenyl hydrazin and hydrochloric acid.

RESULTS AND DISCUSSION

The mildest irradiation study will be presented first. In this study the power output of the generator was monitored or varied to prevent, as nearly as possible, skin damage. Preliminary experiments have shown that about 42°C is a threshold of skin temperature above which, with the type of irradiation used, thermal damage to the skin will occur over periods of exposure of about two and one-half hours. Accordingly, 10 dogs were subjected to power densities averaging 0.5 watt/cm² at the scalp for 150 minutes. The purpose of this experiment was to determine the pattern of temperature changes at the three intracranial sites and at the relatively distant, but physiologically associated, site in the rectum. The results are shown in Table I.

TABLE I

TIME-COURSE OF TEMPERATURES AT VARIOUS SITES IN TEN DOGS SUBJECTED TO IRRADIATION OF THE HEAD WITH MICROWAVE ENERGY MONITORED TO PREVENT EXCEEDING 42°C AT THE SCALP.

| Time Minutes from Onset of Exposure | Temperature, °C | | | | |
|---|-----------------|-----------------|---------------|-------------------|--------|
| | Scalp | Frontal Lobe | Mid- Brain | Cisterna Magna | Rectum |
| Control | 34.1 | 38.4 | 38.5 | 38.2 | 38.1 |
| 10 | 40.0 | 39.1 | 39.3 | 40.1 | 38.7 |
| 20 | 40.1 | 39.6 | 39.6 | 40.2 | 39.1 |
| 30 | 40.3 | 39.9 | 40.0 | 40.3 | 39.4 |
| 40 | 40.9 | 40.2 | 40.3 | 40.7 | 39.7 |
| 50 | 41.0 | 40.3 | 40.4 | 40.8 | 40.0 |
| 60 | 41.0 | 40.6 | 40.4 | 40.9 | 40.1 |
| 70 | 41.1 | 40.6 | 40.6 | 41.3 | 40.3 |
| 80 | 41.4 | 40.7 | 40.7 | 41.1 | 40.4 |
| 90 | 41.5 | 40.8 | 40.7 | 41.3 | 40.5 |
| 100 | 41.6 | 40.9 | 40.9 | 41.3 | 40.6 |
| 110 | 42.2 | 40.9 | 41.0 | 41.4 | 40.7 |
| 120 | 42.0 | 40.9 | 40.9 | 41.4 | 40.9 |
| 130 | 41.9 | 41.0 | 41.0 | 41.4 | 40.9 |
| 140 | 42.3 | 41.0 | 41.1 | 41.5 | 41.0 |
| 150 | 42.5 | 41.0 | 41.1 | 41.6 | 41.1 |

As seen in this table, the temperature was increased more rapidly in the cisterna magna than in any of the other sites where it was measured with exception of the skin. Panting began within the first ten minutes of irradiation. Although the temperatures measured in the brain, cistern, and rectum were initially similar, the temperature in the cistern was increased more rapidly and to a greater extent than in the other locations during the irradiation.

As might be expected the experimental procedure just described had no serious effect upon the animals beyond the hyperthermia and minor skin damage to the scalp. It was suggested by these findings that the relatively slowly circulating fluid within the cisterna magna accumulated heat more rapidly than the other brain tissue sites which represented well-vascularized tissue.

With the thought that perhaps brain cells in relatively close approximation to the fluid-filled ventricles of the brain or to the subdural fluid-filled areas such as the cisterna magna might suffer damage at temperatures similar to or greater than those of the first experiment, a second more severe heating regimen was used. In this experiment 12 dogs were exposed to the maximum output of the generator for one hour and 6 dogs were exposed to the same output for two hours. Cerebrospinal fluid glutamic-oxalacetic transaminase (GO-T) was measured in all dogs at 12-hour intervals from immediately after the irradiation period to 168 hours later. Only the results in the longer exposure will be presented since even in these the evidence for subtle brain cell damage was essentially negative as far as the chemical test chosen was concerned. Table II summarizes this part of the experiment. In this table are shown the scalp and rectal temperature time courses during the irradiation period. The control levels of transaminase in both the serum and cerebrospinal fluid were obtained prior to irradiation. The serum transaminase was increased in accordance with the thermal damage to the scalp which was apparent visually. The lack of a clinically significant rise in cerebrospinal fluid transaminase was interpreted as indicating that not only did damage to brain cells probably not occur, but the blood brain-barrier to this particular enzyme remained intact and allowed essentially no leakage from the blood, where the enzyme was abundant, into the cerebrospinal fluid.

Another series of 17 dogs was subjected to maximum output of the generator until death resulted. The time of irradiation in this group varied from 3 to 7 hours. In all of these animals the scalp and rectal temperatures were measured at 10-minute intervals during the irradiation. In seven of the animals, frontal, mid-brain, and cisterna magna temperatures were also recorded. In most cases the temperatures measured in the various sites soon exceeded 45°C except for the rectal temperature. The upper limit of the recording potentiometer was 45°C. Table III shows the irradiation time which was lethal in each case, together with the terminal rectal temperatures and the increment of each rectal temperature above the control value. Also shown are the weights of the animals. There appears to be no high correlation between the weight, the final rectal temperature increase, and the lethal irradiation time. Whether a high correlation exists

TABLE II

TIME-COURSES OF TEMPERATURES DURING IRRADIATION,
AND SIGMA-FRANKEL UNITS/ML OF GLUTAMIC OXALACETIC TRANSAMINASE (GO-T)
AFTER IRRADIATION IN DOGS SUBJECTED TO APPROXIMATELY 0.8 WATT/CM²
MICROWAVE IRRADIATION OF THE HEAD.

| Time (min.) | During Exposure Period Temperature, °C | | | Time (hrs.) | Post-Exposure Period | | | |
|----------------|---|-------|--------|----------------|----------------------|------|-----------|------|
| | N | Scalp | Rectum | | Serum | | G-S Fluid | |
| | | | | | N | GO-T | N | GO-T |
| Control | 6 | 33.7 | 38.0 | Control | 5 | 17 | 5 | 9 |
| 10 | 6 | 39.8 | 38.8 | 0 | 5 | 20 | 5 | 12 |
| 20 | 6 | 40.2 | 39.2 | 12 | 5 | 105 | 4 | 18 |
| 30 | 6 | 40.4 | 39.6 | 24 | 5 | 117 | 4 | 26 |
| 40 | 6 | 40.7 | 39.8 | 36 | 5 | 93 | 3 | 19 |
| 50 | 6 | 41.0 | 40.0 | 48 | 4 | 59 | 3 | 20 |
| 60 | 6 | 41.0 | 40.1 | 60 | 4 | 38 | 4 | 20 |
| 70 | 6 | 41.4 | 40.2 | 72 | 5 | 39 | 5 | 16 |
| 80 | 6 | 41.9 | 40.4 | 84 | 5 | 24 | 5 | 12 |
| 90 | 6 | 41.9 | 40.5 | 96 | 3 | 23 | 4 | 14 |
| 100 | 6 | 43.4* | 40.7 | 108 | 3 | 18 | 3 | 14 |
| 110 | 6 | 43.4* | 40.8 | 120 | 5 | 20 | 5 | 17 |
| 120 | 6 | 44.4* | 41.0 | 132 | 5 | 22 | 5 | 19 |
| | | | | 144 | 4 | 20 | 5 | 19 |
| | | | | 156 | 5 | 18 | 5 | 16 |
| | | | | 168 | 5 | 25 | 5 | 18 |

* Some individual values exceeded the maximum of 45°C which could be measured with the recording potentiometer.

TABLE III

LETHAL IRRADIATION TIMES AND CORRESPONDING TERMINAL
RECTAL TEMPERATURES AND FINAL INCREMENT OF
RECTAL TEMPERATURE OVER CONTROL VALUES.

| Weight (Kg) | Lethal Irradiation Time (min.) | Terminal Rectal Temp. (°C) | Increment over Controls (°C) |
|----------------|--------------------------------------|----------------------------------|------------------------------------|
| 14.0 | 150 | 43.8 | 5.5 |
| 14.5 | 190 | 44.2 | 6.2 |
| 12.3 | 210 | 44.4 | 6.8 |
| 11.5 | 230 | 43.2 | 4.6 |
| 11.0 | 260 | 44.1 | 6.7 |
| 10.8 | 270 | 42.4 | 4.9 |
| 11.2 | 280 | 44.2 | 7.1 |
| 12.8 | 310 | 42.8 | 4.4 |
| 14.5 | 380 | 43.5 | 6.8 |
| 12.2 | 400 | 43.9 | 6.8 |

between any of the other temperatures measured and the time required for lethal termination cannot be stated since the upper limit of the recording potentiometer was exceeded in this study. The cisterna magna temperatures were increased more rapidly than those of the other two intracranial sites before reaching the upper limit of the temperature recorder.

A final series of irradiations was carried out in 2 dogs. This was performed in the same manner as the group summarized in Table III except that attention was directed to records of the femoral arterial blood pressure, the spinal fluid pressure, the respiratory and heart rates, and the rectal temperature. This attempt to characterize the mechanism of the fatal terminations seen above was partially frustrated by the fact that rectal temperatures in both of the latter dogs did not reach the levels of the previous group (Table III) and neither of the animals succumbed to the irradiation regimen carried out for six and a half hours. The data from these experiments have been abbreviated to show control and half-hour values for each dog instead of the detailed 10-minute interval data that were recorded. These time courses are shown in Table IV. Although the irradiation regimen was not lethal, the animals had begun to exhibit some evidence of cardiovascular embarrassment near the end of the exposure period. Evaluation of the significance of measured biological variables during an experimental regimen of this kind requires knowledge of the effect of the anesthetic employed upon the respective variables.

It has been shown that sodium pentobarbital causes a depression of respiration, essentially no effect on blood pressure except for an initial depression lasting only a few minutes, and an increase in heart rate.⁴ In

TABLE IV

TIME COURSES OF RECTAL TEMPERATURE, ARTERIAL PRESSURES,
CEREBROSPINAL FLUID PRESSURES, AND HEART AND RESPIRATORY RATES
IN TWO DOGS (A AND B) RECEIVING MICROWAVE IRRADIATION TO THE HEAD
AT A CALCULATED POWER DENSITY OF 0.1 WATT/CM².

| Time (hrs.) | Rectal T. °C | | Pressures (mm Hg) | | | | Rates per min. | | |
|----------------|-----------------|---|-------------------|---|-----------|---|----------------|-------|-----|
| | A | B | Systolic | | Diastolic | | C-S-F A | Heart | |
| | | | A | B | A | B | | A | B |
| 0 | 38.2 | | 180 | | 128 | | 7.0 | 180 | 12 |
| | 38.3 | | 170 | | 115 | | 12.0 | 156 | 4 |
| .5 | 39.0 | | 205 | | 145 | | 6.5 | 180 | 14 |
| | 39.1 | | 183 | | 128 | | 8.0 | 120 | 4 |
| 1.0 | 39.6 | | 195 | | 145 | | 4.2 | 180 | 37 |
| | 39.8 | | 186 | | 131 | | 7.5 | 104 | 6 |
| 1.5 | 40.1 | | 208 | | 150 | | 6.5 | 180 | 108 |
| | 40.5 | | 190 | | 133 | | 4.8 | 138 | 15 |
| 2.0 | 40.2 | | 190 | | 138 | | 6.0 | 198 | 140 |
| | 40.5 | | 190 | | 142 | | 11.0 | 156 | 34 |
| 2.5 | 40.5 | | 190 | | 143 | | 6.5 | 198 | 100 |
| | 41.0 | | 200 | | 142 | | 12.5 | 156 | 12 |
| 3.0 | 41.2 | | 200 | | 142 | | 4.5 | 180 | 108 |
| | 41.5 | | 200 | | 142 | | 11.0 | 162 | 52 |
| 3.5 | 41.2 | | 200 | | 155 | | 4.5 | 222 | 140 |
| | 41.6 | | 200 | | 150 | | 11.0 | 198 | 82 |
| 4.0 | 41.1 | | 178 | | 135 | | 4.5 | 198 | 152 |
| | 41.5 | | 190 | | 142 | | 8.5 | 198 | 82 |
| 4.5 | 41.0 | | 175 | | 142 | | 6.0 | 198 | 164 |
| | 41.8 | | 213 | | 165 | | 10.5 | 198 | 14 |
| 5.0 | 41.9 | | 203 | | 142 | | 4.5 | 105 | 92 |
| | 42.3 | | 213 | | 165 | | 15.0 | 240 | 104 |
| 5.5 | 42.1 | | 190 | | 133 | | 6.3 | 120 | 96 |
| | 42.7 | | 195 | | 142 | | 14.0 | 258 | 124 |
| 6.0 | 42.3 | | 165 | | 138 | | 8.0 | 240 | 136 |
| | 42.2 | | 178 | | 130 | | 12.0 | 288 | 136 |
| 6.5 | 42.2 | | 155 | | 120 | | 8.5 | 255 | 136 |
| | ---- | | 155 | | 120 | | ---- | 250 | 96 |

the above experiments (Table IV) the rapid heart rate measured during the entire irradiation period probably resulted, in part at least, from the effect of the sodium pentobarbital anesthesia maintained throughout the period. The decrease in blood pressure near the end of the exposure can be attributed almost entirely to an effect resulting from the irradiation. The decrease in blood pressure without a concomitant decrease in heart rate suggests a decrease in cardiac output, a net vascular dilation, a reduced blood volume, or elements of all three factors. This shock-like trend occurred without any visible sign of central nervous motor excitation of a convulsive nature.

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Effects of Environmental Temperature and Air Volume Exchange
on Survival of Rats Exposed to Microwave Radiation
of 24,000 Megacycles

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Experimental

Female albino rats of Osborne-Mendel stock were used, with the exception of the control rats in the third experiment. These were Sprague-Dawley animals. All rats were housed in air-conditioned quarters ($24^{\circ}\text{C} \pm 1^{\circ}\text{C}$). They were exposed individually to microwave energy in an absorbent-lined, 1130 liter chamber equipped with a 10 db standard horn antenna directed down upon an animal. During an exposure, a rat was restrained in the prone position by a Plexiglas holder described in detail elsewhere. (1). The animal was prevented from turning on its long axis by taping its tail (scotch tape) to the holder. This was important in directing the exposure solely to the lumbar area. In all experiments, exposure was kept constant at 250 mw/cm^2 . Rectal temperatures were determined by a mercury thermometer, and by a flexible plastic-covered probe connected to a thermistor thermometer bridge with a range from 35° to 46°C .

Presented by Wm. B. Deichmann at the Third Tri-Service Conference on Biological Hazards of Microwave Radiation, Berkeley, California, August 25, 26, 27, 1959.

The investigation was sponsored by the Rome Air Development Center Air Research and Development Command, United States Air Force.

Results

Three experiments were conducted. In the first, all rats were exposed continuously until death to determine the relationship between environmental temperature and period of survival. This was found to be as follows:

| <u>No. of Rats Exposed</u> | <u>Temperature in Exposure Chamber</u> | <u>Mean Period of Survival</u> |
|----------------------------|--|--------------------------------|
| | $^{\circ}\text{C}$ | min |
| 10 | 35 ± 0.5 | 17.4 (16.5 to 19 min) |
| 10 | 30 ± 0.5 | 21.0 (18 to 23 min) |
| 10 | 25 ± 0.5 | 26.8 (23 to 30 min) |
| 10 | 20 ± 0.5 | 36.3 (30 to 46 min) |
| 10 | 15 ± 1.0 | 47.0 (40 to 63.5 min) |

The effect of the environmental temperature was reflected in the rates of rise, but not on the maximum rectal temperatures produced. (Table 1). At death, the local effects in the lumbar area were similar, whether rats suffered exposure at environmental temperatures at 15°C or 35°C . The changes ranged from first to third degree burns.

In a second experiment, rats were exposed intermittently until death. In this study, the current was ON for 1 minute and OFF for three minutes. One group of animals (3 per group) was exposed at a chamber temperature of 30°C , the second group at 26.5°C , and the third group at 23°C . The data demonstrate again that the period of survival is markedly influenced by the environmental temperature at which microwave exposure occurs. (Table 2).

The period of survival of a rat depends largely on the animal's ability to eliminate or to compensate for the microwave heat energy absorbed. The lower the environmental temperature (within the limits investigated), the more effective was the degree of compensation.

It was speculated that the period of survival might be further increased by blowing air onto an animal. In the following study, experimental conditions were the same as those described in the first experiment in which the environmental temperature was maintained at 15°C, except that in this case, a blower* was installed in the exposure chamber, circulating the air and blowing it onto the experimental and the control rat. The control rat was placed in such a position that it was subjected to a similar degree of cooling, but not to the effects of microwaves.

As stated above, when exposed continuously to microwave radiation at 15°C, rats died in 47 minutes (40 to 63.5 min) with rectal temperatures ranging from 42.5 to 45.2°C. Rats were found to live considerably longer when benefited by air from the blower. The periods of survival of eight rats ranged from 14 to 24 hours.

One animal (D-1289) died after eight hours of exposure with its heart in diastole. This rat might be excluded since the ventricles of all other rats were found to be well contracted. The auricles were uniformly dilated and filled with large clots of blood. Another rat (D-1084) survived 24 hours of exposure, at

*Blower, 1/15 H.P., manufactured by the Dayton Electric Mfg. Co., Dayton, Ill. Outlet velocity 3230 ft/min, volume 140 cu ft/min at zero pressure.

which time the experiment was discontinued. Control animals subjected to the same experimental conditions but not to microwave energy survived for 12 to 21 hours. The rectal temperatures of the rats dropped immediately when they were placed in the exposure chamber, but the temperatures of the control rats - not benefited by radar - dropped more precipitously than those of the experimental animals. (Table 3).

Discussion

The mechanisms controlling the body temperature of the rat and man are not alike. A rat seems to lack sweat glands, and it should therefore lose water by a mechanism different from that utilized by man. However, it is well known that when a rat is maintained in a stagnant, hot and humid atmosphere, that its fur becomes quite wet. To Sundstroem (2), this implies "that the rat, in addition to the insensible water loss, is capable of covering its surface with a film of water, (this), physiologically, is the crucial point." Sundstroem believes that irrespective of any histological differences, the physiological function of the skin of man and small rodents, in regard to water elimination, may be comparable in a qualitative sense.

The relative humidity was recorded in all of our experiments. Since the values ranged from 60 to 85 per cent, it must be assumed that this factor influenced an animal's ability to maintain thermal homeostasis. Future studies will be designed to investigate this factor in detail.

It was possible to prolong the life of a rat exposed continuously to

microwaves from 17 minutes to more than 17 hours by introducing an effective air volume exchange and by lowering the environmental temperature. Because of the marked effect of these two factors, it is believed that a Maximum Safe Exposure Level of 10 mw/cm² is no longer acceptable without also defining temperature and air speed.

Man enjoys an excellent heat regulating system, this, coupled with intermittent exposures, and what we may have learned from this experiment, may give us some explanation why human discomfort or systemic injury from microwaves has been nonexistent for practical purposes. Certain accepted facts relating to effects by radar may need modification, unless air speed and volume, and environmental temperature were considered and recorded at the time the experiment was conducted.

Conclusions

1.) The environmental temperature was found to influence considerably the systemic effects of microwave radiation of 24,000 megacycles. The period of survival of a rat was more than doubled (from 17.4 to 47.0 min) by a drop in environmental temperature from 35°C (95°F) to 15°C (59°F). A most remarkable prolongation of life was brought about by an effective air volume exchange. Rats exposed continuously to 250 mw/cm² at 15°C lived for 47 minutes (40 to 63.5 min). Rats exposed similarly but aided by air (15°C) from a blower in losing microwave-induced heat energy survived for 14 to 24 hours.

2.) Local effects were influenced similarly. However, at death, the severity of local damage (first to third degree burn) was essentially the same, whether a rat was exposed for 17 minutes at 35°C, or for 20 hours at 15°C when benefited by circulating air.

3.) While the temperature control of the rat and man differs, the physiological mechanisms are sufficiently alike to postulate that local and systemic effects of microwave radiation are least likely to occur in man if he is exposed under conditions of optimal ventilation and low environmental temperature.

Acknowledgement:

The assistance of Mr. William E. Pate is gratefully recognized.

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Table 1

Effect of Environmental Temperature

on the

Rectal Temperature of the Rat

Continuous Exposure to 250 mw/cm² Until Death

(The table gives the highest rectal temperature recorded for (three) rats. Ten rats were exposed individually per group. Rats were in prone position, exposure directed to dorsal area)

| 15°C | <u>Environmental Temperature</u> (Air Speed Essentially Zero) | | | 35°C |
|------|--|------|------|------|
| | 20°C | 25°C | 30°C | |
| | <u>Rectal Temperatures in °C</u> | | | |
| 43.5 | 43.8 | 45.2 | 45.6 | 45.6 |
| 43.6 | 46.0 | 45.7 | 45.8 | 45.6 |
| 45.2 | 46.0 | 46.0 | 46.0 | 46.2 |

Table 2

Effect of Environmental Temperature

on

Period of Survival and Rectal Temperature of the Rat

Intermittent Exposure, 1 min ON, 3 min OFF to 250 mw/cm²

(There were three rats per group)

| <u>Environmental Temperature</u> | <u>Experimental Period</u> | <u>Actual Exposure Time Until Death</u> | <u>Mean Rectal Temperature at Death</u> |
|----------------------------------|----------------------------|---|---|
| | min | min | °C |
| 30.0°C | 120 | 31 | 43.4 |
| 26.5°C | 335 | 85 | 42.2 |
| 23.0°C | 480 | 120 | 39.6 |

Table 3

Effect of Continuous Exposure of Rats to 250 mw/cm²
at an Environmental Temperature of 15°C
and While Cooled in Addition by
a Stream of Air from a 1/15 H.P. Blower

(Rats were exposed individually. They were in the prone position,
 exposure was directed to the lumbar area)

| <u>Experimental Conditions</u> | <u>Ident. No. of Rat</u> | <u>Periods of Survival</u> hours | <u>Rectal Temperatures at Death</u> °C |
|--|--------------------------|-------------------------------------|---|
| 15°C Exposure to 250 mw/cm ² Blower | D-1289* | 8 | |
| | D-1093 | 14 | 30 |
| | D-1095 | 16 | 26 |
| | D-1091 | 16 | 30 |
| | D-1086 | 18 | |
| | D-1296 | 20 | 29 |
| | D-801 | 20 | 20 |
| | D-1292 | 24 | 24 |
| | D-1294 | 24 | 27 |
| | D-1084 | discont. after 24 | 29 when discont. |
| 15°C NO EXPOSURE Blower | D-1291 | 12 | 15.5 |
| | D-1288 | 14 | 15 |
| | D-1094 | 15 | 15 |
| | D-1090 | 15 | 15 |
| | D-1293 | 15 | 15 |
| | D-1087 | 15 | 16 |
| | D-1092 | 16 | 16 |
| | D-802 | 16 | |
| | D-1085 | 21 | 14.5 |
| | D-1295 | 21 | 15.5 |

*This rat died with heart in diastole.

NOTE: Ten rats exposed at 15°C (continuously to 250 mw/cm²) without benefit of ventilation from a blower died in 36 to 63 minutes with rectal temperatures of 43 to 46°C.

The Effect of Repeated Microwave Exposures

on the

Formed Elements in the Blood of Rats

Willard Machle, M.D., and Karin Landeen
Department of Pharmacology
University of Miami School of Medicine

(Presented by Wm. B. Deichmann)

Four adult male rats were exposed (together) to from 6 to 10 mw/cm² for one to three hours per day until they had received a total of 196 hours of exposure on 85 days over a total period of 119 days.

The temperature in the exposure chamber may have fluctuated from 21 to 26°C. Daily records were not taken. Blood counts conducted once every two weeks, immediately before an exposure, revealed no abnormalities in regard to hemoglobin, reticulocytes, hematocrit and the number of total and differential leukocytes.

Results of Studies of Microwave Radiation

by the

Department of Pathology
University of Miami School of Medicine

(Presented by Wm. B. Deichmann)

Dr. S. A. Gunn and Dr. T. C. Gould of our Department of Pathology, have been engaged, for some time, in studies on the rat prostate. These investigators have shown that the amount of zinc which is taken up by the dorsolateral prostates of the rat is under hormonal control, and is an index of the functional state of the gland, as well as a sensitive indicator of the level of male hormone in the circulation.

It is my pleasure to report on a series of experiments which they conducted to determine whether or not exposure of the scrotum and testes to microwaves would induce testicular or other damage that would manifest itself in an altered prostate function.

Wistar rats (20 per group) were used. They were anesthetized with ether and the scrotal area was subsequently exposed to 300 mw/cm². One group was exposed for 15 minutes, one group for 10 minutes, and another group for 5 minutes. One day prior to sacrifice, each of the rats was given an intracardial injection of tracer doses of Zn⁶⁵. Twenty-four hours later the animals were sacrificed and the dorsolateral prostates were removed for weighing and determination of Zn⁶⁵ uptake.

Results

Slide 1 shows the linear relationship between temperature within the testes and exposure time.

Slide 2. In the 15-minute exposure group there is a moderate fall in the weight of the prostate and a marked and significant depression of Zn^{65} uptake. In the 10-minute exposure group, the weight of the prostate is only slightly diminished, but the Zn^{65} uptake is again significantly depressed. In the 5-minute exposure group, in which the weight of the prostate is unaltered, there is again a significant depression of Zn^{65} uptake.

Pathological changes: Five days after 15 minutes of exposure, there were still extensive third degree burns of the scrotal skin. (Slide 3). The testes showed many opaque areas, hemorrhage and collapse. There was extensive coagulation necrosis of the seminiferous tubules and interstitial and vascular tissues.

In the 10 minute exposure group, the damage was somewhat less marked.

In the 5 minute exposure group (sacrificed 6 days after exposure), there was no damage to the scrotal skin. The testes were slightly enlarged, but (Slide 4) there was still moderate to severe edema. Most of these testes showed no damage in the tubules and interstitial tissues.

(Slide 5). Thirteen days after exposure the testes were normal except for mild edema.

Summary

These studies have shown that minimal exposure to microwaves, that is five minutes of exposure to 300 mw/cm^2 produced

- 1) no damage to the scrotal skin
- 2) slight enlargement of the testes
- 3) a moderate degree of edema of the testes
- 4) no change in the weights of the dorsolateral prostates of the rat, but,
- 5) a significant drop in the uptake of Zn^{65} by the prostates.

In conclusion, these studies have shown that exposure inducing only slight enlargement, plus a moderate degree of edema of the testes, is associated with a lowered Zn uptake and hence with a subnormal level of male hormone.

Certain pilot studies were conducted by Dr. Robert Tallarico
and John Ketchum of the Department of Psychology, University of Miami

(Presented by Wm. B. Deichmann)

The effect of microwaves was tested on a certain behavior pattern of the rat.

Before exposure, a number of hooded rats had been trained in a modified Skinner Box to press a lever in order to obtain a pellet of food. During this training period, each rat was permitted to receive a total of 15 grams of food per day. The rats (prone position) were then exposed individually (still air; $23^{\circ} \pm 2^{\circ}\text{C}$) on each of three succeeding days to 109 mw/cm^2 for 15 or 30 minutes. Immediately after an exposure, a rat was placed into the Skinner Box to determine its ability and desirability to press the lever.

Results: After exposure, most of the rats showed an increase in latent period and a decrease in the frequency with which they would depress the lever. These effects became particularly marked after the third exposure. At this time, some rats showed no response at all.

On the fourth day, the animals were placed on standard diet, and for one month, they had free access to food and water.

At the end of this period, they were returned to the experimental pellet diet of 15 grams per day, and after four additional days, they were placed again into the Skinner Box. At this time, all rats displayed again immediately the lever-pressing response.

In conclusion, this study has demonstrated that exposure to microwaves can induce changes in the behavior pattern of the rat. In the case of these animals, complete recovery occurred. The experiment will be continued.

Relation of Interrupted Pulsed Microwaves to Biological Hazards

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Experiments with rats indicate that by choosing a certain cycle for interruption of transmission, it is possible to reduce the hazard from microwave exposure.

In our studies a pulsed magnetron from radar gear was used to generate microwaves with a frequency of 24,000 megacycles or a wave length of 1.25 cm. The duty cycle is 0.0006 or 0.06%. The peak output was 40,000 watts, but because of the duty cycle, experiments were conducted with an average power output of 21 watts.

In a previous communication (1), we concluded that intermittent exposures to microwaves emphasized the potency or prolonged action of this form of energy more than did a continuous exposure. Continuous exposure (300 mw/cm², rat, prone position, total body exposure) killed a rat in 15 minutes; intermittent exposure to 50% of this energy per unit of time (one minute of exposure followed by one minute of nonexposure, etc.) over a period of 31 minutes, killed in 16 minutes of actual exposure time. Seventeen percent

Presented at the Microwave Investigator's Conference, Patrick Air Force Base, Patrick, Florida, January 15 and 16, 1959, and at the Third Tri-Service Conference on Biological Effects of Microwave Radiating Equipment, in Berkeley, California, August 25 - 27, 1959.

of the above microwave energy per unit of time (exposure: one minute followed by five minutes of nonexposure, etc.) killed rats in 34 minutes of actual exposure time. These and related data indicated that besides species, power density and other factors, microwave radiation effects depend largely on the period of exposure and the recovery period after exposure.

It was considered possible to influence further the effects of microwave exposure by altering the exposure and nonexposure times (in secs), but by keeping their ratio constant.

In the first group of experiments a ratio of 1:1 was maintained. For instance, instead of exposing an animal for 60 seconds, followed by a non-exposure period of 60 seconds, exposure and nonexposure periods were each kept equal to 30, 15, 5 and 3 seconds, respectively. In subsequent experiments, the ratios of time on to time off were 1:2, 1:3, 1:4 and 1:5 (Figure 1).

Table 1 presents the data. These demonstrate that the period of survival of an animal increases as the period of exposure decreases, in spite of the fact that the actual period of exposure per unit period of time remains constant. To quote certain experiments conducted with a 1:3 ratio, exposure for 15 seconds followed by a nonexposure period of 45 seconds, or 15 seconds of exposure over 60 seconds, killed all four rats in 55 (from 50 to 60) minutes of actual exposure time; while exposure for three seconds followed by a nonexposure period of nine seconds, also adding up to 15 seconds of exposure over a 60 second period, killed two rats in 105 and 115 minutes, with two surviving. (The experiment was discontinued after 120 minutes of actual exposure over

a period of eight hours.)

Subsequent to the publication of this report (2), it was discovered that environmental temperature and air currents play a most significant role in the effects produced by microwaves. It is for this reason that some of these experiments were repeated under what we now consider, "controlled" conditions. Table 2 presents the data.

Conclusions

By choosing a certain cycle for interruption of transmission — without altering the output of microwave energy per unit of time — it is possible to reduce the hazard from microwave exposure. The significance of these observations is obvious. Although only one frequency was used, it seems reasonable to assume that this finding will also apply to other wave lengths of either pulsed or CW microwaves.

References

1. "Acute Effects of Microwave Radiation on Experimental Animals (24,000 Megacycles)." Wm. B. Deichmann, F. H. Stephens, Jr., M. Keplinger, and K. F. Lampe.
J. of Occupational Med. 1, 369-381, 1959.
2. "Relation of Interrupted Pulsed Microwaves to Biological Hazards."
Wm. B. Deichmann, M. Keplinger, and E. Bernal.
Ind. Med. & Surgery 28, 212-213, 1959.

Table 1

Effect of Intermittent Exposure to Microwaves (24,000 MC, 1.25 cm)
on the Mortality of Rats

(Rats (200 to 275 g) immobilized in Plexiglas and screen holder; exposure of lumbar region; four rats per group; power density 300 mw/cm²; half power diameter 6.35 cm)

| <u>Period of Time (sec)</u> <u>Transmitter was</u> | | <u>Ratio of</u> <u>Exposure to</u> <u>Nonexposure</u> | <u>Time "ON"</u> or <u>Lethal Exposure Time</u> min | <u>Mean</u> min |
|---|-------|---|--|--------------------|
| "ON" | "OFF" | | | |
| 60 | 60 | 1:1 | 15, 15, 18, 18 | 16.5 |
| 30 | 30 | | 16, 18, 19, 19 | 18 |
| 15 | 15 | | 25, 27, 28, 32 | 28 |
| 5 | 5 | | 26, 28, 28, 30 | 28 |
| 3 | 3 | | 35, 37, 38, 52 | 40 |
| 30 | 60 | 1:2 | 35, 37, 37, 47 | 39 |
| 15 | 30 | | 45, 53, 55, 62 | 54 |
| 10 | 20 | | 60, 63, 68, 69 | 65 |
| 5 | 10 | | 72, 77, 83, 96 | 82 |
| 3 | 6 | | 90, 95, 96, 100 | 95 |
| 60 | 180 | 1:3 | 18, 25, 32, 36 | 28 |
| 30 | 90 | | 45, 51, 52, 56 | 51 |
| 15 | 45 | | 50, 55, 56, 60 | 55 |
| 10 | 30 | | 72, 75, 76, 80 | 76 |
| 5 | 15 | | 90, 110; 2 survived 120 min* | 100 |
| 3 | 9 | | 105, 115; 2 survived 120 min* | 110 |
| 1 | 3 | 3 survived 120 min* | 118 | |
| 30 | 120 | 1:4 | 65, 69, 71, 75 | 70 |
| 25 | 100 | | 3 survived 100 min* | 85 |
| 20 | 80 | | all survived 100 min* | |
| 15 | 60 | | all survived 100 min* | |
| 60 | 300 | 1:5 | 26, 33, 37, 40 | 34 |
| 45 | 225 | | 3 survived 80 min* | 69 |
| 30 | 150 | | all survived 80 min* | |
| 15 | 75 | | all survived 80 min* | |
| 10 | 50 | | all survived 80 min* | |

*Actual exposure time

Table 2

Effect of Intermittent Exposure to Microwaves (24,000 MC, 1.25 cm)
on the Mortality of Rats

(The lumbar areas of immobilized female rats (232 to 264 g) were exposed to 300 mw/cm² at a room temperature of 24 ± 1°C in still air. The relative humidity ranged from 60 - 80%)

| <u>Period of Time (sec)</u> <u>Transmitter was</u> | | <u>Ratio</u> | <u>Lethal Exposure Time</u> or <u>Time "ON"</u> min | <u>Mean</u> min |
|---|-------|--------------|--|------------------------|
| "ON" | "OFF" | | | |
| 60 | 60 | 1:1 | 16, 16, 17, 17, 17, 18, 18, 19, 19, 20, 20 | <u>17.9</u> |
| 3 | 3 | 1:1 | 27, 34, 35, 35, 36, 37, 38, 38, 39, 42 | <u>36.1</u> |
| 60 | 120 | 1:2 | 29, 30, 30, 33, 33.7, 35, 37, 39, 42, 42 | <u>35.1</u> |
| 3 | 6 | 1:2 | 72, 80, 83, 85, 87, 88, 90, 92, 93, 97 | <u>86.7</u> |

SOME EFFECTS OF ULTRA-HIGH FREQUENCY ENERGY
ON PRIMATE CEREBRAL ACTIVITY

Sven A. Bach, M.D., Maitland Baldwin, M.D., and Shirley Lewis, R.N.

The clinical effects of irradiation of monkey's heads with u.h.f. of about 390 megacycles have been described previously by Baldwin. The purpose of this presentation is to summarize those findings, to describe additional clinical and pathologic findings and to attempt to relate them to the physical exposure conditions.

The exposure conditions of these experiments are somewhat unusual, since the basic purpose was to get the energy into the head where it would produce an observable effect, rather than to enable us to generalize about exposure to this form of energy as a hazard.

For this reason, our exposures were all partial, restricted to the head, and took place at the bottom center of a cylindrical copper screen chamber, designed so as to resonate in the $TM_{0,1}$ mode at the frequency employed. Since the free-space wave length of 388 mc is about 77 cm, the cavity resonator took on a height of 77 cm and a radius of 29.5 cm. The original chambers were quite crude (Slide 1) but, as you can see, were paired so as to enable simultaneous observation of the control and the exposed animals. Later, with the addition of a sampling probe, the exposure conditions became more refined. (Slide 2)

It is seen that the animal's head is held in a plastic holder which enables precise positioning. (Slide 3) This was found necessary because the head position is quite critical. With the head horizontal, long exposures may be given with impunity. (Slide 4)

On the other hand, with the head tilted upwards, the occiput well down, only short exposures can be given without drastic effect. (Slide 5)

U.h.f. energy was supplied the cavity at the top center by a quarter wave probe which was connected to the output of a Collins T17AGR transmitter which is a hundred watt ground-to-air transmitter operating in the range from 225.0 to 399.9 mc/sec. This was obtained on loan from the Naval Research Laboratory. Later a new Stewart-Warner T282D/GR was obtained through the courtesy of Rome Air Force Base. We hope they will continue to be courteous and let us keep it a while.

The calorimetry was done in plexiglas cylinders of varying radii filled to various lengths with physiologic saline solution and placed at different levels with respect to the cavity floor. Temperature rises in the saline were correlated with the amplitude of the signal from the pick-up probe displayed on a cathode ray tube the vertical plates of which were connected directly to the probe.

The calorimetry was performed through the range of frequencies explored, from 380 to 395 mc/sec. in one-megacycle steps.

Most of the exposures were to continuous wave radiation, although 100% modulation with a 500 and 1000 cycle sine wave was also employed. A crude form of pulsing, by over-modulation was also done in a limited number of exposures.

The animals were all young Macaca rhesus monkeys mostly weighing 7 to 10 lbs. though a few larger animals were also used.

The usual exposure period was from two to ten minutes. The shortest exposure leading to death was two minutes and 55 seconds. The longest single exposure (in the horizontal head position) was about 3 hours without noticeable effect on the animal.

In the pathological studies, some animals were injected with fluorescein 30-40 minutes before exposure and were then immediately sacrificed. Others were injected and sacrificed some weeks after exposure. Brain and spinal cord sections were fixed and stained by the Nissl and myelin staining techniques.

Electroencephalographic recordings were made from electrodes hammered into the outer table of the skull. R.F. pickup on the leads resulted in a number of burns on the scalp. We are still wondering how to get good EEG's during radiation.

Now to summarize the clinical findings during exposures:

Animals displayed arousal and drowsiness which were cyclic in nature. (Slide 6) During the drowsy periods they were akinetic, tending to keep the whole body in a fixed position. This pattern is usually seen within 60 seconds of initiation of the exposure. The animal then might stare (Slide 7), with widened palpebral fissures and fixed gaze. Then agitation, beginning with rapid side-to-side head movements, would occur. These movements often ceased abruptly and the animal would be quiet and unresponsive to touch, pain, light, and sometimes to sound stimuli. Alternating periods of arousal and drowsiness usually occurred. Three animals were deeply anesthetized with phenobarbital, being quite unresponsive to pinching of the Achilles tendon and to deep pin pricks. However, they could be made to move about in the chair within a minute of beginning of radiation. By alternately switching the transmitter on and off, one of these animals was brought to the point of successive arousal and complete relaxation, in a 20-second cycle, reacting like a puppet on the end of a string. This particular effect was elicited most readily at 389 megacycles when compared with exposures at equal power at 384, 385, 386, 387, 388, 389, 390, and 391 mc/sec.

Eye Signs

With continued exposure, the animal would develop bilateral ptosis. Suddenly he would open his eyes and stare upward. The pupils were usually small and equal. Then the eyes would begin to move independently, and the pupils would dilate. Often one pupil would be larger than the other and in

some instances lose its roundness. The pupils would then dilate and constrict irregularly and rapidly. Nystagmoid movements progressing to a rapid, usually vertical nystagmus, accompanied by rapid blinking, would then occur. The nystagmus often persisted for several minutes after cessation of radiation.

Accompanying the eye signs were autonomic changes. The skin of the face would often become flushed and then pale. The nose often became pink and the respiratory rate increased. Salivation and lacrimation (Slide 8) were also observed. With further exposure, the rapid blinking progressed to clonic movements of the orbiculares oculi, bilateral clonic movements of the other facial muscles, a severe grimace (Slide 9) which pulled back the lips from the teeth, clonic flexion of the neck, and symmetrical clonic movements of the upper extremities, trunk, and lower extremities in that order.

The onset of the rapid blinking and the grimace which heralds the generalized seizure was always a serious sign, although several animals with such signs progressed to complete recovery.

Motor Loss

Two animals became quadriplegic. Two others developed weakness of the upper extremities and several became ataxic for varying periods. In all of these there also developed lesions of the occipital muscles and overlying skin. One animal developed a right facial weakness (Slide 10) with a concurrent anesthesia in the distribution of the upper two branches of the trigeminal nerve.

EEG Changes

EEG changes occurred usually at about 2 minutes after initiation of exposure. The records seem typical of alternating arousal and drowsiness. (Slide 11) These changes disappeared within 2-3 minutes after cessation of exposure.

As a summary of the clinical findings, we have prepared a short film which was photographed by the color television technique of Brown and Whitehouse of NIH. (show film)

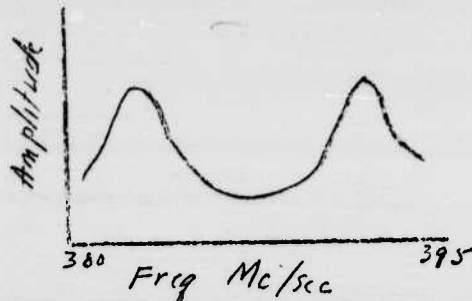
The sequence of clinical signs all point to an effect on the brain stem. They resemble signs produced by direct electrical stimulation in this region.

Before discussing the pathological findings, it would be well to describe the calorimetry performed, since this can be directly related to some of the pathological findings.

Power absorption at the head position was measured by temperature rises in physiologic saline solution exposed in plexiglas cylinders of varying height and radius. (Slide 12) They were placed at the bottom center of the cavity (Slide 13), some resting flat on the floor, others protruding through to various positions. Cooling curves were obtained on each before exposure, with doors closed and the transmitter blowers and filaments on.

The amplitude of the signal from the sampling probe directly above the calorimeter was recorded simultaneously with the temperature rise over a 10-minute period. Measurements were made in one-megacycle steps from 380 to 395 mc/sec.

For each container, the amplitude setting for an equal temperature rise at each frequency was recorded. The resulting curves of signal amplitude for a 5° rise in 10 minutes all had the same shape;



that is, the amplitude ratios were the same for all receivers, even though the actual power absorption varied from receiver to receiver. However, the important thing was that exposures could be made at equal relative power absorption across the frequency range examined.

After numerous exposures of saline columns varying from 6 to 19 cm. in height and from 60 to 1000 cc. in volume, some sort of a pattern began to emerge which might enable us to examine the actual power absorbed.

One can think of the radiation as entering from the top, for example, and calculate the amplitude setting for say 40 mw/cm^2 on the top surface. This results in a series of curves which are not too far apart, but leaves a couple of the sets of data well apart from the rest.

The same can be said for the equally simple-minded view that one should consider the vertical surfaces within the cavity of interest. One could, for example, calculate the amplitude settings for 5 mw/cm^2 coming in radially. This brings the curves closer yet, but again there are a couple of curves which withdraw into even more splendid isolation than before. It was apparent that the shorter containers absorbed even less power than that simply proportional to the height. Also, for some distance below the floor of the cavity, absorption occurred.

If one assumes that the absorber acts like an antenna and has a voltage distribution proportional to the sine of its height in radians, it is possible to calculate approximate power absorbed in any given container. This gives us a picture of the energy entering radially as shown. (Slide 14) Here is a cylinder one quarter wave length in height. If the voltage is distributed as the sine of the angle corresponding to

the height, the power density should be distributed as the sine². Then the power on the surface should be proportional to

$$\int_0^h \sin^2 h \, dh = \left| \frac{1}{2}h - \frac{1}{4} \sin 2h \right|_0^h$$

and the average power density proportional to

$$\frac{\left| \frac{1}{2}h - \frac{1}{4} \sin 2h \right|_0^h}{h} \quad \text{which} = \frac{\pi}{4h_0} \text{ at } h_0 = \frac{\pi}{2} = \frac{\lambda}{4}$$

On any container of height h_1 , the average power density should be proportional to

$$\frac{\left| \frac{1}{2}h - \frac{1}{4} \sin 2h \right|_0^{h_1}}{h_1}$$

Therefore the average power density on a container should be related to that at $\frac{\pi}{2}$ by

$$\frac{4h_0}{\pi h_1} \left| \frac{1}{2}h - \frac{1}{4} \sin 2h \right|_0^{h_1}$$

Now suppose the average power density on a quarter wave length cylinder to be P_0 mw/cm². If this cylinder has a radius r_0 , the average power density on a smaller cylinder of the same height, but radius r_1 , would be

$$P_0 r_0 / r_1$$

since the radiation converges in the horizontal, but not in the vertical planes. A cylinder of radius r_1 but height h_1 would then receive an average power density of

$$P_i = P_0 \frac{r_0}{r_1} \left[\frac{4h_0}{\pi h_1} \right] \left| \frac{1}{2}h - \frac{1}{4} \sin 2h \right|_0^{h_1}$$

Now if one considers absorption to a radius r , since the depth of absorption path = $r_1 - r$

P , the power density at radius r , = $P_i \frac{r_1}{r} e^{-k(r_1-r)}$

$$\text{which} = P_0 \frac{r_0}{r} \left(\frac{4h_0}{\pi h_1} \right) \left| \frac{1}{2}h - \frac{1}{4} \sin 2h \right|_0^{h_1} e^{-k(r_1-r)}$$

The power absorbed in the path $(r_1 - r)$ must then =

$$4 P_0 \frac{r_0}{r} \frac{h_0}{\pi h_1} \left| \frac{1}{2}h - \frac{1}{4} \sin 2h \right|_0^{h_1} (1 - e^{-k(r_1-r)})$$

This expression tells us the average power absorbed for each square cm. of vertical surface.

The total power absorbed then equals

$$8 \pi r_1 H_1 P_0 \frac{r_0}{r} \frac{h_0}{\pi h_1} \left| \frac{1}{2} h - \frac{1}{4} \sin 2h \right|_{h_1} (1 - e^{-k(r_1 - r)})$$

(H_1 in cm h_1 in radians) Since $\frac{h_0}{h_1} = \frac{H_0}{H_1} P_{wr. absorbed}$

$$\text{Using } H_0 = 19.2 \text{ cm} = \frac{1}{4} \quad 8 r_1 H_0 P_0 \frac{r_0}{r} \left| \frac{1}{2} h - \frac{1}{4} \sin 2h \right|_{h_1} \frac{1}{\pi (1 - e^{-k(r_1 - r)})}$$

$$r_0 = 5 \text{ cm}$$

$$P_0 = 2 \text{ mw per cm}^2 \text{ (since this corresponds to the values observed experimentally)}$$

$$r = 0.5 \text{ cm}$$

$$\text{Power absorbed} = 3070 r_1 (1 - e^{-k(r_1 - 0.5)}) \left| \frac{1}{2} h - \frac{1}{4} \sin 2h \right|_{h_1}$$

Since the power absorbed can be observed directly by experiment, one can then calculate the amplitude settings for each frequency which would give 2 mw/cm^2 on the vertical surface of a cylinder $\frac{1}{4}$ wave length high and 5 cm. in radius. This has been done for a variety of positions and sizes. (Slide 15) Only five of the curves are shown. Those for the containers marked C₄ and C₈ are all within the extremes shown, the values at 388 mc/sec. all being between 2.4 and 2.8 cm.

I am sure these curves look terrible to a physicist, but to a biologist they are not unreasonable.

Now this picture of the radiation pattern is not directly relatable to plane wave propagation in a free field. I suppose a crude approximation for the exposure at the surface of the animal would be to make a Flatlander of him and say that his exposure of 2 mw per cm^2 corresponds to this multiplied by the ratio of surface to the profile area which is π making it 6.4 mw/cm^2 at the surface, if he were 5 cm. in radius. Going into the animal, each successive decrease in radius brings an increase in power density, so that at the region of interest, the brain stem, the power density can be considered to be around 10 times this. Since the animal's head is not 5 cm. in radius, but more like 2.5, the surface exposure of the monkey's head corresponds to 12.8 mw/cm^2 and the exposure of the brain stem corresponds to about 64 mw/cm^2 of unidirectional radiation.

Pathology

The pathological studies were performed by Dr. Igor Klatzo. I will not presume to discuss the pathology in any detail since this is somewhat out of my line of work. Dr. Klatzo injected several animals with fluorescein 30-40 minutes prior to exposure. The animals were given what was assessed to be well over a lethal dose by the clinical signs and then immediately sacrificed. Photography under ultraviolet light of brain sections revealed infiltration of the dye into the brain stem adjacent to the 4th ventricle and cerebral aqueduct. (Slide 16) Nissl-stained sections showed tigrolysis in the neurons of the nuclei. This normal section (Slide 17) and this section of affected facial nucleus (Slide 18) in the medulla show the degree of change seen in the acute cases. Here is the same sort of change in the dentate nucleus of the cerebellum. (Slides 19 and 20)

Some animals were exposed and then 90 days later injected with fluorescein and sacrificed. There was no fluorescein infiltration in these animals. Sections showed in some areas that the neurons were preserved, but there were perivascular cystic spaces and a microglial response. These sections from the nucleus cuneatus of the medulla show this effect. (Slides 21 and 22)

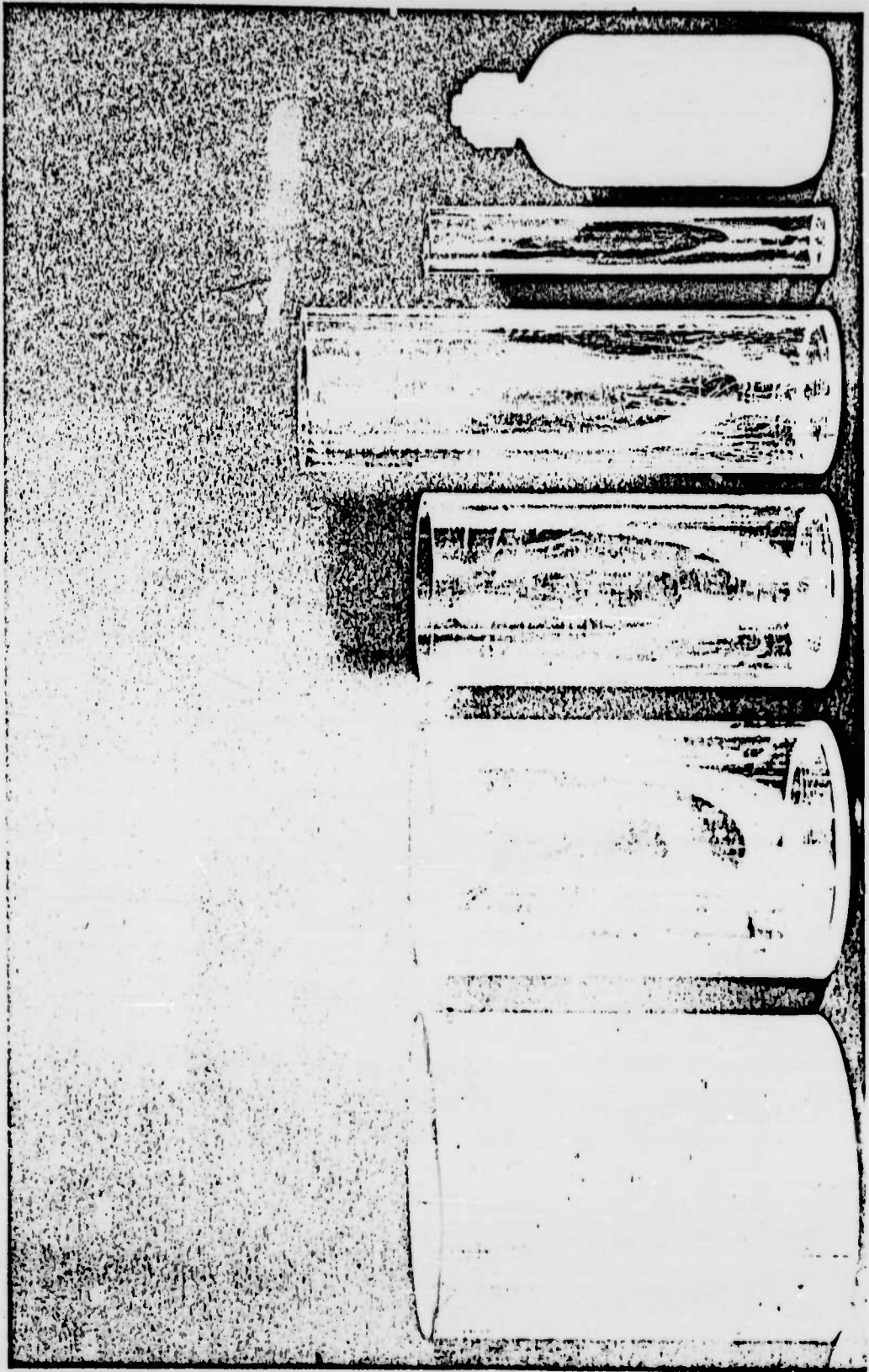
In four animals where the neck was tightly pressed against the posterior portion of the ring of plastic encircling it, skin and muscle lesions occurred. These animals showed fluorescein infiltration in a wedge-shaped area of the dorsum of the cervical cord. (Slide 23) The myelin stained preparations indicated a zone of damage with an intervening normal zone. (Slide 24)

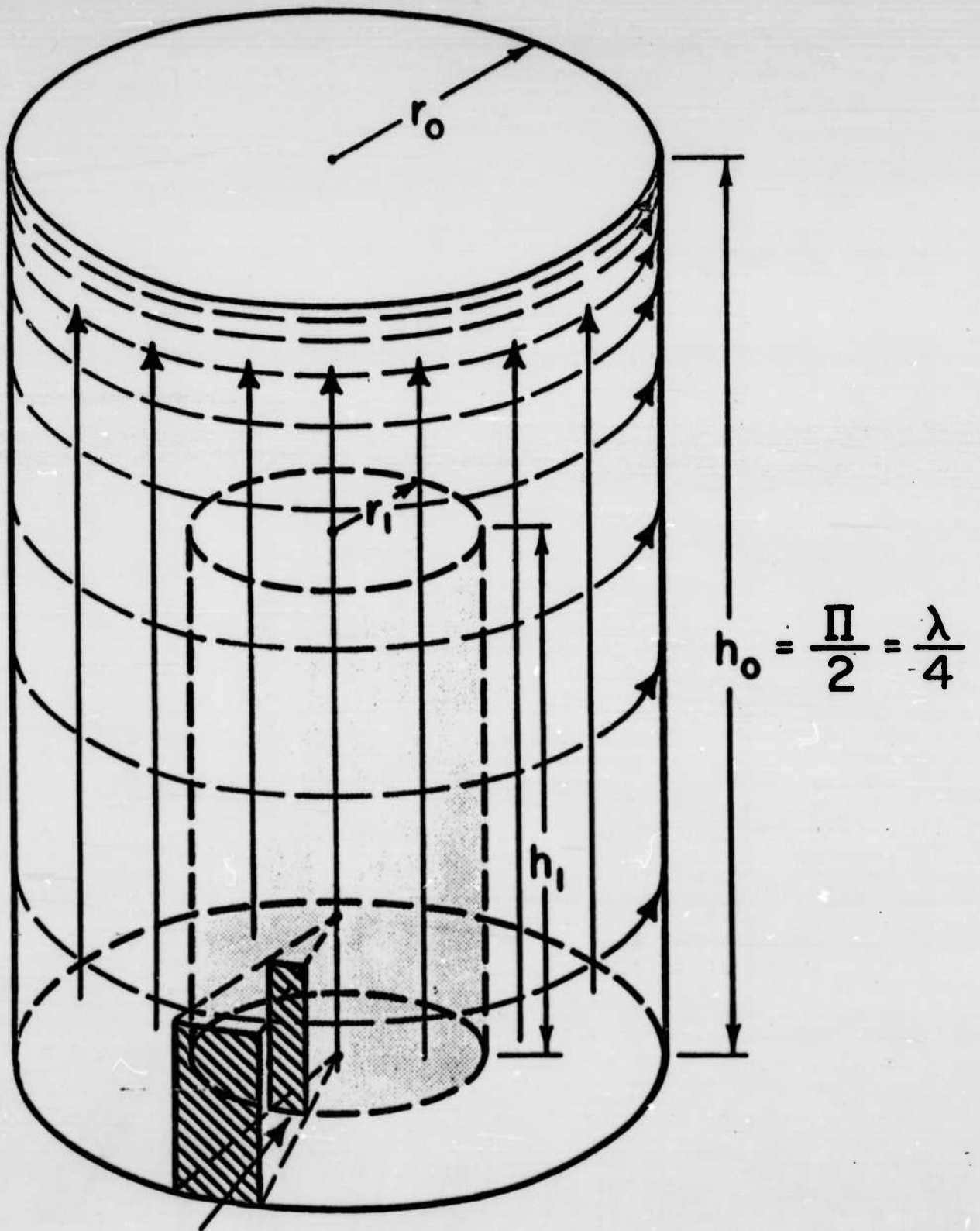
It seemed apparent that unless adequate separation was provided at this point between the neck and the floor of the cavity, a high current path was available through capacitative coupling. In one animal, the occiput was deliberately jammed tightly against the rim of the opening. He was injected with fluorescein and exposed long enough to produce an edematous swelling at the point of contact and then sacrificed. The gross specimen looked like this (Slide 25) and under ultraviolet light like this (Slide 26). Parasagittal and transverse sections of the occipital pole showed this (Slide 27).

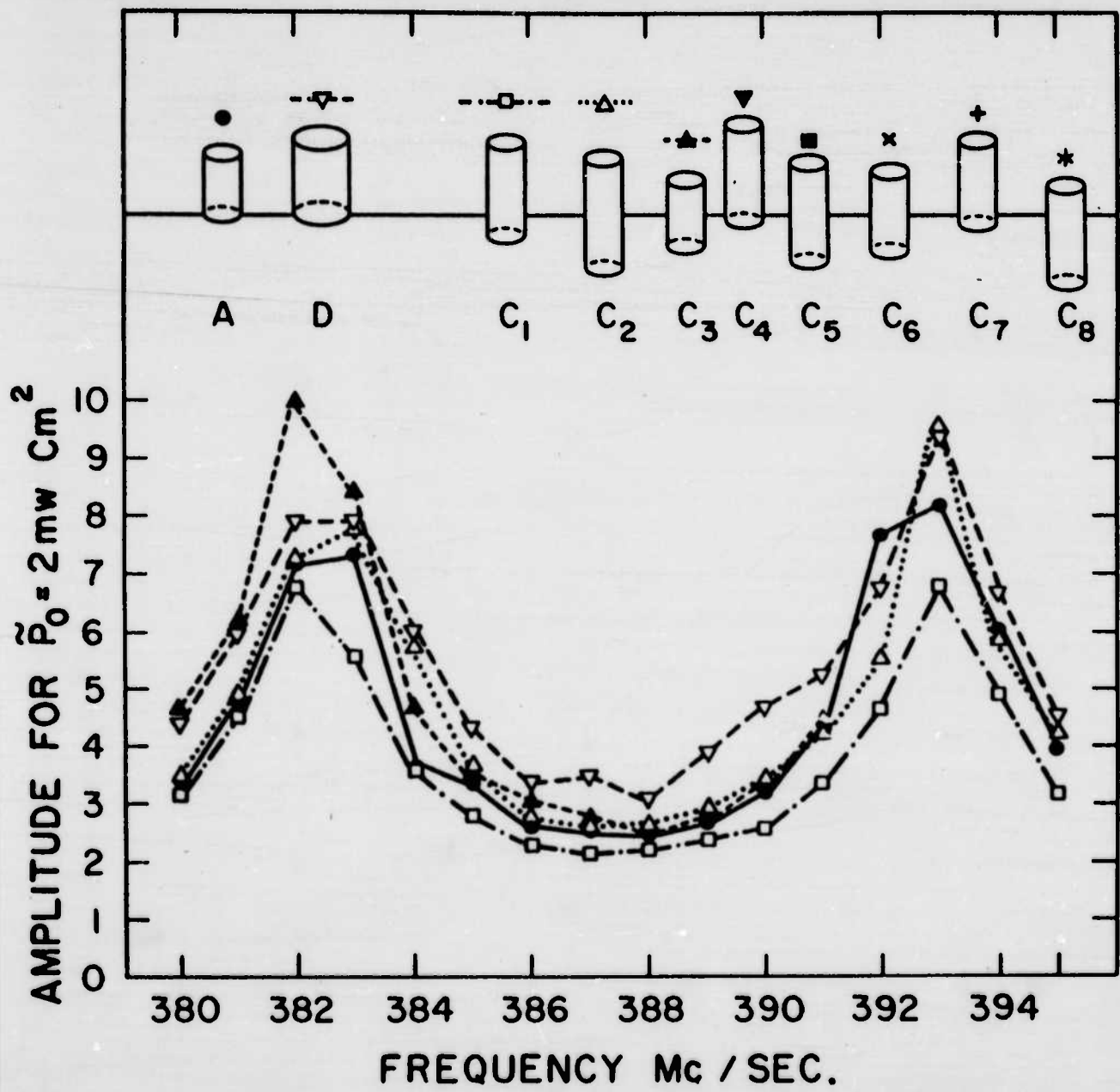
It must be emphasized that the pathological findings illustrated were all of animals exposed to lethal or near lethal doses. Animals which were used repeatedly for arousal studies for possible frequency response showed no residual signs at all and were apparently quite normal in every way immediately after exposure.

It is interesting that the changes seen in the brain stem are restricted to the nuclear masses. It is also interesting that the signs which develop resemble those produced by direct electric stimulation in this region. Obviously we have shed no light in this study on the question

of how much effect might be thermal and how much might be due to some other more specific interchange of energy. However, the apparent frequency sensitivity of the arousal under anesthesia and the rapid response time (10 seconds) makes one wonder if gross heating is the only explanation. It is perhaps not unlikely that pulsing of the radiation at physiologic rates of a few per second up to 100 per second or so may elicit responses which can be measured clinically.







Comments on the paper titled "Neurological Effects of Radio-Frequency Energy" by S. A. Bach, S. A. Lewis, and M. Baldwin.

by

David E. Goldman, CDR, MSC, USN*

This paper is of unusual interest in two respects. On the one hand it provides a technique for producing a set of neurological responses which are of considerable value in studies of the central nervous system. On the other hand the circumstances suggest the possibility that these effects may not be due simply to the generation of heat. Clearly the work will have to be continued and extended in order to delimit the conditions under which these phenomena are produced and to obtain quantitative relations between dose and effect. Statements at the present time as to the mechanism of these effects would be premature. The possibility should not be overlooked that the intense electric fields which occur may have some sort of direct action on nerve tissue. The extent to which these observations bear on the problem of hazards under field conditions, of course, remains to be determined. I for one look forward with great interest to following the progress of this work.

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THEORETICAL CONSIDERATIONS
PERTAINING TO THERMAL DOSE METERS

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SUMMARY:

The relationships which apply to the performance of thermal dose meters are derived. The discussion of these relationships permits the statement of optimal performance criteria for each frequency. Performance data of optimized thermal dose meters are given and a most practical design outlined.

Some commonly used field intensity meters, such as horns and dipoles are useful if high gain characteristics are desired, i.e. when small field intensities, under normal circumstances near the noise level, must be evaluated. Inasmuch as we are concerned with flux values near 10 mW/cm^2 , i.e. field strength values near 2 Volt/cm on an average basis, or correspondingly more during the pulse, noise considerations are immaterial. High gain characteristics of the receiving device are, therefore, unnecessary. On the other hand, it must be realized that high gain characteristics are associated with considerable disadvantages. They are:

- a. The high directionality of the high gain device. Proper orientation becomes the more critical the higher the gain. This complicates rapid measurements desired in survey studies.
- b. The gain of a horn or dipole is a function of the field to which it is exposed. It is usually defined for homogeneous fields and waves, whose curvature of wave front is small across the aperture of the directional device. In the non-homogeneous field, such as in the interference zone of antennas, the gain of the directional receiving device becomes a complicated function of the field to be evaluated and is therefore often useless, giving erroneous results based on non-applicable assumptions underlying the calibration of the device.

We conclude that for the purpose of interest here low gain devices are preferable. It is furthermore desirable that their dimensions be as small as possible in order to minimize the volume where the originally existing field pattern is disturbed by the insertion of the detecting device. Thus meaningful measurements will be possible in areas of changing field intensities which can not be investigated with directional devices whose performance integrates over a large area (linear dimensions usually about half a wavelength). Non-directional devices which interfere with as little as $1/100\text{th}$ to $1/10\text{th}$ of a wave length are possible, depending upon frequency.

The least directional device is the sphere by virtue of its shape. However, to measure in turn the field strength in a sphere by means of a non-thermal field strength measuring device is undesirable. It would require arrangements which take cognizance of the field direction in the sphere, i.e. require orientation and thereby defeat the end we hope to achieve. This situation suggests investigation of the usefulness of thermal techniques.

Before entering into a more detailed discussion of related thermal aspects we wish to point out that the field strength within a sphere is well defined if the sphere is thought to be exposed to an originally homogeneous field. The neighborhood of any other dielectric mass within a distance comparable to the dimensions of the sphere or mass, will yield further modifications of the field. The field within the sphere and its consequent heating rate will change drastically if it is carried on or near the surface of the human body. Hence readings obtained in such a fashion are meaningless and impossible to interpret. The same statement applies, of course, to all field devices carried on the body. From our previously published investigations of the variability of impedance match of the human body surface to air it becomes obvious furthermore, that errors arising from the location of the sensing device on the body surface can not be calibrated out.*

*Pertinent calculations show that the square of the field strength, which is a measure of the radiant energy, can vary near the body surface by considerably more than two orders of magnitude and changes critically with such parameters as skin and subcutaneous fat thickness, frequency, etc.

In summary: Any sensing device must be used at a "sufficient" distance from the human body. How large the distance should be is impossible to state at the present in the absence of pertinent data, but it most certainly is sufficiently large to prohibit the device to be carried on the body surface. The electrical field strength C in a solid sphere of a complex conductance

$$\Lambda_i = \kappa_i + j\omega\epsilon_i\epsilon_r \quad (\epsilon_r = 8.84 \cdot 10^{-14}) \quad (1)$$

and immersed in a medium of the conductance

$$\Lambda_a = \kappa_a + j\omega\epsilon_a\epsilon_r \quad (2)$$

can be shown to be related with the field strength E of the external field in large distance from the sphere, assuming the latter quantity to be constant and independent of location. This statement includes the assumption that the wave length of the radiation is large compared with the dimensions of the sphere.

$$C = \frac{3E}{2 + \Lambda_i/\Lambda_a} \quad (3)$$

We are interested here in the special case when the outside material is air:

$$\Lambda_a = j\omega\epsilon_r \quad (4)$$

and hence

$$C = \frac{3E}{2 + \epsilon_i - j\frac{\kappa_i}{\omega\epsilon_r}} \quad (5)$$

The heat development in the sphere per unit volume is equal to

$$H = K_i K_i^2 = \frac{9E^2 \alpha_i}{(2 + \epsilon_i)^2 + \left(\frac{\alpha_i}{W \epsilon_r}\right)^2} \quad (6)$$

while the free flux

$$F = \frac{E^2}{377} \quad (7)$$

Hence the "relative absorption cross section" of the sphere

$$S = \frac{4\pi R^3 H}{4\pi R^2 F} = \frac{3 \cdot 377 \alpha_i}{(2 + \epsilon_i)^2 + \left(\frac{\alpha_i}{W \epsilon_r}\right)^2} R \quad (8)$$

In order to evaluate now the possibilities of using small spheres as dose meters we enter into a discussion of the equation (6). First we wish to point out that it would be desirable to optimize the heating rate as much as possible. This is simply a reflection of the fact that flux levels comparable to the one proposed for tolerance do not provide much heat: 10 mW/cm² will heat, for example, 1 cc of water or tissue by 1.5° C after 10 minutes exposure, provided that the 10 mW are completely absorbed.

Differentiation of either equation (6) or (8) with regard to K_i yields the optimal condition

$$2 + \epsilon_i = \frac{\alpha_i}{W \epsilon_r} \quad (9)$$

Introducing this into equation (6) gives the optimal heat rate

$$H(\text{max.}) = \frac{9}{2} E^2 \frac{(\omega \epsilon_r)^2}{\kappa_i} = \frac{9}{2} E^2 \frac{\omega \epsilon_r}{2 + \epsilon_r} \quad (10)$$

The following conclusions are evident from equations (9) and (10):

1. In order to obtain optimal heating at a given frequency, the conductivity has to be adjusted in linear proportion with the frequency.
2. For optimal heating the dielectric constant should be chosen as low as possible.

The optimal cross-section is obtained by introduction of condition (9) into equation (8). The result

$$S(\text{max.}) = \frac{3}{2} 377 \frac{(\omega \epsilon_r)^2}{\kappa_i} R = \frac{3 \cdot 377 \omega \epsilon_r}{2(2 + \epsilon_r)} R \quad (11)$$

again suggests reduction of the dielectric constant ϵ_r in the interest of optimal relative absorption cross-section. The relative absorption cross-section is furthermore increasing with the radius of the sphere and the frequency.

From the dioxan electrolyte data discussed before it is apparent that it will be possible to obtain variability of conductivity for dielectric constants as low as 8. Hence further discussion will be based on this value. Equations (9), (10), and (11) reduce to

$$H(\text{max.}) = \frac{9}{20} E^2 \omega \epsilon_r \quad (12)$$

$$S(\text{max.}) = \frac{3 \cdot 377}{20} \omega \epsilon_r R \quad (13)$$

$$\kappa_i = 10 \omega \epsilon_r \quad (14)$$

TABLE I represents values of "Optimal" conductivities and heating rates and relative absorption cross-section (per cm Radius) at various frequencies of interest.

| f (Mc) | κ : (opt.) (mMho/cm.) | H(max.)/E ² (mWatt/Volt ²) | S(max.)/R (%/cm) | ΔT /min (°C/min) |
|-----------|---------------------------------|--|---------------------|-----------------------------|
| 100 | 0.55 | 0.025 | 0.31 | 0.013 |
| 300 | 1.66 | 0.075 | 1 | 0.04 |
| 1 000 | 5.5 | 0.25 | 3.1 | 0.13 |
| 3 000 | 16.6 | 0.75 | 10 | 0.40 |
| 10 000 | 55 | 2.5 | 31 | 1.35 |

Optimal conductivity required for maximum heating, heat development per cc and field strength 1 Volt/cm, relative absorption cross-section for a 1 cm-radius sphere and average temperature rise of spherical dose meter per minute. The data are given for various frequencies. A dielectric constant of 8 (relative to vacuum) is assumed. The ΔT -data are calculated assuming a free field flux value of 0.1 Watt/cm² ($E^2 = 37.7 \text{ Volt}^2 \text{cm}^{-2}$).

TABLE I includes furthermore optimal temperature rise data per minute, assuming a flux of 100 mW/cm²=0.1 Watt/cm². For a flux of 0.01 W/cm² temperature rise values are, of course, tenfold smaller. The quoted conductance values can be readily materialized as may be seen from dielectric data pertaining to dioxan-electrolyte mixtures.

Our next statements pertain to the frequency dependence of the heat development in a given spherical absorber. From the statement of the optimal conditions outlined before it may appear that the device is highly frequency dependent in its response characteristics. This is by no means so. Figure 1 gives a graphical representation of the frequency response of heat rate. The device which has been designed for optimal response at a given frequency f_0 (marked by arrow) increases its sensitivity somewhat at higher frequency. Actually f_0 appears as the cut-off frequency where the heat rate starts to decrease from a constant value by 3 db. At lower frequencies the resolution decreases rapidly as f decreases. In conclusion: The device is useful for the total frequency range above the "optimal frequency" for which it has been designed. It is frequency independent for the total frequency range up from $f=2f_0$ (error 25%).

The curves in Figure 1 demonstrate why there is no contradiction between the statement of an optimal choice of conductivity and the monotonous frequency behavior of the heat rate: The sum of all points of optimal design values as defined by equation (9) determines a straight line with a slope of 45° (dashed line). It is not possible to leave the area below this line, no matter what conductance-frequency combination is considered. The points of optimal design appear in this demonstration as the best possible heat rate values to be obtained at any frequency. The only means to increase the heat rate is by means of change in dielectric constant. A decrease of the latter quantity from 8 to 1, i.e. to the dielectric constant's theoretical limit, shifts the border line of the region of possible heat rate-frequency combinations by a factor of 3.3 upwards, as may be seen from equation (10). If a dielectric of very low dielectric constant, but appreciable conductance in accord with equation (9) could be realized, the

temperature values quoted in TABLE I could be increased by half an order of magnitude.*

The curves demonstrate also that high sensitivity (high heat rate and temperature rise) are to be traded against poor low frequency response: The higher the heat rate at frequencies above the design frequency, the lower the heat rate at frequencies below the design frequencies. Any change in conductance will vary the heat rate at frequencies on one side of the original design by the inverse of the factor which determines the changes at frequencies located at the opposite side of the original design frequency. The best possible compromise may well be near a design frequency of 1000 Mc. In this case conductance specifications can be easily realized (for example by proper dioxan-electrolyte mixture) and yet a temperature elevation of 1° C per 6 minutes is possible for the 0.1 Watt/cm² figure. This is sufficient to permit detection of flux levels as low as 10 mW/cm² accurate to 1% within a few seconds, as may be recognized from available resolution of temperature detection systems to be mentioned below.

* All above outlined relationships are immediately evident to those familiar with the dielectric relaxation behavior of inhomogeneous dielectrics. The equivalent and "effective" conductivity of a suspension of spheres in another medium is characterized by a simple relaxation function

$$\kappa = \kappa_{\infty} (\omega T)^2 / 1 + (\omega T)^2$$

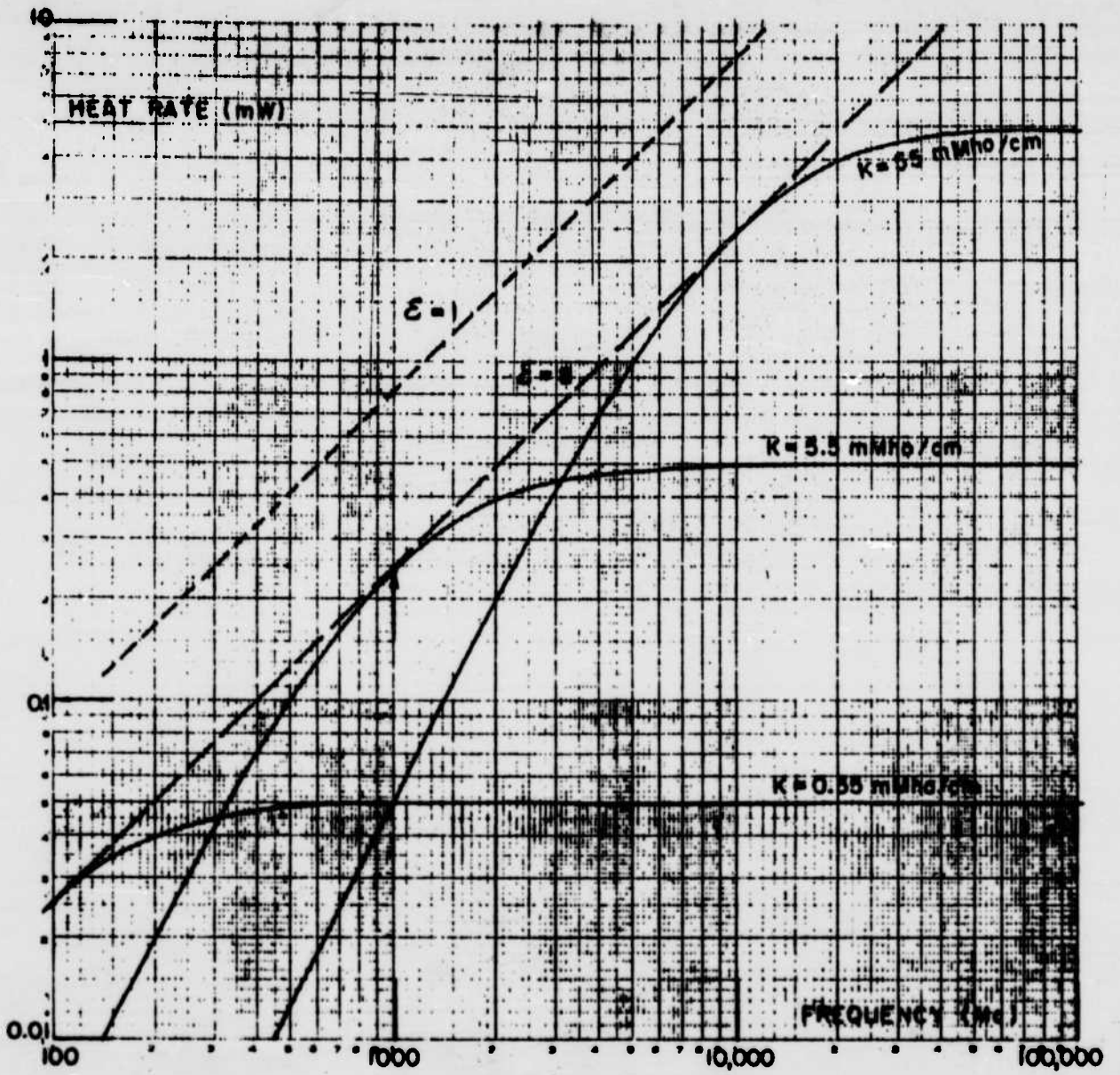
involving one time constant T which is proportional to the resistivity of the lossy spheres. The relaxation function can be presented in terms of an equivalent circuit, involving frequency independent circuit element. It consists of a capacitance in parallel with a series RC-arrangement. From this the frequency independent characteristics at high frequencies and the square dependence upon frequency at frequencies low in comparison to the "characteristic" frequency (identical with design frequency) are readily realized.

It is noticed that for flux levels near the accepted tolerance level only small temperature increases can be expected. This has its advantages and disadvantages:

1. The disadvantages of a small temperature rise per minute is, of course, due to the difficulty of detecting such small temperature increases or the necessity for waiting long periods of time before noticeable increase is obtained.
2. The advantage of small temperature rise is given by the fact that the observed temperature rise will truly reflect the absorbed energy. If the temperature rise were large, conduction losses to the surrounding of the absorbing sphere would influence the temperature quite rapidly. This would create a situation where parameters of a variable nature (heat loss depends on air temperature, wind velocity, etc.) would be introduced, making it impossible to relate temperature rise with energy flux.

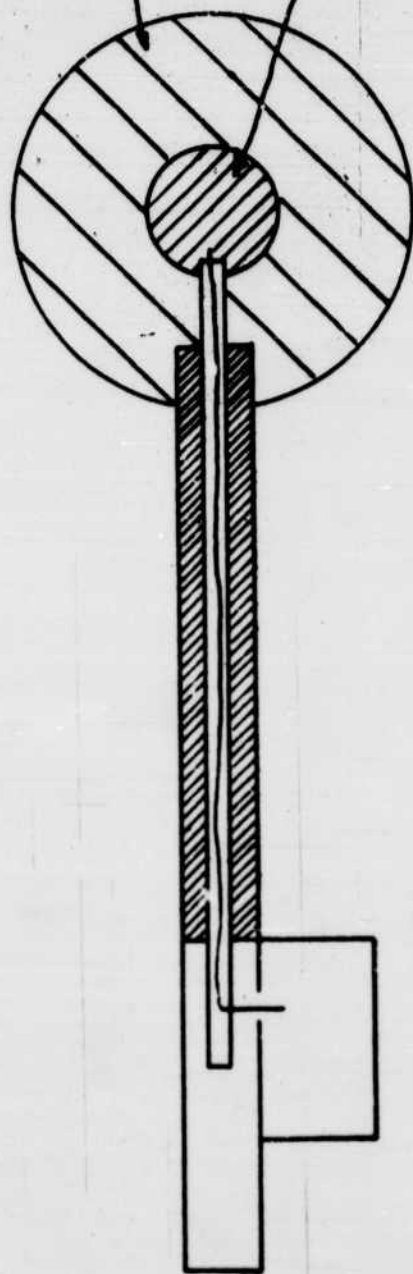
We propose that thermal devices be used only during the linear part of the transient period when the absorber increases its temperature. The steady state temperature which is ultimately approached, is in a very unpredictable manner related with energy flux and external factors, and therefore, in our opinion, useless for quantitative work. Utilization of the linear part of the transient relates temperature change rather than temperature with energy flux. The time it takes to change the temperature of the absorber by a given amount is then directly proportional to the energy flux or the square of the electrical field intensity to be determined. The linear range of the device can be extended considerably by surrounding the absorber with a material of low heat conductivity. This material should, on the other hand, not interfere substantially with the electrical field. Some of the low dielectric constant materials commercially available are quite useful since their porous structure guarantees at the same time very low dielectric constant and heat conductance.

The measurement of small temperature changes is not difficult by presently available means. It can be done either with thermistors or thermocouples. They must be designed to avoid selective and uncontrollable energy pickup from the field by proper shielding of the leads to the sensing device in the absorbers. A simple technique utilized in our design consists of the measurement of the low frequency conductance of the absorber material with the help of small microelectrodes in the absorber. The latter's conductance has a temperature coefficient of about 2.5% per ° C. Hence the temperature rise data given in TABLE I can be translated immediately into percentage changes of low frequency conductance. Instrumentation to measure relative resistance changes accurate to 1 part in 10,000 is easily constructed. It can be developed in transistorized form in such a manner that it is portable and attached to the handle of the stick which carries at its other end the microwave absorber sphere (Figure 2). All above numerical considerations are based on the assumption that the dimensions of the absorbing sphere are small compared with the wavelength. Since heat rate and temperature rise are not a function of radius, a small size of the absorber is possible, about or less than 1 cm in diameter. With such a device it should be possible to apply above formulated quantitative relations up to about 3000 Mc. For higher frequencies corrections are necessary which are best determined experimentally, i.e., calibrated into the instrument.



LOW LOSS
HEAT INSULATOR

ABSORBER



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FIG. 2

A TECHNIQUE FOR RELATIVE ABSORPTION
CROSS-SECTION DETERMINATION

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SUMMARY

Following a definition of "Relative Absorption Cross-Section" and the factors which affect it, an experimental approach to its determination is described. To get meaningful measurements that are useful in prediction, the experimental work must be carried out in the far field of an antenna (plane waves). The object to be irradiated is filled with an electrolyte approximating the dielectric properties of the human body. The average temperature rise of the object is measured after exposure to a microwave field (3000 mc) of known power density. It is shown that a transmitter average power of about 330 watts is required to get a temperature rise of 1°C over a time period of 30 minutes for a 10 inch diameter sphere. The power density incident on the sphere is about 20 mw/cm². Preliminary measurements have shown that the set-up is feasible.

* * *

We define as "relative absorption cross-section" the ratio of two power values P_1 and P_2 . P_1 is the power absorbed by the object of interest when exposed to a uniform plane wave. P_2 is the total power value given by the product of the power flux prior to the insertion of the obstacle into the field as measured at the position of the obstacle and of the "projected cross-section" of the obstacle of interest. The latter is obtained for viewing in the direction of the propagation of radiation upon a plane perpendicular to the direction of propagation. It is apparent that the knowledge of this relative absorption cross-section is a prerequisite to a determination of the power which is absorbed by animals or mankind from free field measurements. It is also apparent that estimates of power absorbed by mankind must be based ultimately on free field measurements where power density and field strength are simply related. We have pointed out previously that the concept of power density fails in the presence of standing wave patterns and that field strength values near the surface of the human body are related only in a very complex manner with the energy absorbed by the body.

The relative absorption cross-section of a sphere can be shown to be:

$$S = \frac{1131 K_1 R}{(2 + \epsilon_1)^2 + \left(\frac{K_1}{\omega \epsilon_1}\right)^2} \quad (1)$$

where K_1 and ϵ_1 are conductivity and dielectric constant of the sphere, R its radius and $\epsilon_0 = 8.84 \cdot 10^{-14}$ farad/centimeter¹. Suppose $f \sim 100$ Mc, and $R \sim 30$ cm, i.e. we consider the case of a sphere whose average dimensions compare with those of the human body. Then for typical body properties in the microwave range $K_1 \sim 10^{-2}$ mho/centimeter and $\epsilon_1 \sim 60$, $S \sim 1\%$, i.e. the relative absorption cross-section is in this case only 1%. This sample is chosen to demonstrate that the relative absorption cross-section can be extremely small. On the other hand, at frequencies so high that the wavelength is small in comparison with the dimensions of the illuminated obstacle, the surface of the human body may be considered as a first approximation to be a plane surface hit under right angle of incidence, provided that the broad side of the trunk is oriented towards the radiation, i.e. the person faces the antenna system. In this case, we may anticipate absorption up to 100% efficiency as shown by previously published work from our laboratory. In summary: The relative absorption cross-section which is introduced to characterize the efficiency of absorption of energy incident upon the projected cross-section of an illuminated obstacle, can vary in the case of exposure of mankind between 100 and less than 1%. This means that we are at the present left virtually without any knowledge whatsoever as to the amount of energy picked up by mankind. In view of recommended tolerance flux levels of 10 mw/cm² absorbed energy corresponding free field flux levels are completely uncertain. Another consequence of this finding: Any type of animal work, where test animals are exposed to electromagnetic fields in order to learn from their responses on what level to establish tolerance flux or field strength levels for mankind, appears irrelevant.

The definition of the relative absorption cross-section involves several quantities which must be known in order to establish its value:

1. A homogeneous plane field of constant intensity at the location where the scattering and absorbing obstacle of interest is to be introduced into the field. It should be of known value. This value may be given either in terms of flux F , i.e. in units of Watts/cm², or in terms of field strength E , both being related in air by the equations $F = E^2/377$. Absence of any standing wave pattern is a prerequisite to this equation.

2. Knowledge of the "shadow cross-section" or "projected cross-section" of the obstacle illuminated by the radiation and determination of the amount of energy absorbed by the lossy obstacle. While the former is readily established, different possibilities exist to ascertain the second quantity.

The achievement of a homogeneous field is not easy, and this subject is discussed in another place.² Here we will discuss alternate means of determining total energy absorbed by the illuminated obstacle.

The following different approaches may be used:

- a) Measurement of field strength inside the obstacle as a function of space and determination of the absorbed energy from the volume integral of E^2K where K is the electrical conductivity of the lossy material filling the obstacle.
- b) Measurement of temperature rise for a given time of illumination. The volume integral of all temperature readings divided by the volume itself provides the average temperature rise. Its value, when multiplied by the specific weight and heat of the test substance, provides the total energy absorbed.
- c) Is essentially the same as b), but replaces individual temperature readings by a determination of the average temperature. The latter is established by vigorous stirring of the illuminated test substance immediately after exposure and then measuring it.

The a) and b) approaches have in common that they require many readings, particularly when the distribution of E is very variable. This may be expected almost with certainty in view of the limited depth of penetration of the field in comparison with the dimensions of the obstacles of interest. The b) and c) approaches share the disadvantage that they require more illuminating power in order to provide sufficient resolution in the temperature readings to be of significance. It is felt that the first disadvantage is more serious than the second. Since approach c) is furthermore very much simpler than that of b), the latter is proposed for consideration.

The following data demonstrate how to determine the feasibility of the "thermal approach" c):

| | |
|--|--------------------------|
| Resolution of temperature with thermoprobes | 0.1°C |
| Desirable accuracy in temperature | 10% (min) |
| Size of obstacle to be illuminated comparable with head, trunk, etc. | diameter about 10 inches |
| Thermal time constant of object to be illuminated | about 1 hr. |
| Desirable exposure time (limited to "linear transient" where heat losses to the outside have not yet to be considered) | at most 30 minutes |

| | |
|---|--------------------------------|
| Total absorbed energy flux required to affect in 30 min. a temperature rise of at least 1°C, | about 20 mW/cm ² |
| Total absorbed energy necessary to affect in 30 minutes 1°C temperature rise, | about 10 watts |
| "Available" power requirement over the obstacle cross-section assuming an "absorption efficiency" of 30%, | about 33 watts |
| Total equipment radiant power output, assuming that uniformity requirements are such that the illuminated area absorbs only 1/10th of the power output, | about 330 watts |

In order to be on the safe side, it is desirable to undertake the thermal approach only if about 1 KW of radiant power on an average basis is available. Similar calculations may be carried out for other object sizes. They do not give substantially different results, since objects of smaller size tend to scatter more energy. This offsets to some extent the gain resulting from their smaller depth in the direction of wave propagation.

Considerable effort was undertaken to improve the power output of a radar unit, made available to this laboratory by the Office of Naval Research. As a result a total power output of about 1 KW, as determined with a water load, was achieved.

Figure 1 is included to demonstrate the advisability of positioning the object to be illuminated in a region of the far field where the "shadow cross-section" of the obstacle is not hit by more than 10 to 20% of the total power output. The figure presents that fraction of the total power which is contained by the space angle surrounding the shadow cross-section of the obstacle. It is presented as function of the flux reduction from the center of the beam to the envelope of the space angle for three horns of different gain. The horn characteristics of gain 4 and 6 are calculated assuming $\cos \delta$ or $\cos^2 \delta$ functions for the aperture illumination, the horn of gain 32 is one presently used in our laboratory (experimental data). It is seen that for a 20% variation in flux only 18% of the total power can hit the target, a situation which is considered undesirable for work which is based on the assumption of uniform illumination.

Our present electromagnetic radiation source operates at a frequency near 3000 Mc. Requirements for placing the obstacle to be illuminated only in the distant field and at a sufficient distance so that curvature of the wave front is minimal across the illumin-

ated obstacle, have been outlined in a separate report³, (previously presented and submitted). They are, therefore, omitted here. The choice of horn and room dimensions was made to achieve a tolerable compromise between the necessity of heating the object, its placement at sufficient distance, etc., in accordance with previously outlined principles. Power density was monitored with a probe which was essentially shielded with echosorb, except for its free tip. No highly directional devices, such as horns or dipoles were utilized. They are unnecessary in view of the high power available, and undesirable in view of the directional characteristics of such devices.

A number of preliminary exposures of vessels of the approximate size of the human head filled with proper absorbing solutions have demonstrated the feasibility of the above outlined thermal approach.

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2. Ibid, Chapter III
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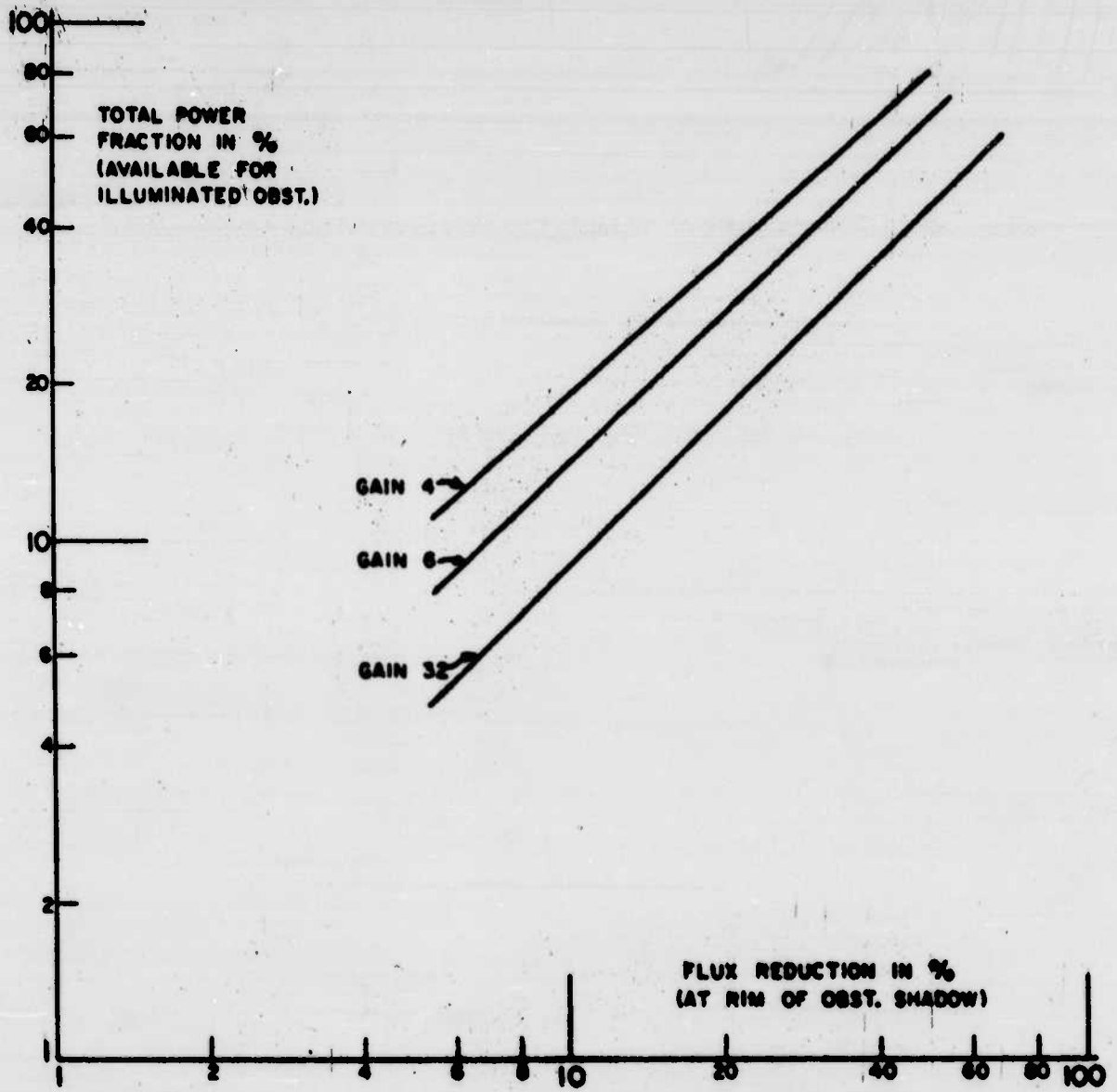


FIG. 1

ELECTRICAL SUBSTITUTES FOR HUMAN TISSUE

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SUMMARY:

Electrical substitutes for body tissues are desirable and necessary in order to evaluate the mode of action of microwaves on mankind. Various mixtures of dioxan and electrolytes have been prepared and their dielectric constant and conductivity determined. The determinations have been carried out at frequencies sufficiently high to establish properties throughout the total microwave frequency range. The chosen mixtures are found suitable to permit simulation of the electrical properties of body tissues. The data will be reported and related characteristics summarized.

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The development of electrical substitutes for body tissues in the microwave range is useful for all purposes where phantom studies, simulating the human body, are planned. A typical example are absorption cross section studies pertaining to mankind and intending to relate mankind absorbed power with free field values of either field strength or energy flux. It had become obvious from previous work carried out predominantly in our laboratory that all body tissues fall within the following ranges of electrical values throughout the total microwave frequency range:

Dielectric constant 5- 70
Specific resistance 10-10,000 Ohm-cm.

Depending on frequency and type of tissue (Hard tissues, soft tissues with high or low water content), a variety of probable combinations of dielectric constants and specific resistances within these ranges are possible. Hence it was decided to develop an electrical substitute for body tissues useful for phantom work which fulfills the following requirements:

1. It must be available in the form of a solution in order to a) permit ease of filling the various cylinders, spheres and other body shapes chosen for absorption cross section studies; and b) be able to be stirred up immediately after exposure in order to establish its average temperature rise.
2. It must permit continuous variation of the dielectric constant throughout the range from about 5 to 70.
3. It must permit independent adjustment of conductivity of the dielectric constant range quoted.

Requirements 2 and 3 are necessary in order to permit successful simulation of any tissue of possible interest at any frequency throughout the microwave spectrum used for radar purposes.

It is obvious that requirement 3 can be met only if electrolytes form an essential part of the mixture. Dioxan is ideally suited as a means of lowering the dielectric constant of water and electrolytes. Consequently a variety of electrolytes of varying electrolytic strength were mixed to different ratios with dioxan and their dielectric parameters determined. The investigations benefited from the following facts:

- a. The frequency dependence of the dielectric properties of an electrolyte is given by a rather simple expression involving only one Debye term

$$\epsilon = \epsilon_0 - (\epsilon_0 - \epsilon_\infty) \frac{(\omega T)^2}{1 + (\omega T)^2}; \quad \kappa = \kappa_0 + \frac{(\epsilon_0 - \epsilon_\infty)\epsilon_r}{T} \frac{(\omega T)^2}{1 + (\omega T)^2} \quad (1)$$

where $\epsilon_r = 8.84 \cdot 10^{-14}$, ϵ_0 and ϵ_∞ dielectric constants at frequencies much lower or higher respectively than $1/2\pi T$. $1/2\pi T$ is about 20,000 Mc for dioxan-water mixtures.¹

- b. The frequency dependence of the dielectric properties of an electrolyte is not essentially affected by the presence of dioxan.^{1*} The latter substance simply lowers the volume taken by the electrolyte and thereby reduces its dielectric constant and conductivity. But it does not affect the characteristic frequency $f_0 = 1/2\pi T$ of the microwave dispersion expressed by equation (1). It merely shifts the constants ϵ_0 and κ_0 . This makes it possible to calibrate all water-electrolyte-dioxan mixtures at relatively low frequencies, thereby determining the constants ϵ_0 and κ_0 as they change with dioxan and electrolyte concentration. The use of equation (1) permits then prediction of ϵ and κ for the total microwave spectrum, using the further fact that ϵ_∞ changes linearly with the mole fraction of water from 2,5 (ϵ of dioxan) to 5 (ϵ_∞ of water).^{1**}

^{1*} See ref.1. A variation of T with dioxan content, as indicated by Cook's work, is statistically not significant.

^{1**} See Ref.1. This linearity has been established by Cook up to a water mole fraction of 0.45. The linear extrapolation to full water content yields $\epsilon_\infty(\text{water}) \sim 5$ in agreement with known water data.

- c. The dielectric properties of pure water mixed with dioxan are already known¹ and serve as a standard check on our method of determination of dielectric properties.

Measurements were carried out to check the validity of the assumption stated above. Pertinent work will not be reported in detail here.

Measurements were carried out at frequencies ranging from 1 to 200 Mc with an RX-Meter developed and sold by the Boonton Radio Corporation, Boonton, New Jersey. Details of the development of necessary sample test cells, correction techniques for distributed inductances and capacitances are not included in this paper since they have been given in greater detail in another place.²

TABLE I summarizes the dielectric constants ϵ_0 for water-dioxan mixtures as obtained in our laboratory, and Figure 1 compares these data with literature values.³ The agreement is seen to be satisfactory. TABLE I furthermore shows dielectric constant and conductivity data for 0.1, 0.033, and 0.01 molar KCl solutions mixed with dioxan. Figures 2 and 3 present these data graphically. The data illustrate:

1. The dielectric constant is nearly independent of the presence of ions. A small decrease with increase in ionic strength, particularly at low dioxan concentration, is in agreement with literature data.
2. The conductivity changes about linearly with the concentration of the electrolyte, particularly at low dioxan concentrations. It furthermore decreases rapidly with increasing dioxan content.

Finally, Figures 4 and 5 summarize some data, previously published by Cook (op.cit.). They apply pure water-dioxan mixtures and pertain to the time constant T and "infinite" dielectric constant ϵ_∞ as function of water content.

TABLE I

| Dioxan (weight-%) | Dielectric Constant ϵ_0 | | | | Conductivity κ_0 (mMho/cm) | | |
|----------------------|----------------------------------|-------|------|------|-----------------------------------|-------|------|
| | 0 | n/100 | n/30 | n/10 | n/100 | n/30 | n/10 |
| 0 | 78.5 | 78.0 | 76.4 | 76.0 | 1.43 | 4.78 | 13.0 |
| 10 | 69.5 | | | | | | |
| 20 | 62.4 | 61.2 | 61.0 | 60.7 | 1.09 | 3.23 | 9.00 |
| 30 | 54.7 | | | | | | |
| 40 | 45.7 | 45.4 | 47.0 | 44.0 | 0.745 | 2.16 | 5.73 |
| 50 | 37.8 | | | | | | |
| 60 | 28.3 | 28.5 | 28.9 | 29.0 | 0.439 | 1.25 | 3.12 |
| 70 | 20.2 | | | | | | |
| 80 | 12.6 | 12.9 | 14.3 | 15.6 | 0.119 | 0.287 | 0.59 |
| 90 | 6.63 | | | | | | |
| 100 | 2.35 | | | | | | |

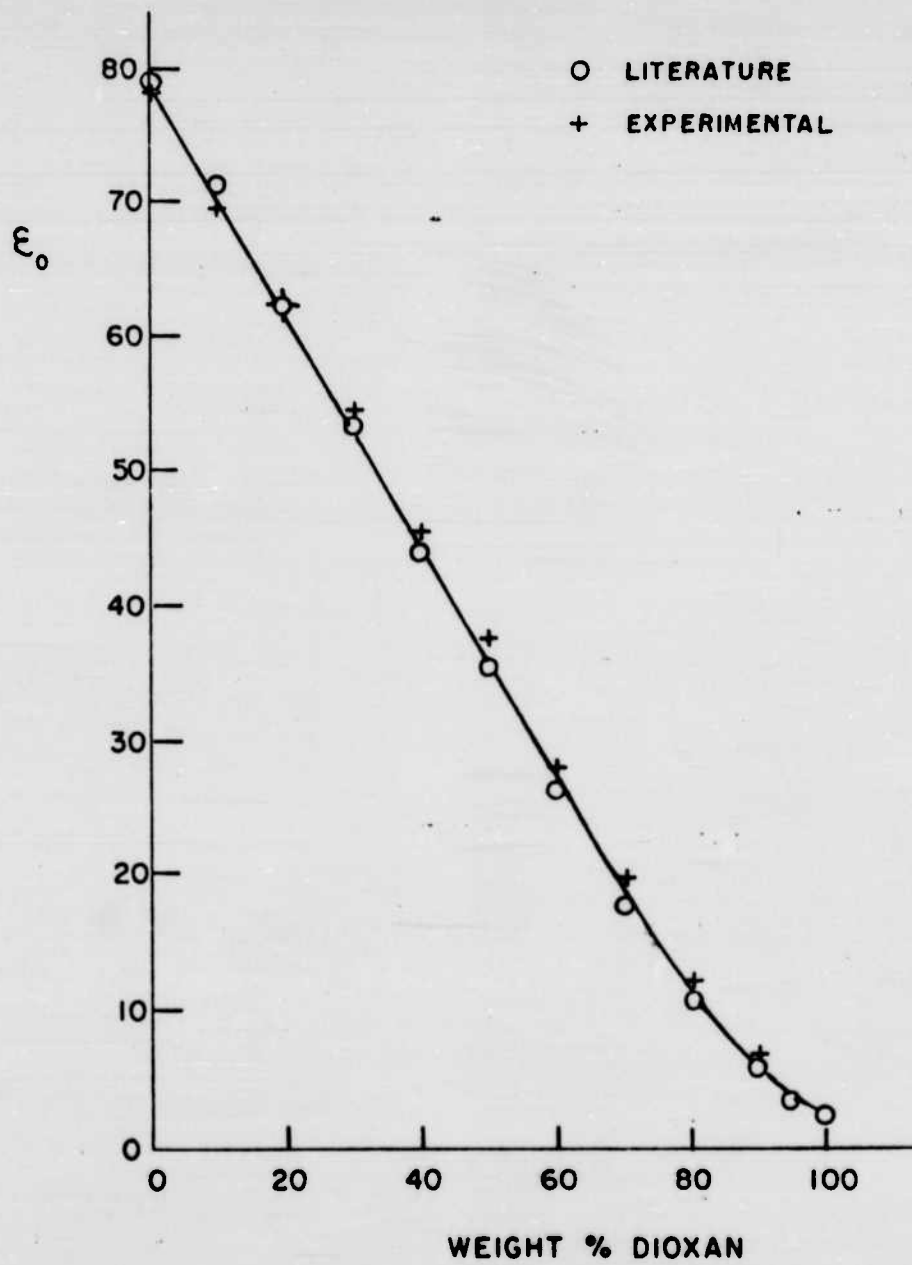
Dielectric constant ϵ_0 and conductivity κ_0 (in mMho/cm) for various KCl-dioxan-water mixtures. The data are given as function of the weight percentage of dioxan and pertain to water and molar KCl-solutions. All data hold for a temperature of 25° C. The conductivity increases with temperature by about 2% per °C temperature change, and the dielectric constant decreases about $\frac{1}{2}$ unit per °C. Estimated accuracy about 2%.

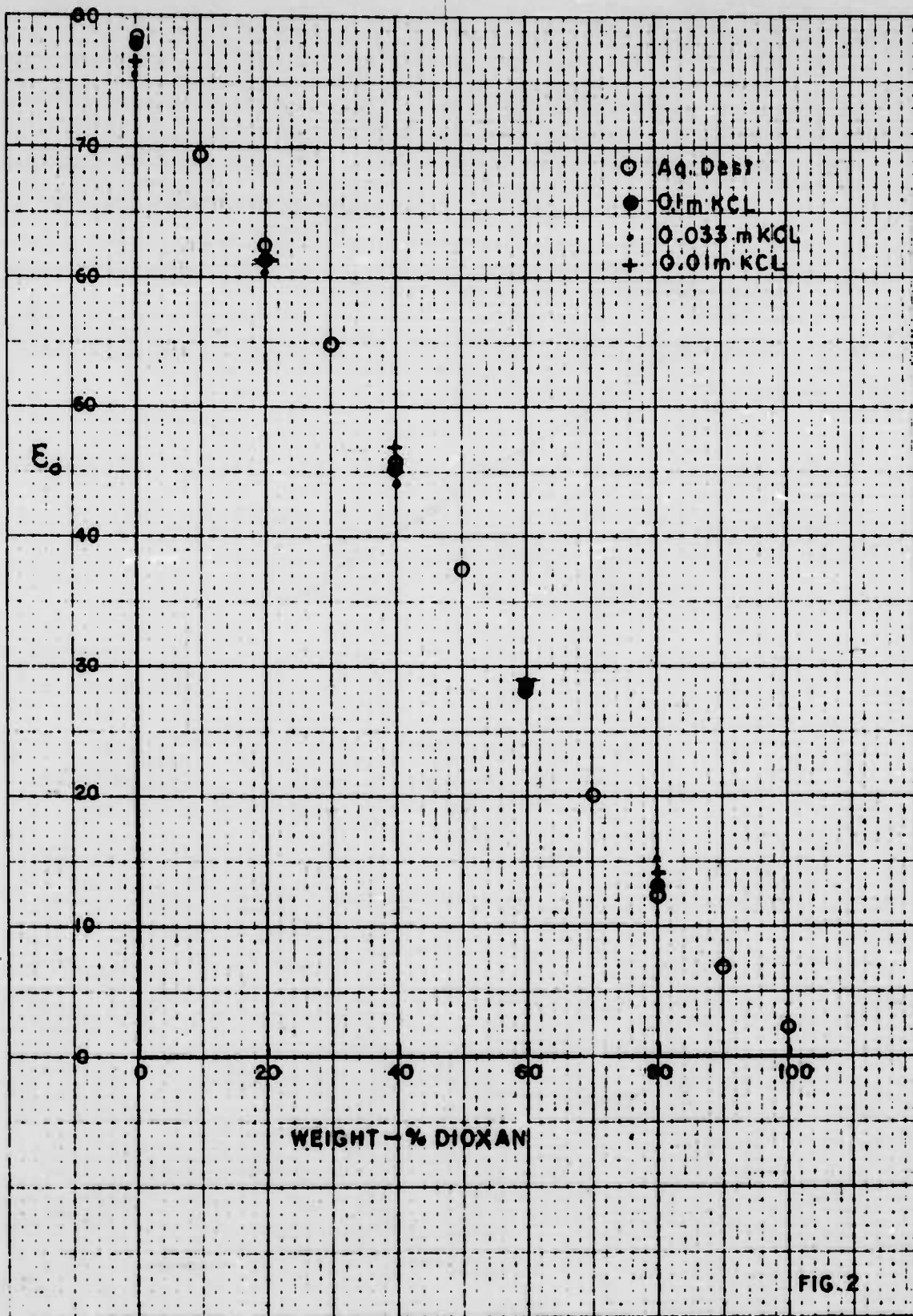
Since in the above table and figures the constants $\epsilon_0, \epsilon_\infty, \kappa_0$ and T have been presented, equation (1) permits the determination of ϵ and κ for water-electrolyte-dioxan mixtures for any frequency of interest, provided that the electrolyte is KCl. Usually it will be found that the data are identical with the "low frequency" data ϵ_0 and κ_0 given in Figures 2 and 3 and TABLE I. Corrections, utilizing equation (1) become important as the frequency increases above 1000 Mc and are simple in application. The realization of a particular suspension of defined dielectric properties is easy with charts 2 and 3: Graph 2 permits statement of the dioxan weight percentage to yield a given dielectric constant, and chart 3, in combination with the desired conductivity, establishes by interpolation the concentration of the required KCl-solution.

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2. ONR-Report, Contract 551(05), July 1, 1959.
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Figures 4 and 5 are based on Figure I of "The Dielectric Behavior of Water in Water-Dioxan Mixtures", by H. F. Cook, from the Transactions of the Faraday Society, No. 343, Vol. 47, Part 7, July, 1951.





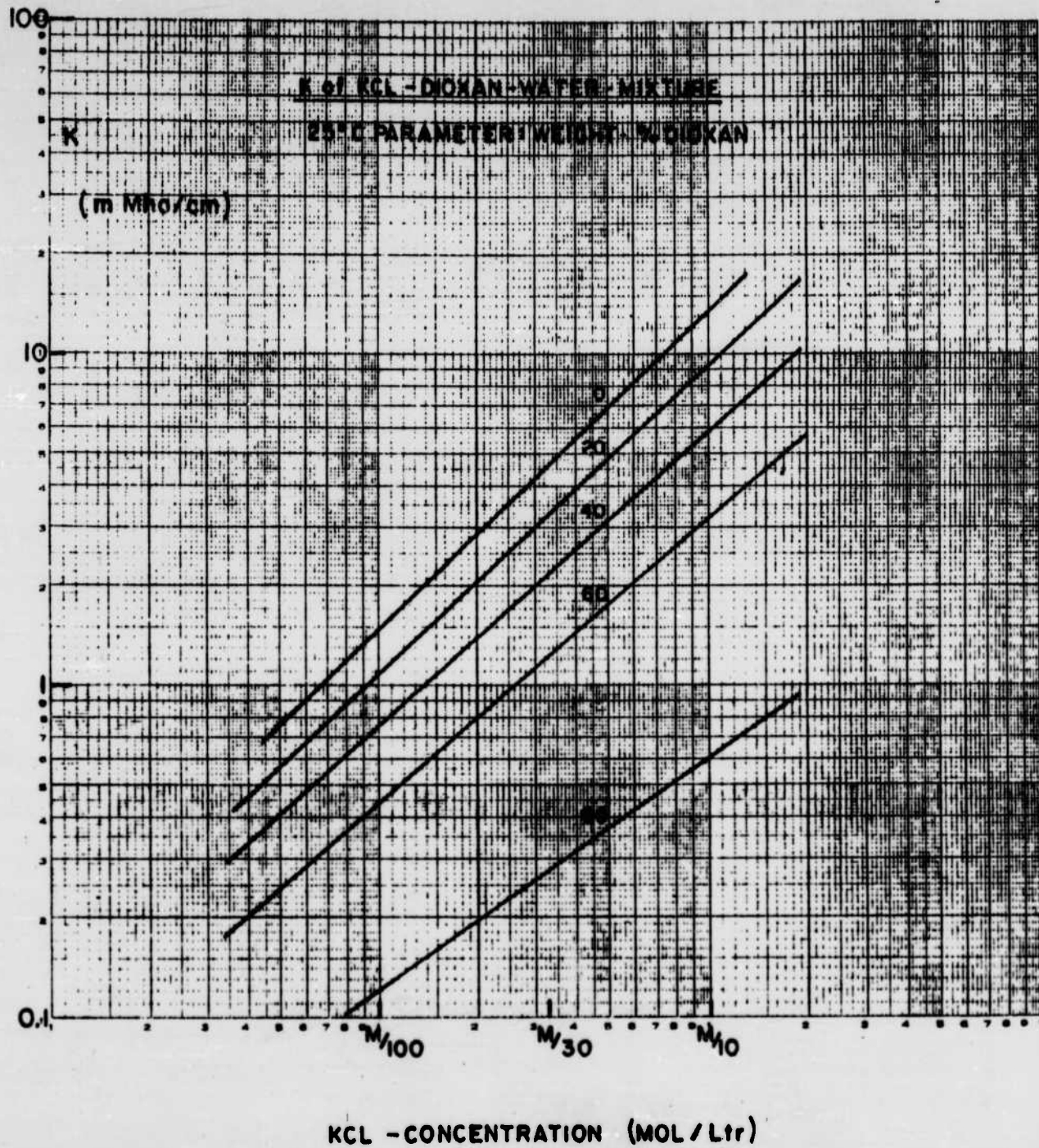
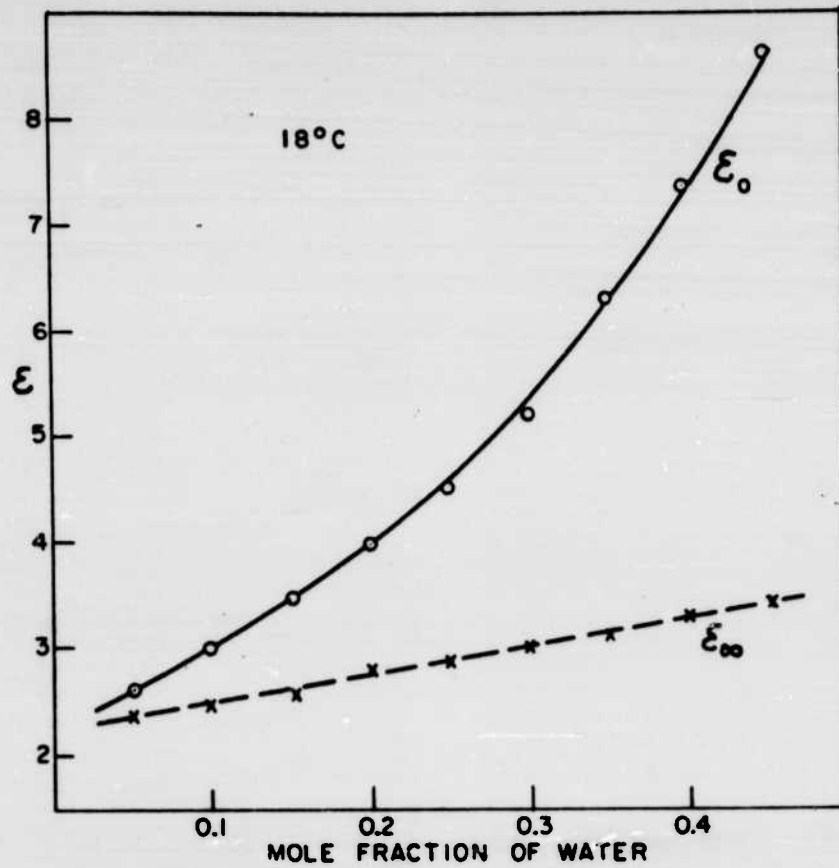


FIG. 3



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FIG. 4

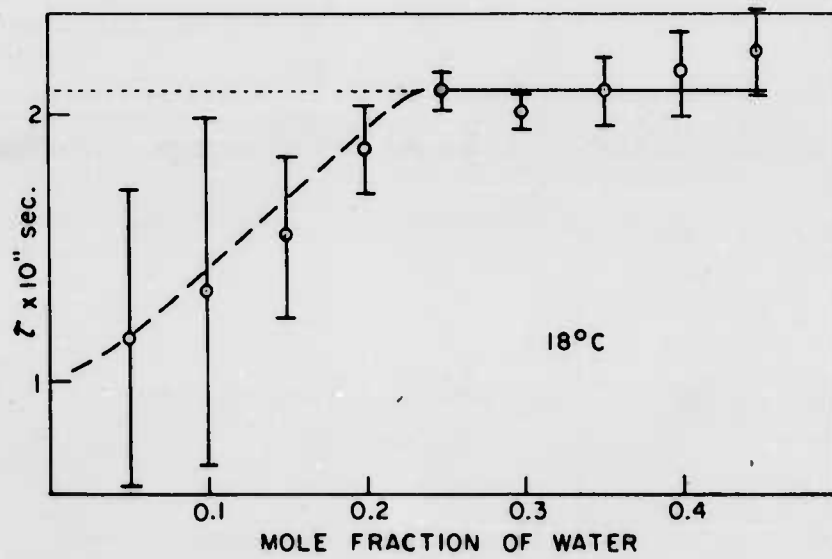


FIG. 5

SOME RECENT DEVELOPMENTS IN PULSED ENERGY SLEEP

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Summary

Pulsed Energy Sleep is a general state of inhibition of the cerebral hemispheres brought about by direct action of low intensity and low frequency pulsating currents with a very short pulse duration. It is entirely different from electroshock and electronarcosis, and is based on functional parabiosis and protective inhibition concepts. Electroencephalographic, electrocorticographic and electrothalamographic studies prove cortical inhibition and parabiogenic nature.

Under the effect of pulsed energy subjects fall to moderately deep sleep, which resembles in many aspects to physiological sleep.

This procedure is entirely safe, free from side effects, and undesirable reactions like tonic-clonic seizures, respiratory or cardiac accidents.

Pulsed Energy Sleep procedure offers vast potentialities as an investigative as well as an effective therapeutic tool.

Rand Development Corporation has recently been interested in some basic neurophysiological work in relation to higher nervous activities of human beings. While searching for non-pharmacological means to influence central nervous system without causing side reactions or sequelae, our attention was brought to a Russian instrument utilizing pulsed energy. Since then we have spent considerable effort to gather as much information as we could about this technique, and have tried to visualize its vast potentialities. Colonel George N. Knauf, from Tri-Service Committee, expressed his wishes that we should reflect these informations at the Third Tri-Service Conference on the Biological Effects of Microwave Energy, and General Flickinger encouraged us to do so. We would like to extend our appreciation to General Flickinger and Colonel Knauf for their suggestion and advice.

Our thanks are also due to the Programming Committee of the Directorate of Technical Services, and particularly to Mr. H. S. Brownstein, Secretariat to the Tri-Service Committee, for their kind invitation and for giving us ample time for this presentation. ———

I have deliberately omitted the name of this instrument to eliminate a possible confusion in the minds of audience before entering the subject. This new technique and its implement is called, in Russian, "Electroson", which has been translated into German as "Elektroschlaf", and into French as "Sommeil électrique". It could be coined in English either as "Electro-sleep" or "Electric Sleep". Although such a designation would be correct from etymological and semantic standpoints, its phonetic similarity to electroshock has an important and even hazardous consequence. It easily could convey the idea that it is a form of electroshock, which it is not. Because of this consideration it is advisable to use another, and more descriptive designation. We suggest, therefore, the term, "Pulsed Energy Sleep". Such a terminology will not only help the physician differentiate it from electroshock and electronarcosis, but it will particularly provide a sense of security, and therefore acceptance by the patients. It has been the uniform experience of Russian investigators that they met with resistance from their patients because the patients did not want to receive electroshock treatment.

We felt, therefore, it necessary to clarify, first of all, the independence of pulsed energy sleep from electronarcosis and electroshock. After definitions we will try to give a faithful picture of the work on pulsed energy sleep which has been in progress in Soviet Russia for the past 10 - 12 years. While doing this we will limit ourselves to the duties of an abstractor rather than a critic of the Russian publications. We feel that only in this way can we fulfill the intended purpose of this presentation: i. e., to relay more information to you as it appeared in Russian. In this way we hope that many questions, which will undoubtedly arise in your minds, will help to stimulate and initiate research in this field.

Electronarcosis is a forced condition of the central nervous system brought about by application of either interrupted continuous current (square waves), or alternating sine waves of 60 - 150 ma. with a pulse rate of 100 cycles per second. Considerable intensity of the current causes spread of electrical front to large areas of the cortex, subcortical area, brain stem, and if it is continued, penetration to deeper layers, reaching finally to medulla. Based on this, a

progressively advancing front of electrical stimulation develops which may be represented, schematically, as

Initial hypnotic narcotic convulsive coma

Due to the intensity of the current, the clinical signs of these phases proceed very fast, and the subject enters into a narcotic stage in 15 - 30 seconds. Total immobility, hypotonicity of the muscles, loss of all forms of sensibility, and unconsciousness indicate diffuse inhibition of the cerebral cortex. However, if the current is kept on, the increase of the tonus of subcortical-stem sections is witnessed by the change of myogenic tonus, appearance of elements of spastic phenomena, and hyperkinesis. Finally, as the front of electrical stimulation reaches to deeper sections of the central nervous system, the medulla becomes involved, respiratory and cardiac embarrassments appear. It is therefore necessary to limit the time of application of the current to 5 to 7 minutes.

Electroshock, or more descriptively, electrocoma is a very fast shift of the patient to the stages of convulsion and coma. As it is known from practice, patients are suddenly charged with 100 - 500 ma. of sine waves for a duration of 0.6 - 1 second. The result of stimulation of both cortical and subcortical areas is the development of signs of severe tonic and clonic seizures requiring pre-treatment with muscle relaxants to prevent rupture of muscle bundles or fracture of bones. In addition, respiratory difficulties with occasional cardiac arrest may occur.

Pulsed Energy Sleep is an entirely different phenomenon from electroshock and electronarcosis. It is based on the concepts of "parabiosis" and "protective inhibition". Pulsed Energy Sleep develops as a reaction to the passage of a very weak alternating current with some d-c component through the brain tissue. The intensity of the current is not higher than 0.2 ma. with a pulse duration of 0.3 milliseconds. The pulse rate varies between 1 and 100, according to the age of the patient and the type of the case under treatment.

This type of energy has been proved to be free from any side reactions like intoxication, electric accumulation, and opisthotonus, or any adverse effect upon respiratory or cardiac centers.

Proper understanding of Pulsed Energy Sleep requires familiarization with the "doctrine of parabiosis" of Wedenskiy, if we are to see the facts as a Russian investigator or a reader would understand

it. There is another reason for me to explain this doctrine, and I am very reluctant to say so, because in one of the recent medical meetings in this country, this problem came up, and the explanations given were very confusing, if not entirely erroneous!

The history of parabiosis starts with the interesting observation of Grunhagen, made in 1876. This author found that if a portion of a nerve fiber is placed in a glass tubing and exposed to a flow of carbon dioxide, the irritability of the nerve at the exposed part steadily decreases up to its complete loss. The conductivity, on the other hand, remains intact. A conclusion was drawn from this observation that the "irritability and conductivity should be separated from each other as different processes." Grunhagen's experiments were repeated by others, and with various agents like chloroform, ether, alcohol, carbon monoxide, and chloralose. The results obtained were uniformly corroborating. A French physiologist, Herzen, went a step further and applied an induction current to the non-irritable portion of the nerve. He could detect a negative deflection of the galvanometer needle, although there was no action; he, therefore, called this "les courant d'action sans action" - "action currents without action".

Nikolai Evgenevich Wedenskiy, professor of physiology at Leningrad and a successor of Sechenov, who discovered central inhibition of the reflex arc by applying strong salt solutions to the central nervous system, repeated Grunhagen's experiments in the following manner:

He made a nerve-muscle preparation conserving the severed part from the spinal cord, and carefully brushed chemical agents to it or applied other noxi at a section of the nerve as shown with shaded area in the Figure 1. Agents like cocain, chloralhydrate, phenol, as well as mechanical, thermal, and electrical agents were used in his experiments. Wedenskiy, too, noticed that as a response to these noxious agents the irritability would drastically decrease to necessitate stronger stimuli so as to bring about minimal contraction on the muscle. Conductivity, however, seemed to be left intact because application of a minimal induction current at point A could cause contraction of the muscle as before. However, conductivity was later also abolished "suddenly" because the stimuli at any intensity would fail to cause a contraction. In addition to these Wedenskiy observed, through his telephone, another phenomenon: at a stage when low intensity impulses were still passing through the blocked part, the nerve tone in the telephone changes, it loses its pure musical tone and becomes muffled. Wedenskiy called this "transformation stage."

As time passes, the treated or poisoned area of the nerve still undergoes some changes, and at this time response to strong stimuli

disappears, but weak stimuli bring the muscle to a tetanized state. This phase was called "paradoxical stage."

Paradoxical stage is followed by a further lessening of the irritability at the treated point, which had started earlier. In conjunction with this another fact was observed by Wedenskiy. During paradoxical stage, or the stage immediately following to this -- during which conductivity of treated area seems to have completely disappeared -- stimuli coming to this portion of the nerve cause an inhibitory effect upon the stimuli applied to the treated area. This was called, correspondingly, "inhibitory stage." The actual meaning of this stage was the ability of a portion of a nerve to exert an inhibitory effect upon another section of the same nerve. In the absence of any histological changes, the only explanation which could be given to this peculiarity is a change in the functional state of different parts.

The entire pattern of this reaction with its various stages was recognized by Wedenskiy as a "general reaction of the nervous tissue to most varied stimuli," and he considered it "even more universal than their state of stimulation or state of activity in ordinary meanings of these terms."

Wedenskiy chose the term "Parabiosis" for this peculiar condition. When this condition was fully developed the nerve seemed to be void of its fundamental characteristics -- irritability and conductivity -- as if it were dead. If we stop for a moment, and remember that in Russia a tradition of neurophysiology exists, then the impacts of such a broad formulation as the doctrine of parabiosis could be grasped fairly easily. The possibility of inhibition of a nervous tissue by another part of the same unit could be advantageously used to explain various normal as well as pathological phenomena.

We see that Pavlov, too, was interested in parabiotic inhibitions as these are reflected in his writings. He stated that "constant stimulants without after effects lead to development of inhibitive conditions in the cortex. This inhibition gradually irradiates to occupy the entire cortex, and descends to the subcortical layers." This point was proven by various experiments in Pavlov's laboratory. For instance, as early as 1912, while studying conditioned reflex, it was found that intermittent electrical stimulations of the skin did cause not only secretion of saliva, but the animals uniformly fell asleep. These observations led Pavlov to study the mechanism of natural or physiological sleep. He denied the observations of Hess that the stimulation of lateral portion of the ventral half of the massa intermedia of

thalamus induces sleep in cats. He assumed that this represented an exceptional case, and the sleep was not brought about by stimulation of the sleep center but by transfer of the electrical stimuli to cortex by some tracts. Pavlov called the inhibition of the cortex brought about by external irritants "protective inhibition" and classified these irritants into three categories: very weak, very strong and extraordinary. Stimuli with or without physiological meaning can introduce various degrees and depth of inhibition in the central nervous system.

Based on Wedenskiy and Pavlov concepts, minimal pulsed energy has been applied to central nervous system as a weak, monotonous, rhythmic stimulant to induce generalized cortical inhibition, and it was observed that as a result of this inhibition a state develops in animals which could be compared with the transitory phases between vigilance and sleep.

The above given considerations led the investigators at the Institute for Higher Nervous Activities, Psychoneurological Research Institute of Academy of Medical Sciences, Electrophysiological Laboratory, and Physio-Technical Department of the Institute of Physiotherapy to participate to a collective work for evaluation and human application of pulsed energy.

Following a rather detailed study of physiological and clinical aspects of electronarcosis, they came to the conclusion that the only admissible form of pulsed energy for clinical use would be a current of minimum intensity, frequency and pulse duration, which will not cause any unpleasant sensations, vegetative displacements, or spastic-kinetic manifestations. It was therefore necessary to select and define these criteria. Electrophysiological considerations led them to choose rectangular waves, initially, with very short duration at their peak, and a rapid drop to zero. Due to relatively long pause between pulses helped, at the same time, to bring the time average of the intensity to a lower level than the amount which was actually applied to the subject. Frequency of pulses were studied in a spectrum from one to 130 cycles per second, and it was found that although the great majority of cases responded to frequencies between 5 and 25 cycles per second, it should be selected in each case individually. Duration of pulses should correspond to moving chronaxies of the nervous formations which lie between 0.3 - 0.5 msec. Various wave shapes like rectangular, trapezoid, bell-shaped, and combinations of these have been tried, and a conclusion was arrived at that all possible wave forms should be investigated to find out the most physiological patterns.

After these criteria have been established, it was a simple matter for electronic engineers to construct working models. The first clinical model was built at the Institute of Physiotherapy in 1950, and since then several changes have been made on the basic circuitry. Recently transistorized units and portable models have been constructed.

The pulses generated in these units are applied to the patients with orbital and occipital electrodes. Orbital electrode represents cathode, and occipital electrode represents anode. Occipital electrode is made in a bifurcated form to fit on the mastoid processes.

After the electrodes have been placed the current is turned on, and its intensity is increased until the threshold of the patient is reached. This corresponds to a level at which the patient feels a slight prickling sensation or crawling ants under the eyelids or in the back of orbital cavity. Gradually a heaviness develops in the eyelids, sometimes after a slight dizziness and drowsiness thoughts disappear and patients fall into a steadily deepening physiological sleep. During this the patients will be found in a quiet, relaxed position, usually lying on their side. Respiration becomes deeper, less frequent, but even; pulse slows down to a few less beats per minute. Upon discontinuation of pulses patients awaken in a few minutes; however, sleep may continue more or less longer after interruption of the electrical current.

Four types have been distinguished as reaction to pulsed energy:

- (1) Without perceptible change in the condition of the patient.
- (2) Development of sleep toward the end.
- (3) State of drowsiness interrupted periodically with short cycles of actual sleep.
- (4) Continuous sleep during procedure.

With the exception of children, almost all patients fail to fall asleep during first application (fear of being attached to an electrical circuit, personal experience with electroshock, etc.). After discovering the absence of any unpleasant sensations and lack of after-effects, patients become firmly assured so that they fall asleep without any difficulty, and accept the treatment with willingness. However, certain percentage of them will never fall asleep; these make up the actual representatives of the first group in above given classifications.

Physiological and Biochemical Changes during the state of cortical inhibition in animals were investigated by means of conditioned reflex activity as used earlier in Pavlov's laboratory. However, these methods present certain difficulties when they are applied to human beings because they are actually conditioned reflexes by themselves. Therefore, to study sleep inhibition kinetic chronaxie, skin galvanic potentials, registration of the movements of the eyelid, etc. were used. Since the development of electroencephalography this technique has been in extensive use. Present studies have been undertaken mainly by means of pneumographic, plethysmographic and electroencephalographic techniques. Besides this arterial O₂ saturation and complete biochemical analyses of blood were done.

Pneumograms showed slower frequency with increment in depth of inhibition, and periodic undulations which are characteristic for the hypnotic phase of sleep. Plethysmograms showed a slow, gradual dilation of peripheral blood vessels. Appearance of respiratory waves as well as waves of third order indicated the normalization of plethysmogram which is disturbed in psychiatric cases. Arterial oxygen saturation remains high during the procedure in spite of deep sleep and vasodilation. This is contrary to a sharp fall in arterial O₂ content during physiological as well as pharmacological sleep. Electroencephalographic changes may be summarized as initial depression of the alpha rhythm, then appearance of slow waves, and during more prolonged action some increase of the high frequency waves in the frontal region (Figure 2).

The depth of sleep was studied by means of combination of conditioned, unconditioned and defensive reflexes coupled with electroencephalographic recording of electrical activity of the brain. Evaluation of the data obtained according to accepted criteria allowed for establishing four phases of Pulsed Energy Sleep which are characterized by various degrees of diffusion and depth of the inhibition.

- (1) Absence of inhibition - no visible change in the status of the subject.
- (2) Partial inhibition - hypnotic phase.
- (3) Considerable irradiation of inhibition extending to the auditory analyzer - sufficiently deep sleep.
- (4) Spread of inhibition to subcortical - stem level - state of deep sleep.

In the majority of patients the second and third phases were reached showing very close relationship between physiological sleep and pulsed energy sleep. Continuation of electroencephalographic changes following interruption of the current, and its resemblance to that obtained during normal sleep corroborates this similarity. However, it is proper to add a qualifying statement in this connection, that Pulsed Energy Sleep is by no means the physiological sleep itself. Its manifestations are very similar to it, and it may be followed by it, but in the body pulsed energy produces deeper changes than those which could be obtained by a few additional hours of sleep brought about by indifferent rhythmic stimuli. Even after a short and shallow sleep during the procedure of pulsed energy application, the patients feel refreshed and in good spirits just as after a deep and long night sleep.

Site of Action of Pulsed Energy in the Central Nervous System is one of the problems to be decided upon. While defining pulsed energy sleep, it was stated that it is the result of direct effect of the pulsating currents upon nerve cells. It is obvious that, as long as electrodes are applied externally, peripheral nerves will participate in the development of inhibition. Due to its significance this aspect was studied first by implanting intracranial electrodes in rabbits. By gradual advancement of the probes from the skin, through brain mass, and reaching to the base of the skull, it was found that current density shows two maximums: immediately under the skin, and near the base of the cranium. In the brain tissue itself the current density decreased from base of the skull toward calvarium. This proved that the major portion of the applied current was passing directly through the brain tissue, and basal area of the brain was subjected to the effect of current more than the cortex.

In another group of experiments, electroencephalogram, electrocorticogram, and electrothalamogram were recorded during and after the application of square-wave pulsating currents. Development of certain pattern under the effect of these pulses with or without sleep, continuation of these changes after prolonged application of the procedures, and their resemblance to those obtained during physiological sleep have been accepted as signs of a direct effect of the pulsating current upon brain cells. These experiments also helped to eliminate the question of peripheral irritation in the mechanism of sleep induction because electroencephalographic recordings obtained during sleep were similar regardless whether the energy was applied intracranially or extracranially. Likewise in 40 cases with open trauma to the head of human beings by placing active electrode to exposed brain, no changes could be seen in electroencephalographic tracings as compared to those obtained by conventionally applied electrodes.

A comparative study of electroencephalogram, electrocortico-gram and electrothalamogram before, during, and after sleep disclosed some additional information which can be summarized as follows: during pulsed energy sleep optic thalamus is stimulated to give rise to small amplitude optimal rhythm (5 - 6 firings per second), which slows down after the inhibition is developed in the cortex. This retardation is parallel to the duration, as well as depth of sleep. After the awakening of the animal normal rhythm of excitations appear in electroencephalography.

Electroencephalogram on the other hand gives an optimal rhythm for cortical structures of 8 - 12 cps alpha rhythm. The unchanged responses of thalamus in animals and in humans to a pulse rate of 6 - 10 - 12 or even 18 shows that the brain tissue is reacting independently from the excitor, and with its own oscillation rate; this too was explained in the theory of parabiosis of Wedenskiy as the internal nature of parabiosis. This way the mechanism of action of pulsed energy becomes clear, once again, that nerve cells resume their normal rate of firing, or in Wedenskiy's terminology, their functional lability.

Application of Pulsed Energy Sleep has been carried on in approximately 500,000 procedures. A great majority of these cases comprise neuroses, schizophrenia, choreic form of rheumatic encephalitis, presenile psychosis, hypertension, duodenal ulcer and toxemia of early pregnancy and serious forms of headaches. Discussion of indications, therapeutic regimes and detail of results obtained is beyond the scope of this conference. However, we can summarize the results of findings with some broad statements: with the exception of presenile psychoses Pulsed Energy Sleep has provided, in the hands of Russian investigators, a safe and effective means of treatment. Usually from the very first procedure on patients became calm, lively, cooperative, critical toward their disease. Hallucinations, suicidal tendencies disappeared, disturbed night sleep became normal. In children with chorea hyperkinesis disappeared, and no recurrences observed.

However, it should be added that therapeutic success depends, largely, on the state of development and reactivity of the thalamo - cortico - subcortical relations of the patients. For instance, in schizophrenia in cases where no beneficial effect is obtained alone from Pulsed Energy Sleep, combination with insulin - shock or other known means of therapy leads to recovery.

Conclusion

To conclude this presentation on the biological effects of the pulsed energy in which we have tried to expose only its most salient aspects as it pertains to the realm of this Conference, the following could be repeated.

Pulsed Energy Sleep is a parabiologic - protective inhibitive state of the central nervous system achieved by direct action of low intensity, low frequency, and short duration pulsating current upon the nerve cells, and developed in these cells. This cortical - subcortical inhibition restores the neurons to oscillate with their own rate of firing.

Pulsed Energy Sleep is for this reason entirely different from electroshock and electronarcosis, because the latter are forced conditions in the brain with certain, and sometimes dangerous, side or after effects.

Pulsed Energy Sleep has been used for therapeutic purposes with definite success and could be tried as an investigative tool with definite advantages. Further research for its effects as well as possibilities which might be derived from it deserves serious consideration.

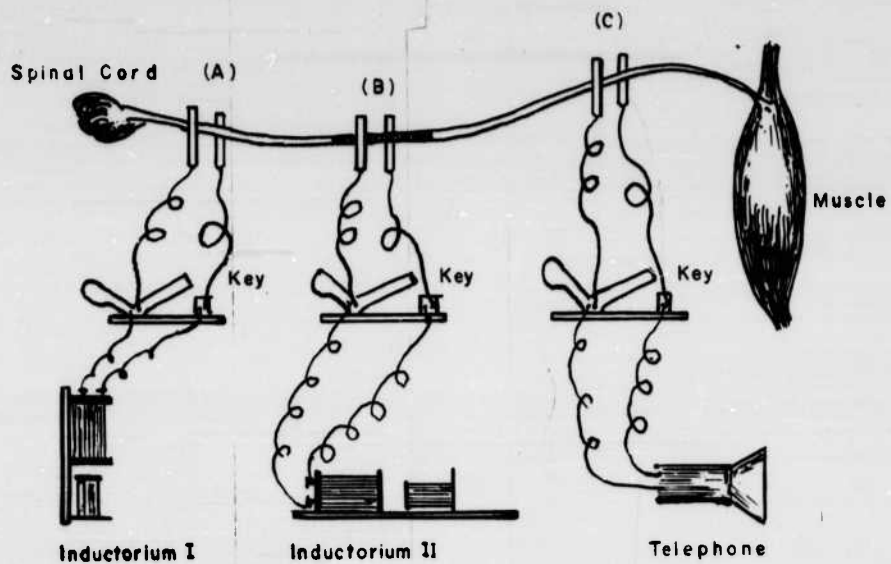


Fig. 1. Wedenskiy nerve-muscle preparation to study parabiogenic phenomena

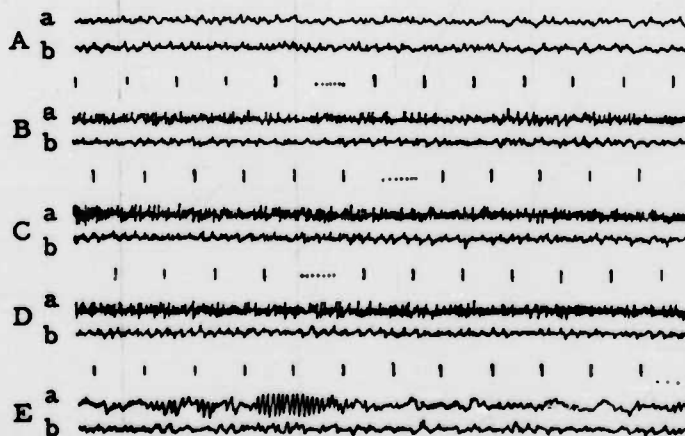


Fig. 2. Electroencephalogram (a) and Electrothalamogram (b) of rabbit. A - before application of pulsed current. B, C, D, E tracings at 15 minute intervals following application of the current (after I. S. Robiner).

HUMAN BODY AS AN INCONSTANT HEAT SOURCE AND ITS RELATION TO CLOTHES INSULATION

1 - Descriptive Models of the Heat Source

by

A. S. Iberall*

ABSTRACT

A precise characterization of the thermal resistance of clothes requires an accurate description of the static and dynamic thermal characteristics of the human-heat source. Experimental measurements on the human have revealed a frequency spectrum of sustained thermal-power oscillations that mask theoretical long-time equilibrium adjustments. This points to the number of degrees of freedom that must be involved in the thermoregulation of the human, and to the specific nonlinear characteristics of the system. Therefore, at best, a resistance model for clothes is possible only as an ohmic relation among time-averaged equilibrium values, and for a specific mode of operation of the system. The validity of this hypothesis, however, has not been proven.

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Human Body as an Inconstant Heat Source and Its Relation to Determination of Clothes Insulation¹

A. S. IBERALL

1 - Descriptive Models of the Heat Source

A client of the Rand Development Corporation requested development of a method for assessing, among a variety of clothes of interest to him, the differences in their ability to provide protection against extremes of temperature. Because of some rather novel and controversial elements in the method of providing protection in these clothes, the client requested that the method be absolutely reliable and highly accurate.

A review of the literature revealed the following methodology for describing and determining the "insulation" value of clothes. Clothes are described as an equivalent "conductance". For example, Gagge, Burton and Bazett (1)¹ defined the "clo" - a unit of thermal insulation of clothing - as the amount of insulation necessary to maintain comfort (a mean skin temperature of 92 F) of a resting subject in a room at 70 F with air movement of 20 fpm, humidity not over 50 per cent, with a metabolism of 50 kilocalories (k cal) per sq meter per hr. Equivalently, they have taken this to mean that, on the assumption that 76 per cent of the heat is lost through clothing, a clo may be defined in physical terms as the amount of insulation that will allow the passage of 1 k cal per sq meter per hr with a temperature gradient of 0.18 deg C between the two surfaces of clothing. Much of the ideology and experimental science that has gone into developing these concepts may be found in the authoritative book "Physiology of Heat Regulation and the Science of Clothing," by L. H. Newburgh (2); a book by Winslow and Herrington (3); and in a published symposium on temperature (4).

Whereas as a goodly first approximation, there may appear to be little criticism of the "equivalent conductance" concept of clothing, for precise work a very carefully constructed

scientific foundation in theory must be laid to justify such a concept.

The specific difficulties are to determine, not how to make a laboratory test of the equivalent thermal resistance of a sample of clothes, but, how the body determines its surface thermal boundary conditions so that its characteristics as a potential source are understood. In particular, since the body is a self-actuated thermoregulated system, what is needed is a dynamic description of its operation. It is in the domain of lack of pointed emphasis on the dynamics of the system that past descriptions of the effect of clothes are weak.

Related difficulties existed in the science of color which were not resolved until distinctions were made between the psychology of color (how the eye characterizes color); the psychophysics of color (by what physical laws does the eye respond to or "add" color); and the physics of color (the physical laws that describe color).

It is the purpose of this preliminary study to lay at least a phenomenological basis for a valid description of the thermoregulation of the human that will permit a precise characterization of the effect of clothes.

DESCRIPTIVE MODELS OF HUMAN HEAT SOURCE AS A DYNAMIC THERMOREGULATED SYSTEM

The chemically reactive thermal field that the body represents has been described by the following power balance:

$$M = E + W + K (T_s - T_o) + S$$

M = rate at which energy is produced by the heat of combustion (i.e., metabolism)

E = rate at which energy is lost through evaporative mass transfer (i.e., evaporation)

W = rate at which external work is done

S = rate at which heat is stored in the system (i.e., storage)

K (T_s - T_o) = rate at which heat is conduc-

¹ Numbers in parentheses designate References at end of paper.

ted through an insulating sheath in contact with the body.

K = "equivalent" conductance of insulation surrounding the body

T_s = "effective" skin temperature

T_o = "effective" surrounding temperature

In this description, clothes are characterized by their effect in determining the conductance K .

The physiological description of the thermoregulation of the human, given in the literature, describes three regions of regulation (illustratively, for a nude subject doing no external work). In the expressions that follow, zero subscripts are used to denote parameters that may be regarded as constant. Numerical temperatures given are only meant to be approximately correct values.

Evaporative Control

$$M_o = E + K_o (T_{s_o} - T_o)$$

for $T_o > 31$ C

T_{s_o} = constant (= 35°C)

See (4), for example (pp.33, 518, 549); or (2) (pp. 142, 205).

The implication has been permitted that E becomes a function of T_o , say through the potential mechanism of increasing the wetted surface area of the body.

Vasomotor or Conductance Regulation

$$M_o = E_o + K_o (T_s - T_o)$$

for 31 C $>$ $T_o >$ 29 C.

See (4), for example (p.548)

The implication has been permitted that the internal conductance of the system changes, so that the skin temperature becomes a function of ambient temperature.

Zone of Body Cooling

$$M_o = E_o + K_o (T_s - T_o) + S$$

for 29 C $>$ T_o

The conditions of measurement are not at equilibrium. A heat debt is built up. It is left by logical implication that the control is through shivering and increased metabolism, see (2) (p. 235, next to last paragraph).

The defects in this general description are that it is not complete - it simply represents a partial balance that contains no information about the dynamic state of the system; and the effective conductance is not satisfactorily treated - it does not lump all fluxes that may pass through the clothes.

An elementary energy equation that may be written, more correctly, is

$$M = L + K (T_s - T_o) + (dH)/(dt)$$

M = metabolism

L = rate at which heat is lost through all other avenues than through clothes

$K (T_s - T_o)$ = rate of all sources of flux that pass through clothes

$(dH)/(dt)$ = rate at which thermal energy is stored in system

The assumption that all sources of flux, passing through clothes, regardless of their further partition, may be described by an equivalent conductance is a hypothesis. Its general validity may be borne out by careful study of (6) (chapter 28). It is shown, as a tremendous achievement of the theory of similarity, that there is considerable similarity in the results of temperature difference driving forces and concentration difference driving forces. Thus under the assumption of small concentration gradients, the combined radiative, conductive, convective, diffusive exchange at the surface of the skin, between the skin and clothes, may be regarded as functionally homogenous in the temperature difference $T_s - T_o$. However, this does not necessarily imply that the relative contributions of each mode of heat exchange remains a constant proportion of the whole: i.e., the equivalent conductance is no longer necessarily a constant.

The second significant aspect of this description is that beginning to describe the system by means of a differential equation creates the possibility of ultimate description of its dynamic performance. For example, this formulation states that the net rate of production of heat is instantaneously stored in the system. Since it is reasonable to assume that no phase change takes place instantaneously in the body, one may assume plausibly that the storage takes place in heat capacity of the system. Thus

$$M - L - K (T_s - T_o) = C_v W (dT_b/dt)$$

where

C_v = specific heat per unit weight (or mass) of system

W = weight (or mass) of system

T_b = volume averaged temperature of body

To this must be added the thermoregulating assumption

T_r = constant (say 37 C)

T_r = average value (in time) of "deep body" temperature (the regulated temperature)

This equation set is phenomenologically sound. It carries with it the implication that in a system which is regulated, in some sense (while not yet explicit, it is now possible for

T_r to make its appearance as an as yet undetermined portion of T_b , a self-powered system accepts a burden of heat storage in order to regulate its controllable avenues of heat exchange around a set point. If a more restrictive assumption is to be made, it may be, further, that there is no other physically known means for regulating a self actuated system. Thus intrinsically the regulation of a system must be dynamic.

In order to assess the order of magnitude of experimental accuracy that may be required for assessing the storage term, the following numerical values are assumed (for a cold environment):

$$\begin{array}{ll} M_0 = 50 \text{ k cal/m}^2 \text{ hr} & T_S = 31 \text{ C} \\ L_0 = 10 \text{ k cal/m}^2 \text{ hr} & W = 30 \text{ kg/m}^2 \\ K_0 = 8 \text{ k cal/m}^2 \text{ hr} & C_v = 0.8 \text{ k cal/kg} \\ & \text{deg 0} \end{array}$$

$$40 - 8 (31 - T_0) = 0.8 \times 30 (dT_b/dt)$$

These numerical values have been chosen from data of Winslow, Herrington and Gagge (1937) to give a "storage" of about $-40 \text{ k cal/m}^2 \text{ hr}$ at 21 C . This loss represents a drop in body temperature of about 1.7 C/hr . To achieve an approximate desired accuracy of about 4 k cal/m^2 requires a measuring accuracy of the order of $0.003 \text{ deg C per min}$. Thus short-term power measurements on the human body require rate sensitivities of the order of $0.001 \text{ deg C per min}$ in temperature measurement or control, which, perhaps are not so remarkable with regard to the precision of the temperature measurement as they are with regard to the time stability of the temperature measurement.

In order to obtain some information about the long-time scale for equilibrium results, it is necessary to make some additional assumptions. The crudest (single lumped time constant) model of the system may be taken to be

$$M_0 - L_0 - K_0 (T_S - T_0) = C_v W (dT_b/dt)$$

$$M_0 = C_0 (T_r - T_S)$$

The second equation assumes that the system can be treated as a central core in which temperature is well mixed and which has a constant layer, for its outer core, of conductance C_0 . This model permits the assumption that:

$$T_b = a T_r + b T_S$$

$$a + b = 1$$

Validation for such a model may be taken from (2) (p. 128) in which Burton gives

$$T_b = 0.65 T_r + 0.35 T_S$$

Hardy and DuBois give

$$T_b = 0.8 T_r + 0.2 T_S$$

These models are equivalent to assuming fixed spatial zones of temperature, in each of which, the temperature distribution consists of a separable spatial and temporal function. In fact (2) (pp. 132-133) justifies a linear temperature distribution in the peripheral zone and a flat distribution in the core, in the case at least of the forearm and thigh. A linear model perhaps most validly permits

$$T_b = 0.75 T_r + 0.25 T_S$$

Since this result is close enough to the empirically justified measurements of Burton, and Hardy and DuBois, a two-zone model may be regarded as having some merit.

Eliminating T_r and T_S , respectively, from the three-equation set results in

$$(C_v W/K_0) T_S + T_S = [(M_0 - L_0)/K_0] + T_0$$

$$(C_v W/K_0) T_r + T_r = (M_0/C_0) + [(M_0 - L_0)/K_0] + T_0$$

These single time-constant results permit simple interpretation. Let the single time constant λ denote

$$\lambda = C_v W/K_0$$

These equations then state that the skin temperature is driven by the temperature drop created by the metabolism across the external resistance, plus ambient temperature, with a lag that depends on a single time constant. The deep body temperature is additionally driven by the drop of temperature across the body resistance.

Of interest here is the time constant

$$\lambda = (0.8 \times 30)/8 = 3 \text{ hr}$$

Thus the primary lumped-element time constant of the human is of the order of 3 hr. No transient can be expected to die out fully in less than 3 hr.

These equations are still not satisfactory

for a description of the system. If the system were a passive single lumped-time-constant system like a liquid thermometer bulb (in which, to simulate correspondence, one may imagine a constant power heater immersed in the mercury), the previous assumptions would be reasonably well based, and do in fact furnish the basis for assessing performance of glass thermometers. The glass is the resistance. It is assumed that the central core of mercury is well stirred by convection. The mercury and glass make up the heat capacity. The basic weakness in description, however, is that it is not for a regulating system. A better example might be taken of a thermostatted house, say, considered as a single lumped-time-constant system to which has been added a simple on-off thermostatic "controller." One would find an exponential rise and fall in temperature; in response to a step-function change of external temperature the on-off cycles would have to be observed for a time greater than, but of the order of, the longest lumped single time constant of the system as a whole before a steady state on-off cycle were achieved. Roughly, these results may be regarded as a dynamic description of the simplest regulating systems.

The literature was therefore searched for dynamic data taken on the human. It is suggested, by implication, that, in the vasomotor and evaporative zone, a passive equilibrium is achieved in which the deep body temperature remains constant. While time-dependent data (sparse in quantity) perhaps indicate differently, there is no other useful interpretation given of the presentation adopted by Winslow, Herrington, Gagge and Hardy and DuBois in representing the partition of the avenues of heat versus operative temperature.

The lack of pointed emphasis on the time domain of measurement and response, while stressing the reproducibility of results, permits only the logical inference of static or quasistatic results. Thus it was possible only by detailed examination of a limited amount of data to derive some a priori ideas on the true dynamic state of affairs in human thermoregulation.

For example, in the zone of body cooling, it is stated that there is a loss in storage, and it is suggested that ultimately a rise in metabolism makes up for the loss. In particular, data of DuBois' (4) (p. 37) suggests that the regulation is that of a simple on-off controller in the zone of body cooling. More particularly, the figure suggests that the exponential rise and fall of an on-off controller operating around a thermostat set point occurs with a time constant of the order of an hour.

The unsatisfactory state of a description of the dynamic characteristics of human thermoregulation therefore made it necessary to perform some primary and basic experiments on the human. The guiding assumption was made that what might be expected generally in any simple thermoregulated system is that the storage indicator - here the rate of change of mean body temperature - will give some information about the dynamics of the system. The level of man temperature (definitely, the level of deep body temperature) becomes uninteresting. Control is implied in the rise and fall of mean temperature around the set point. However, in order to have consistency with the first law of thermodynamics, it is necessary that equilibrium in some period of the order of 3 hr be found.

The average body temperature was the first parameter chosen for experimental investigation. A number of thermocouples, connected in series to sum up temperature readings, were inserted in a subject resting quiescently, through all easily available re-entrant openings into a human. These included the urethra, the esophagus, and the skin itself. Number 40 copper - constantan thermocouple wire was used.

The basic conclusion from these experiments (performed first in the regime of body cooling and then in the vasomotor and evaporative regime) was that the "noise" far outweighed the expected response to step functions. After exhausting most sources of possible criticism of experimental technique, it was finally concluded that this was not noise and that a real frequency spectrum of thermal oscillations existed. Specifically, there appeared to be a number of temperature cycles with periods equal to or longer than a 90-sec period. These faster cycles by far masked any long time (say 3 hr) cycle. As another tentative working hypothesis, it appeared more nearly true to regard skin temperature as being regulated than not, although in some, as yet, undisclosed sense. A similar tentative conclusion might be drawn from the figure on page 564 in (4).

Thus basically in these experiments, which were considerable in number, and which beyond the major observations made, are not worth more extensive description, it was finally concluded that a static model of the thermoregulation of a human was hopeless, that a simple single time constant on-off controller model was hopeless; that in all temperature regimes there is a cyclicity which must be taken into account; that the dynamic equations must be nonlinear to account for the hard operating conditions (see reference 5 for background); that the periodicity

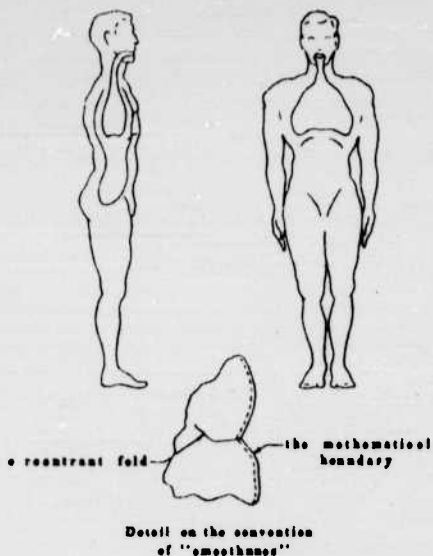


Fig.1 Prototype of a stationary model for the mathematical boundary of a human (reentrant into the lung cavity).

ties approximately noted could not be explained by purely passive thermal elements with a constant heat of combustion source, and by simple thermal lags; that there is a considerable number of degrees of freedom in the system; and that the sensing and control functions that take place in the body have not been adequately described in a phenomenological sense.

These conclusions are critical of previous work in this field. The intent is not to suggest that previous work has had no merit, but to point up that the jig-saw puzzle of how thermoregulation is achieved requires rearrangement. A correct end solution must in good measure validate all previous work, but the new assembly of ideas must stand out to the reader, in particular to the physiological reader, in such sharp fashion that he can reexamine the physiological evidence and create suitable justifications of the re-assembled story, or criticize its indefensible elements. In this context, he must be sympathetic toward the physical arguments by which the story is re-assembled.

Therefore, it is necessary to review the theoretical background in creating a suitable phenomenological model. The most precise energy equation that one may assume for the human body must be derived from a field equation. (This is the same procedure that, in principle, must be used and is used to "justify" Ohm's law.) By

starting with a hydrodynamic field energy equation in which chemical reactivity is permitted, and by adding suitable assumptions that seem to be obeyed in the body, a simplified equation is finally obtained in field form that may be integrated over the entire body. However, certain comments must be made. The body processes are intermittent (they certainly are keyed at breathing frequency). The problem is to replace them by an equivalent continuous process. The following model provisionally may be assumed as the most suitable mathematical boundary to take for the body that anticipates this problem. It is illustrated in Fig. 1. The boundary will be taken as a "smooth" boundary fixed in space, corresponding to the position of the skin averaged or filtered in some small interval of time, that has no resultant folding, and which is re-entrant into the lung cavity. This integration boundary now may be assumed to bound only liquid or solid. If the convention is taken that any other gas pockets are lumped into the lung cavity, then as a goodly approximation, one may assume that the enclosed body substance to be incompressible.

Integration over the entire fixed volume envelope then results in

$$W C_v (dT_b/dt) = \left[k(dT/dn) \right]_1 A_1 + H$$

with respect to the inside boundary of the envelope.

W = weight (or mass) of body

C_v = specific heat of average body tissue

T_b = averaged (over liquid and solid) volume weighted temperature throughout body envelope

H = total heat of combustion (averaged over the filtering time interval produced in the body)

k = body conductivity at the inner surface of envelope

$(dT)/(dn)$ = normal surface averaged temperature gradient over inside surface of envelope

A_1 = area of fixed "smooth" volume envelope

These terms now must be assessed at the exterior boundary.

The conductance term is assessed by continuity of flux at the boundary. It may be broken into two parts, one over the outer skin, and the other re-entrant into the lung cavity.

Consider first the loss from the lung cavity. The heat exchange there is intermittent. However, by the concept of the instantaneous filtered average, it can be replaced by an equivalent continuous heat exchange. The filtered av-

erage of an equivalent continuous-flow process is represented by the heating of the total wet-gas equivalent of inspired air (the minute volume) assessed as all air, from ambient temperature T_0 to the mean deep body (constant because thermostatted) temperature T_r plus the latent heat of evaporation required to saturate the expired gas.

It may be shown, ultimately, that the approximate heat loss from the lungs is given by

$$0.095 (1 - 0.011 (37 - T_0)) M$$

M = metabolism (as measured at a measuring station) assuming zero humidity ambient air.

The loss through the outer skin, by the ideas previously discussed may be written as

$$h (T_s - T_0) A_0$$

where

h = over-all effective heat-transfer coefficient

A_0 = external smooth boundary area

The heat of combustion H is the integrated total instantaneous filtered average. However, for the process of measuring, the measuring or the control station - if the body controls the metabolism - most probably is located at discrete points. Thus there are process lags. Most probably these transport lags are distributed lags. However, even the simplest model (a single-time-constant model) would require that the measured metabolism M (say as measured by gas exchange in the lungs) and the heat of combustion H are related by

$$M = H + \lambda_m \dot{H}$$

λ_m = a single time constant
or more generally

$$M = (O + 1) H$$

O = a suitable differential operator: Its necessary property is that the mean value of $O(H)$, in time, is zero, so that M and H average to the same value. Thus the simplest possible equation might be illustrated, schematically, by

$$W C_v (dT_p/dt) = (O + 1) M - fM - K (T_s - T_0) + O_1 (T_s) + O_2 (T_0)$$

where

f = fraction of metabolic heat that does

not pass through the clothes

$O_{1, 2}$ = suitable differential operators on T_s and T_0 , respectively. These terms average to zero in time

If it is assumed that these equations are linear, except for the clothing term, then one might expect, either exactly or approximately, that the time average of this equation would result in

$$K (T_s - T_0) = M (1 - f)$$

The bar notation denotes time average. These equations replace the looser definitions given previously.

As a matter of fact, this is as far as one may go a priori. These equations are now thermodynamically correct. They say that in a cyclic system which hunts around a regulated variable (not now necessarily a conservative system), there exists a modest period of time (if not too long so that the loss in weight is not excessive) over which equilibrium energy exchanges should take place. In the dynamic equation, the possibility exists of a variety of coupled sources of oscillation.

Thus, while the time averaged over-all thermal equation holds promise for a definition of the effect of a load, the problem still remains in what true sense may this equation be used. It may not be used during one breathing cycle (say 4 sec). The previous estimate indicated that it might have to be used over a period as long as 3 hr. Thus, in order to be able to proceed with any confidence, in the best sense in which to use the time-averaged equation, one must have at least a phenomenological basis for the true dynamic equations. Translated into "practical" terms (at least for a mathematical physicist) this means that one must be able to discuss in physical or mathematical terms the degrees of freedom of the system, the types of mechanisms, and their characteristics that might go to make up the governing nonlinear differential equation. It is only by a description of the dynamic thermoregulation of the human that the effect of clothes as a load on the system can be understood.

The sparse dynamic data in the literature were reviewed again. It appeared quite clear that, in some undisclosed sense, system variables seemed to include metabolism, the effective wetted-surface area, the body conductance, the skin temperature. Since the experimental data that had been taken indicated considerable "high-frequency" harmonic content (at periodicities in the minutes level) that, by far, masked the low

single-time-constant frequencies (cycles in the hours range) and that could not be ascribed to "noise," the basic problem seemed to lie in accounting for high-frequency phenomena. It is believed that a major conceptual breakthrough occurred from considering the problem in this light.

It is already implicit that the real time constants (whether zonal or not) are too slow (the fluid-exchanger system is of no help against the large capacitance of the system and boundary resistances). On the other hand, the electrical (nervous) signalling is much too fast, as is the fluid-circulation system. There is an intermediate range of modest speed that must get at time constants associated with fluid transport, and slower diffusive processes. The problem was where to find a mountain from which to view the terrain. Very plausibly one may start from the zonal concept. The implication in

$$T_b = a T_r + b T_s$$

and in particular that

$$T_b = 0.75 T_r + 0.25 T_s$$

is that a considerable degree of linearity in the variation of skin temperature through the outer periphery and flatness in the inner periphery exists. More particularly the approximate mean equivalent depth over which linearity would be required in the skin zone is 1 in. The first immediate thought, also suggested in the literature, is that this would correspond to the "peripheral vasoconstrictive" zone of the body; i.e., a zone that encompassed through the fat layer to the fascia. Direct clinical evidence appeared to exist in (2) (pp. 132 and 133), of temperature probes into the forearm and thigh, and some other incidental data in depth surveys through the anus, vulva, possibly the urethra and esophagus. Mouth data indicate that "in a short distance" one is "substantially" up to deep body temperature. The first reference, however, is the most important in establishing in some quantitative sense, a real gross linearity in temperature through the outer zone. One significant detail was originally overlooked which will be mentioned soon. To a crude extent the limits of linearity extended to the order of magnitude of the fascia. Consistency is thus achieved with regard to a vasoconstrictive-control concept that brings the vasomotor zone into compatibility with a two-zone concept of temperature distribution. However, here arises a

major physical paradox. A linear (this includes a flat) temperature zone implies negligible distributed heat production (at least averaged over time). The presence of a distributed fluid-exchange system could only go to produce a stationary average instantaneous distribution (i.e., temperatures may only rise and fall in unison) in the radial dimension. In any case then the heat production must be concentrated in the transition zone between the linear temperature distribution of the outer zone, and the flat temperature distribution of the inner zone. Thus there must intrinsically exist three zones, with a relatively concentrated middle zone. Structurally, the only mechanism that could exist at that depth was the muscle system! Thus, all of a sudden another piece of the jig-saw puzzle fell into place. Re-examination of the previous data now began to make sense. The end of the linear temperature zone is not "in the vicinity of the fascia," but is "in the vicinity of the middle of the muscle system." As a physicist reporting, any responsibility is disclaimed for the validity of the following concept. However, a biological colleague pointed out that basically one could regard the human system from its embryological development, as made up of three systems, in which the "muscular system" is part of the mesodermic layer. Thus from a physical point of view, it began to make considerable sense that the major source of heat production and control is lodged in the "muscular" system, and that this should not be interpreted as an equivalent uniform sheath geometrically located (except for equivalent model purposes) but as a system roughly organized as a sheath with reentrant pocket attachments to such mesodermic organs as the brain, liver, heart, and so on (which have always been noted as having slightly higher temperatures than deep body temperatures).

It is obvious that the reason for such organization for other biological purposes is not in the domain of physics so that it is simply accepted as an empirical fact. However, a much more palatable picture is now obtained, from a control point of view, of how an inner zone is regulated with a flat temperature distribution inside. It is the same system that would be used in a laboratory. An inner zone consists of a well-stirred liquid bath, with possibly a minor amount of immersion heater distributed for fine control. However, major control lies in an electric heater wound around the inner zone. By virtue of its forcing a conductance of heat inward (it is at higher temperature) it creates a concave upward distribution of temperature in to the center. Modest central heating creates

a concave downward distribution. Thus a relatively flat central distribution is achieved, by virtue of a considerable degree of harmonic compensation (as in a Helmholtz coil magnetic field, there is considerable ripple but it averages about a constant value). Further plausible details in a laboratory thermal container are a well-stirred liquid bath, a high degree of insulation outside, and at least a modest degree of temperature regulation of the outer layer.

Ascribing now the "major source of heat production" into the "muscular" system forces another consequence. The heater must be controlled in a lively fashion. In an electrical heater system with an external power supply, this would be easily achieved by a rheostat control permitting easy and rapid swings in power level. Unfortunately in the body system, what is needed is that the power supply or prime mover (i.e., the steam plant, turbine, generator) must be part of the system. Equilibrium reaction chemistry where the temperature is quite regulated, so that wide rate changes cannot occur from temperature changes, preclude equilibrium reaction-rate shifting, although transport of catalyzers triggered by nerve impulses have more plausibility. However, the basic problem is that if chemical plant equilibrium (in the cells) exists in a limited sense, a ready supply of fuel must be made available and converted at a temperature equilibrium rate into heat. The basic physical way of translating this is that the only thing that can respond fast enough is a heat engine!

In analog to a system which is beginning to come into prominence in aircraft, what must happen is that an engine must be running at an idling rate (producing heat just at its standby frictional level -- i.e., the cells are being "fed" energy); and in response to a demand for heat, a work load must be thrown onto the engine. Thus the engine is always available for doing work. However, this work is not yet available as heat. The work, however, is called for by a device -- say a Prony or an electric brake -- which immediately degrades this work into heat.

In the body, presumably the muscles become a chemical heat engine in which the entire chemical panoply of reactions (phosphate reduction, and regeneration, and so on, is taking place through the "mechanical" moderation of internal muscular activity. Thus the best point of view one might postulate is that the muscles are always "shivering" subcutaneously, degrading internal mechanical work into heat in proportion to signalling demand. This then provides a lively enough engine (i.e., in the minutes or even seconds scale; however, information for electri-

cal signaling may arrive only at a diffusive time scale so that a time scale in the minutes range only may be achieved). Thus, then, metabolism is a lively changing quantity rather than a static constant quantity, that may be running standby in part of an external temperature-load range but is ready to go into action to produce ready heat if it gets too cold or if external work is required. The muscles are thus always in an active state of operation.

Thus an equivalent stationary temperature distribution has now been validated as describable by

$$T_b = a T_r + b T_s$$

but with a new interpretation. The distribution derives from three zones, not two, and these are only averages in time. It is not necessarily true for the instantaneous filtered average; i.e., time lags of modest degree may exist. This could mean specifically that terms involving mean body temperature or its integral may be replaced mathematically by a linear combination of skin and rectal temperature, but not under the mathematical operation of differentiation.

These remarks are as far as one may go toward establishing a satisfactory a priori model for measuring clothes conductance.

SUMMARY

In order to know how to measure the thermal "resistance" or "conductance" of clothes as they affect the human, it is necessary to know something about the physiological physics of the human as a potential heat source. On one hand, the large heat capacity of the system and rough estimates of the thermal resistances suggest that a period of time of the order of 3 hr may be required for thermal equilibrium. On the other hand, experimental measurements have indicated a considerable number of important sustained thermal and thermal power oscillations with periods as short as 90 sec that mask long-time adjustments. These point up what may be expected in a self-actuated regulated (here thermoregulated) system that the system must be nonlinear, and that equilibrium data from such a system can only be obtained by operating the system in some rationally understood mode.

This preliminary study has suggested that the only hope at present for determining the conductance of a set of clothes is according to the definition

$$K = (\bar{M} - \bar{L}) / (\bar{T}_s - T_o)$$

where

- T_0 = constant ambient temperature
 T_s = average external skin temperature, averaged over a long period of time (at least 3 hr)
 L = thermal power losses that do not pass through the clothes
 M = metabolic power generated by system, averaged over a long period of time
 K = total average effective conductance of entire sheath (clothes plus air) surrounding the human

Since the major loss that does not pass through the clothes is the loss from the lungs, which is essentially a constant fraction f of the metabolism, the foregoing expression may be written

$$K = \frac{M(1 - f)}{T_s - T_0}$$

It has not been demonstrated or proven how the system should be operated or monitored to give the conductance a rational meaning since the dynamic characteristics of the various control elements of the body have not been described. However, it has been possible to begin to create a rational model for the system that would begin to account for the high-frequency power oscillations found experimentally and to account for the success of a two-zone temperature model that has been used to describe the human system (a linear temperature distribution in the outer body sheath and a flat distribution in the core). It has been postulated that there are three zones in the body, the outer vasoconstrictive zone,

the inner core, and a middle (mesodermic) "muscular" system in which the major metabolic heat is produced. This muscular system is always "shivering" and producing thermal power oscillations as a heat engine. This very active middle layer may therefore restrain core temperature in a time domain much shorter than 3 hr. Since these power oscillations must be limited by some as yet undisclosed control function, the possibility of determining clothes conductance is enhanced if a rational external work schedule can be arrived at for the system. At this point none is suggested.

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HUMAN BODY AS AN INCONSTANT HEAT SOURCE AND ITS RELATION TO CLOTHES INSULATION

2 - Experimental Investigation into
Dynamics of the Source

by

A. S. Iberall*

ABSTRACT

Quantitative measurement on the human in the so-called evaporative, vasomotor, and metabolic-control regimes has revealed frequency spectrum of sustained power oscillations with approximate periods of 2, 7, 35 min, and $3\frac{1}{2}$ hr independent of the regime. Step-function adjustments take place with a time constant of about 7 min. It is believed that the $3\frac{1}{2}$ hr cycle represents the shortest equilibrium cycle. The hypothesis that it might be possible to measure the resistance of clothing as an ohmic relation among time-averaged equilibrium values, and for a specific mode of operation of the system has now been put in rational context in the time domain. Two equilibrium modes of the human system were explored. The active mode of operation of the system, to which the resistance concept of clothes is most applicable, is as a feedback system in which the extremities are used as error indicators of deviations from a comfort-level set point. In response to deviations, the human feeds back a signal to generate an activity level in which only internal work - immediately degraded into heat - is done to maintain the comfort level. This is referred to as the comfort mode of operation of the system. Another "survival" mode of operation of the system is also described.

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Human Body as an Inconstant Heat Source and Its Relation to Determination of Clothes Insulation

A. S. IBERALL

2 - Experimental Investigation into Dynamics of the Source

In an earlier paper (1)², some tentative characteristics were derived for a dynamic model of the human as a heat source. It was suggested that, if an equilibrium thermal cycle of the order of 3 hr could be found in the human, it would be possible to determine the conductance of clothes as an ohmic relation among time averaged equilibrium values of flux and thermal potential. However, the nonlinear operating characteristics of a self-actual-regulated (here thermoregulated) system require specification of the mode of operation of the system. In the usual quiescent mode of operation of the system described in the literature, it is not clear how the independent variables of metabolism, evaporative heat loss, skin temperature, and body conductance vary dynamically to establish a long-time equilibrium. Particularly, it is not clear how these variables adjust as a load on the system (here clothes) is changed. The purpose of this study was to determine experimentally how the human system may be operated to obtain rational and comprehensible values for the conductance of clothes.

EXPERIMENTAL EXPOSE OF DYNAMIC THERMOREGULATING CHARACTERISTICS OF THE HUMAN

The previous paper (1) touched on some preliminary qualitative experiments made on the harmonic content of average body temperature. At this point, it was timely to attempt to measure experimentally, and quantitatively, what the harmonic content of the system might be. It is already implicit in treating any complex system that such harmonic content would make itself evident in the response of any variable that could clearly show the dynamic characteristics of the system. In particular, it was

²Numbers in parentheses designate References at end of paper.

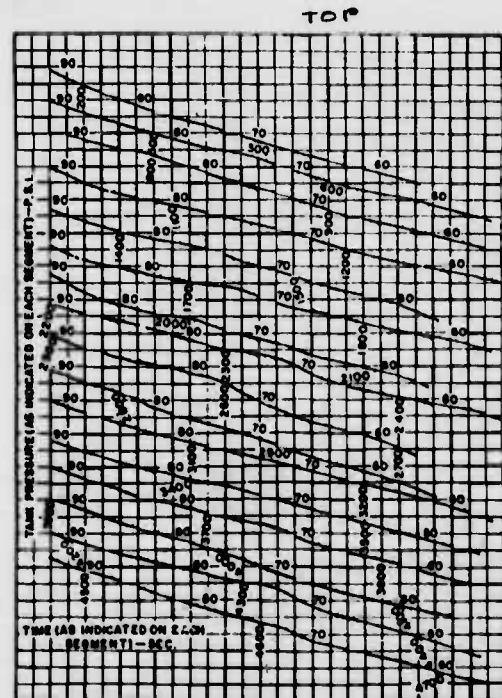


Fig.1(a) Air consumption of human subject at 26 C (by decay of supply pressure in a fixed volume).

desirable that changes should be shown in the steady-state level with step-function changes of load, due either to changes of external temperature, or changes of external clothes. Because of the considerable doubt cast on clothes measurements, it was desirable that the simplest step changes possible be explored. Thus, ambient temperature as a step variable was chosen, under a condition of wearing a constant set of light clothing. Since doubt has been cast already on whether skin temperature was or was not a controlled variable, and since both

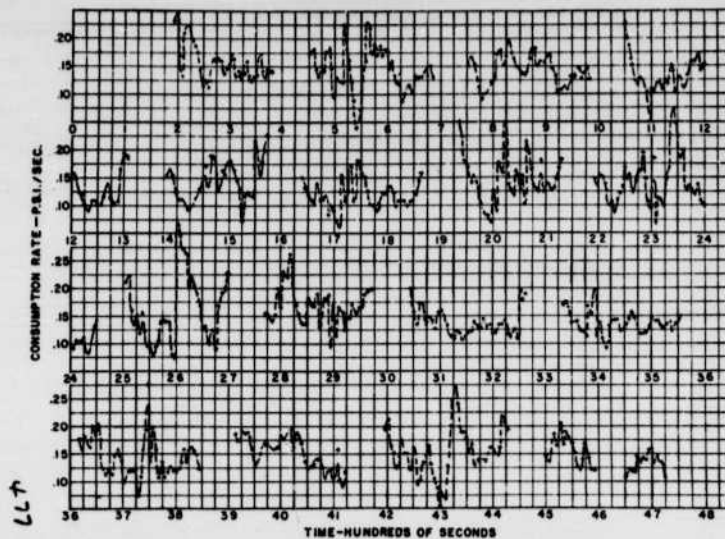


Fig.1(b) Air consumption derived from Fig.1(a) (running five breath averages).

skin temperature and rectal temperature required considerable sensitivity of measurement, i.e., sensitivities of the order of 0.001 deg C per min, which could raise doubts in the reader's mind, as to the measurement accuracy, the measurement of skin temperature was excluded. (However, it is note-worthy to call attention to the reader the experimental observation made in dynamic measurements of skin temperature that as one regulated a sensitive potentiometer to null balance on a subject, it was easily evident that a cycle of perhaps 90 sec was going on in skin temperature. The observer would soon almost begin to feel the "pulse" of the cycle.) Thus "instant metabolism" was chosen for the variable to be explored. Subject, of course, to criticism, actually instantaneous air consumption was chosen for the variable to be explored. This is heuristically referred to as metabolism on the theoretical assumption, checked by experimental validation with an oxygen partial pressure meter (Pauling meter) measuring in the exhaled breath, that the variation in partial pressure at say the end component of each breathing cycle reflected changes that were only a small modest percentage of the changes indicated in air consumption (the metabolism may be computed loosely as the product of air consumption and the fractional change in partial pressure of oxygen from inspired tank air to expired air). The basic system chosen was thus an open system measuring demand on the high-pressure supply side. This

was chosen because of the cleanness of time lags, and the precision with which rapid changes could be easily detected.

The basic supply system thus consisted of a high-pressure tank of known volume, a precision pressure gage teed into the tank (accuracy 0.1 per cent), a pressure regulator, a shutoff valve, a demand regulator, a known volume and precision pressure gage teed in between the demand regulator and shutoff valve, or a high-pressure, differential-pressure flowmeter teed into the same point, and a small demand mask.

The first basic experiments were designed to determine the time domain in which significant dynamic phenomena occurred. Thus, in a first experiment, a small volume was chosen for the volume intermediate between the shutoff valve and demand regulator. This volume was repetitively charged to about 100 psig from the supply tank each time the pressure fell to 50 psig (the minimal desirable supply pressure for the demand regulator). Each breath would lead to a significant decay of pressure; i.e., about 50 breaths would be taken per 50 psi charge. By plotting supply-volume pressure at say the end of exhalation against time, segmental portions of pressure decay, each approximately 200 sec long were obtained. From slopes, the "instantaneous", i.e., averaged over one, really one or two breaths, air consumption could be obtained. From a few hours of such data, it was ascertained that nonrandom cycles in the 1 1/2 to 2-min range, in the 5 to 8-min range, in the 25 to 50-

min range, and possibly in the 3 to 5-hr range, existed. Randomness of cycle commensurate with the experimental accuracy, in the two or three breathing cycles range of time, seemed to exist. In particular, it appeared that the first reasonably "solid" response could be obtained by averaging over approximately five breaths; i.e., over an approximate period of time ranging from 20 to 30 sec. Thus, this represents as a first approximation the minimum period of time or the number of breathing cycles over which one must integrate for "instantaneous filtered averages." The approximate dilemma that this sampling period presents is that one is caught between choosing not too short a period of time that the "random high-frequency noise" ("random" erraticness, if any, of individual breathing cycles) will disturb results, and the necessity for choosing not too long a period of time that the first significant harmonic (which appeared always to lie in the 90 to 130-sec range) could be with reasonable accuracy. Thus, all subsequent work oscillated in a choice of sampling period referenced either to a 20 or the 30-sec sampling period.

Segmental sections of pressure decay for about 5000 sec are shown in Fig. 1. Also shown are the results of differentiation obtained by taking running five-breath averages. Cyclicity is quite evident.

In order to insure that the results achieved by graphical differentiation were real, i.e., to free these results from any question, an instantaneous averaging flowmeter was constructed and installed just upstream of the demand regulator. The shutoff valve was eliminated and replaced by a rather large volume to help filter out any pressure-reducer pressure ripple. (The reducer was chosen and set to maintain an extremely constant supply pressure). The instantaneous flowmeter consisted of a carefully designed linear viscous flowmeter of parallel capillary tubing whose response would be very purely proportional to volume flow at the constant supply pressure. The differential-pressure gage used to indicate flows (at a high absolute pressure, which was varied from 50 to 150 psig to change flow sensitivity) was carefully fitted with a balanced combination of linear pneumatic resistances and capacitances to produce differential pressure indicating lags at levels in the 10 to 40 second range. Thus, the differential pressure was averaged (or filtered) over corresponding periods of time. By plotting the flows thus indicated, the reality of the dynamic cycles was validated, at least for the faster cycles. The absolute accuracy of this instru-

ment, in particular the differential-pressure gage, did not warrant considering it for primary use, since too wide a differential scale range is required (it must practically extend to cover peak breathing flows, say perhaps in the 60 - 90 lpm), while at the same time requiring high percentage accuracy at low mean level flows, (which might be at the 3 - 4 lpm flow level). To cover the 20 to 1 flow range (in the dynamic measurements made, at least 8 to 1 variations in instantaneous filtered flows were found), required rather frequent changes in sensitivity, achievable by changing the absolute pressure, so that these measurements were interpreted only as rough quantitative indicators of the reality of cyclic changes. From these experiments, it was only possible to confirm that either the 20 or 30-sec averaging period had comparable probability in indicating the "100" second cycles and, since the 20-sec period still showed modest breathing pulsation, the 30-sec period seemed to have a little more merit; i.e., 7 breaths are perhaps a shade better than 5-breath averages.

From these results, the form of the first primary quantitative experiment was cast. A decay volume was rechosen (approximately 2 liters) for coupling into the line between the shutoff valve and the demand regulator. A precision 300-psi test gage was chosen for indication. This instrument permitted readings with a sensitivity of about 1/2 psi, and an accuracy of about 1 psi. If an approximate consumption level of 5 lpm atp, and 15 breaths per min is assumed, then a decay cycle (from 200 psi to 50 psi) of about 4 min is obtained; permitting approximately 8 1/2-min decays, of about 20-psi decays each. Thus, the accuracy of an individual reading is of the order of 5 per cent. Since each refilling of the cylinder from 200 psi to 50 psi involves an adiabatic heating of the gas, the time for re-establishment of "good enough" equilibrium was investigated and found to be about 30 sec. It was thus decided that a serial recording would be made at 30-sec intervals in which a recording interval would be skipped each time the cylinder decayed to the vicinity of 50 psi and, instead, the cylinder would be refilled and the time permitted for equilibrium.

Primary Experiment

The primary experiment performed was a step function of a young "healthy" athletic male (18 years old) wearing light clothes from a "normal" temperature environment of about 75 F and activity level to a quiescent level, while lying horizontally in a small room held constant at a series of preselected temperatures. The bed

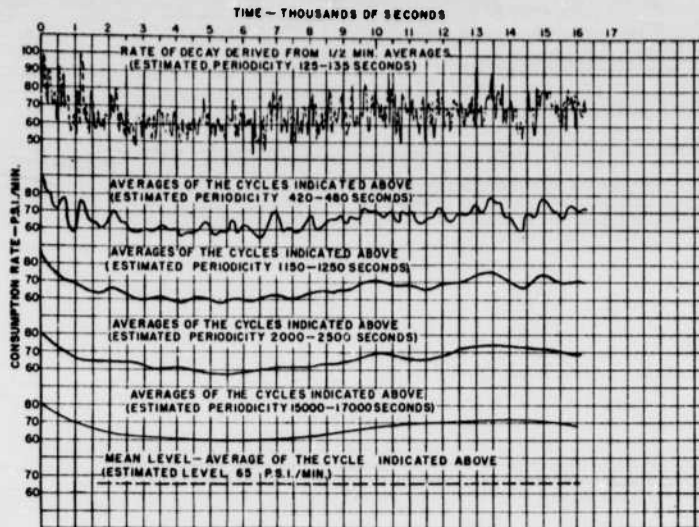


Fig.2(a) Air consumption of human subject at 17 C ambient temperature (expressed as the number of psi/min of air used from 2 liter volume).

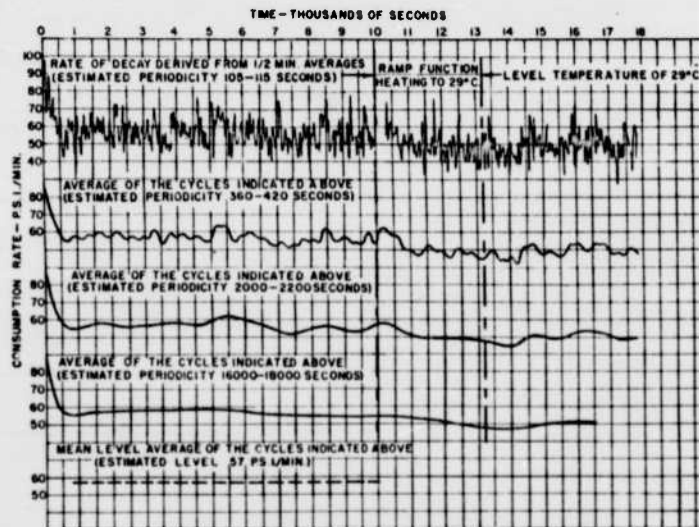


Fig.2(b) Air consumption at 21.5 C.

chosen was an open network of a minimal ... of 1/4-in-diameter hemp rope and 1/8-in-diam clothes line in a wooden box frame mounted about 2 ft from the floor. The bed was designed to "give" very little so that the chance for much vertical motion was quite small, and to possess very little heat capacity or too good thermal conductivity. In other words, the subject was mounted as close as possible on a

nonyielding massless plane, with instructions to move about minimally. Temperature control in the room was achieved by use of a judicious choice of room air conditioners, heating elements and fans to give a broad circulation pattern in the room with only modest breeze over and under the subject. The subject was shielded from radiant surfaces at higher temperature, and as many other minor precautions were taken as

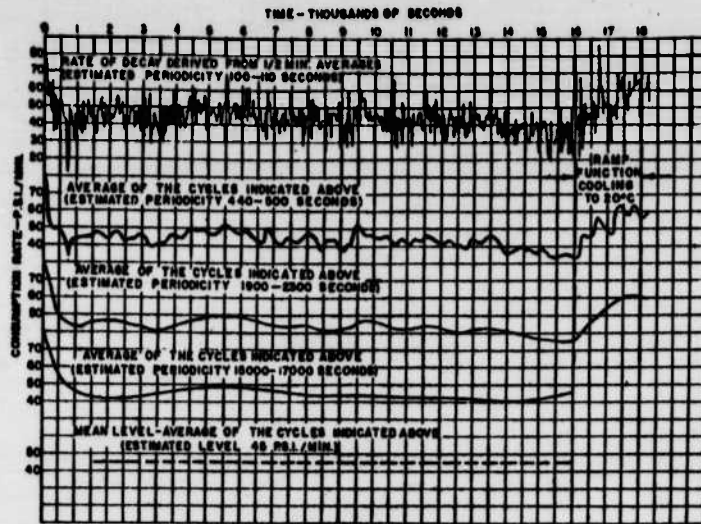


Fig.2(c) Air consumption at 26 C.

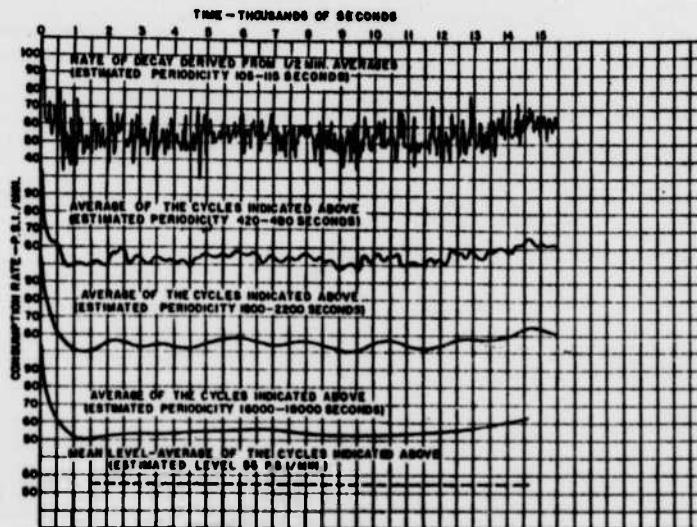


Fig.2(d) Air consumption at 35 C.

possible, to assure that to a large extent the subject was immersed in a "black-body enclosure" of a temperature corresponding to air temperature. Temperature control was manually adjusted to as close to 0.1 deg C as possible. Thus, the basic interpretation that these experiments permit is relative data on the dynamic course of air consumption of a "constant" quiescent subject wearing "constant" clothes in a "constant" temperature at various temperature levels. One experiment was done each day starting under

comparable conditions of time, outside temperature, and diet. Each experiment was run for somewhat more than 5 hr.

To a crudar extent the air consumption is a mirror of metabolic change. To verify this, a relatively rapid responding Pauling meter (with time delays of the order of 1/2 sec) was connected into the outlet of the demand mask in a tube furnished with a light check valve; the check-valve resistance was chosen to be sufficient to bleed a sample of exhaled gas through

the Pauling meter. The oxygen partial pressure was noted at the end of each expiration cycle and averaged over five breaths. By multiplying the instantaneous air consumption by the oxygen partial-pressure change from inhalation (tank) oxygen partial pressure and the exhalation partial pressure, a quantity very nearly equal to the oxygen derived metabolism is obtained. This was done for selected experiments to note that there is no tremendous difference in dynamic performance between air consumption and "metabolism." Thus in the light of the general philosophy, that any indicator of changes in any generalized displacement (or of a coordinate corresponding to a degree of freedom) of a complex mechanical system allows inspection of the dynamic performance of the system, the first primary data present only relative air consumption, to retain the highest possible degree of accuracy. It is proposed later to build a more complex (and expensive) rebreather system that may have comparable accuracy for recording over the length of test period required.

Results of Tests

The data from four such tests are shown in Fig. 2. These data were taken at room temperatures of 12, 21.5, 26 and 35 C respectively. In each of the curves are shown the individual 1/2-min averages, expressed as a decay rate in psi/minute (decay of air pressure in the 2 liter storage volume); and four steps of filtering, obtained in each case by graphically averaging the individual cycles obtained from the previous filtering.

The obvious most noticeable characteristic of the primary data is the rich harmonic content of the air consumption. The second most noticeable characteristic is the similarity in harmonic content in all temperature zones, whether in the "metabolic" zone, the vasomotor zone, or the evaporative zone. Other characteristics that may be noted are that the significant harmonics seem to be discrete in number and essentially the same in frequency and amplitude independent of temperature, that the initial transient response is an approximate dampened oscillation at one of the salient harmonic frequencies, that these harmonics persist indefinitely in time (i.e. that the system is nonlinear), that the time for an equilibrium cycle is quite long (the data suggests that it is not less than three hours), and that the mean level of consumption changes significantly with temperature. The data also suggest that the order of magnitude of the instantaneous filtered average "metabolism"

fluctuates by a factor of two in a "steady state" situation independent of the control region.

The basic tentative inferences that were drawn from these data are that the major thermoregulation in the body derives from a lively on-off heat engine (the muscles) which sheath the internal zone of the body, and that other auxiliary controllers control the level of internal temperatures through at least three other control mechanisms, and that the shortest time over which thermal equilibrium exists is of the order of a few hours. Thus finally for the first time some idea was available as to what time domain one must average over in order to apply equilibrium assumptions.

Integrating System Tests

As a sharper test, both of this hypothesis, and of the apparent demonstration of dynamic cyclicity in human response, an integrating system was constructed, in which a medium-sized supply tank (approximately 2/3 cu ft internal volume) was used as a supply volume, and connected directly to a demand regulator with a precision 0-3000 psi (0.1 per cent accuracy) gage teed into the tank. By this means, the air consumption over a longer period could be averaged. In particular the average over increasing time in the presence of a dynamic repetitive bounded cycle should be a damped oscillation whose asymptote decayed hyperbolically. The period of time that it takes the average to level off is a measure of the longest dynamic cycle present.

Data on a selected experiment performed this way at a temperature of 20.5 C is shown in Fig. 3. Pressure readings were taken on the tank every 15 min for 5-1/2 hr. The data in this case were obtained in a more normal environment. Another young male (about 18 years old) lightly dressed sat in a rather large air-conditioned conference room, in which a modest sweeping circulation of air existed but with no direct breeze. He sat in a rather open chair and performed the sedentary activity of reading books with a minimum of squirming. He was introduced into the environment as a step function from normal activity at normal temperature (25 C). The room, and tank, had been left cooling to equilibrium for the previous 16 hr.

The air consumption averaged over increasing time is shown in Fig. 3. Since these data include the initial transient, what is also shown is the average consumption for each 15 min, the approximate (but filtered to the 15-min level) cycle (these data thus contain all cycles longer

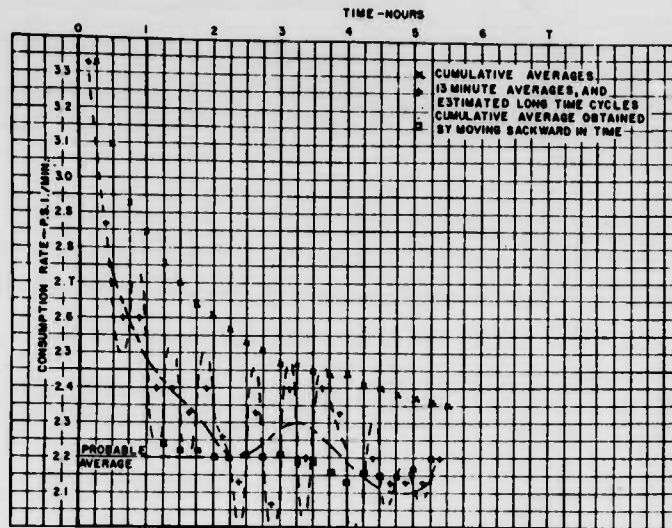


Fig.3 Cumulatively integrated average air consumption of human subject at 21 C (expressed as the number of psi/min used from an approximately 2/3 cu ft volume).

than 15 min), and also the increasing time average integrated backwards from the end of time. It is clear from these data that the integrated damped oscillation takes a period of the order of 3 hr to substantially disappear. Thus again, in an independent fashion (with even considerably more precision), the reality of dynamic cycles is demonstrated, as well as the reality that thermodynamic equilibrium is only reestablished in a test or averaging period of the order of 3 hr.

Constant - Temperature Tests

It appeared at this point, that having demonstrated a control action in the human being, a period of time over which equilibrium thermal exchange could be rationally invoked, and a rational variation with ambient temperature changes [Recall that the needs for a conductance model $K(T_s - T_o)$ involve three elements, "constancy" of load K ; definiteness in the meaning, effect and determination of T_s and T_o . The effect of these experiments had been to place T_o in a rational context], that a sufficient description was available to demonstrate the effect of clothes; i.e., that there should exist a sensitivity to change in clothes cor-relary to a change in temperature. Thus the next series of experiments was at constant temperature [Temperature in the range 21 - 22 C were used. This temperature was chosen to be

low enough to lie in the metabolic range, where it could be estimated both from the literature and from the experiment that the subject would shiver], with an incremental change in clothes. Specifically, a somewhat light jacket was added to the subject's clothing. An approximate estimate suggested that the incremental change in clothes value would be of the order of "a modest fraction" of a clo unit. Experimentally, the subject reported reasonable comfort with the jacket, but chill over a long period of time without it. These experiments were independently performed; i.e., a step fraction from normal activity and temperature to quiet reading at 21 C. The results in integrated air consumption were disappointing. Inadequate change in air consumption of metabolism was found. These experiments were repeated until it was quite clear that reproducible accuracies of better than 2 per cent were being achieved but that only small changes in metabolism were obtained with incremental clothes change. Yet the subject was conscious of changes in "comfort" level. Thus it was clear that the subjects sensors (or controllers) were either more sensitive or responded in some other fashion than had been assumed.

After some confusion and experimentation, a final hypothesis was arrived at, which appears to forge the last link in a correct phenomenological description. Again, this latter hypothesis, while related to concepts previously

discussed in the literature, puts them into a proper light.

It is often stated in the literature that the extremities are the major sources of heat exchange. This concept could not be put into useful and operational meaning, except to take it at face value that the exposed body area in extremities is large. However, there existed a more fruitful possibility in the previous analysis.

Discussion of Tests

The negative results of the incremental clothes experiments could not be gainsaid. The net equilibrium averaged metabolism of the body remained substantially constant even though clothes were changed. Thus either a change in clothes conductance or a change in skin temperature had to take place. Yet earlier experiments had indicated that there is a tendency to regulate skin temperature at fixed values. Thus, either this rough regulating hypothesis was incorrect or the clothes conductance changed. The basic clue to resolving the dilemma lay in the nonlinearity of the system. The nonlinearity created the possibility of more than one singularity (here say a point of stable equilibrium) around which the system could operate; i.e., that the controls were not single valued. For this an additional controller degree of freedom had to be found. This lay in the extremities, or more particularly in the "valves" to the extremities.

The common description of vasomotor control involves a control of radial conductance in the body through the fluid-exchange system (i.e. the conductance of the layer outside of the muscles can be changed by vasoconstriction or vasodilation. This is considered to be the controller mode for the vasomotor zone). However, there is also an "axial" control of conductance. While as a first approximation one may regard the human system as a sphere with three shell-like zones as representing only a modest deformation of its elongated form, it now becomes intrinsic that conductance control is not single moded, but is lumped into more than one element in length. This represents an independent degree of freedom.

The question on how the control function involving axial conductance was called into operation was given the following interpretation: There is either no control (or actually a positive look on this control) or there is an independent feedback control system of the engine as a whole. In a quiescent situation, either consciously or unconsciously imposed on the system,

the body is ruthless. If a heat loss persists in the system, the body shuts off the heat exchange to the extremities and lets them hang on essentially as passive capacitances in the ambient environment (i.e., they assume essentially ambient temperature). The complete experimental details of this process are not clear. Presumably ever-increasing chunks of the body are shed off into the ambient environment, until perhaps only the vital organs are regulated. (Data on this point, however, would have to be gathered.) This represents one branch of operation, which perhaps may be best described as the "survival mode of operation of the system." In another branch of operation, the system has a more active mode of control. The heat engine is excited by central control system signals which derive from sensor signals in the extremities. Here the extremities are used intrinsically only as null indicators. If an extremity temperature begins to drop, an extremity temperature indicator sets up a feedback signal in response to which exercise is performed - perhaps by that extremity. The engine "shuffles," mills about, slaps or rubs the extremities, or puts the hands in the pocket, and so on. These acts are done with no external work (the point is not that they are necessarily done with no external work, but that they may be done with no external work. Masses of the body are raised and lowered with no net mechanical work done on the environment. The same over-all average heat-balance equation may apply but the values of various terms must change. Thus a multivalued system of equilibria may exist). However, internal work is done by the muscular heat engine which is degraded into heat. It is to be expected that this mode of equilibrium operation will occur with increased metabolism. It may be referred to as the active metabolic mode or comfort mode of temperature control, for in this mode the body (namely using the extremities as null indicators) indicates its own comfort.

While this interpretation of events uses similar concepts to those commonly alluded to in the physiological literature, it may be noted that the sequence and interpretation of events is different. The extremities are not the main sources of heat regulation in the body; they are null temperature indicators. A distinction must be made between radial and axial conductances. A meaning can be assigned simultaneously to the statements that average skin temperature changes with external temperature, and that the skin temperature tends to be regulated. If one

imagines a localized skin temperature which is constant for all environmental temperatures above a definite "control" temperature, and which then drops to ambient temperature for lower temperature one has the beginnings of an idealized model. More precisely instead of a sharp break from a level temperature, if there is a slightly drooping regulating curve with a break taking place at some point on the knee of the curve a more probable description obtains. Data that point to the validity of this concept may be found in Sheard's article in (2). Either due to varying break sensitivities of different parts of the skin, or different immediate environment temperatures, in the survival mode of operation, increasing areas of the skin drop more nearly to ambient temperature. This is interpreted as a drop in mean temperature, whereas the more precise interpretation should or could be that regulated portions of the skin are still regulated, but an increased surface area of the body is not regulated.

It would thus appear that the horns of the dilemma in the description of the heat balance given in the literature have been bent into shape. The conventional description has utilized the unit area of body surface as a normalized variable. However, the "true" heat loss of an equivalent conductance term must be derived from

$$\int_{A_0} h (T_s - T_0) dA$$

Now either this term is computed as an average over the entire nonre-entrant body area A_0 as

$$\overline{h (T_s - T_0)} A_0$$

in which T_0 and h are both undescribed functions of the operating conditions (i.e., they must be independently measured); and h may be considered to depend on the mode of operation of the system (For example, in the extreme, a sock is of little insulating value to an element which has become passive, except as contributing to a time delay); or it may be computed by

$$h_0 (T_{s_0} - T_0) A$$

(assuming a sharp break to ambient temperature of skin temperature) in which h_0 is the effective conductance of clothes per unit area of active skin (which may vary with the portion of skin that is active); T_{s_0} is the average regulated skin temperature (presumably in the "vicinity" of 35 C); and A is a variable which

is determined by the physiological mode of operation.

The net practical result of this discussion is that the characterization of clothes as a conductance model depends on the mode of operation of the system; that it is stretched thin for application to the survival mode of operation in the cold (not that it may not be used, but that it must be used with extreme caution); and that it is most applicable to a comfort mode of operation in which the user, say, exercises to "maintain comfort," which finally can be given the unique operational and nonpsychological meaning that conditions of exercise are such that the extremities as null sensors are held at a "constant" level. As a final practical consequence, it suggests a new or truer point of view in the design of clothes.

Thus clothes can be considered from two points of view (particularly in the cold); as "normal" comfort level elements; or as "extreme" survival level elements. The former point of view becomes the manufacturer, designer, and evaluator of "normal" clothes for active environments. The latter becomes those interested in survival activities (such as the quiescence of sleeping, or other forms of forced inactivity in extreme temperature). Thus it becomes clear that the prescription too often imposed in both the experiments here initially reported, and in the literature in which the subject actively co-operates to remain passive are artificial representations of only one extreme of operation, and as such are not necessarily the "normal" mode of physiological thermoregulation.

This, therefore, closes as much commentary on physiological matters as a physicist may be entitled to. The remainder of the task of physiological interpretation and validation of control and sensing mechanism must be applied by the physiologist. The dynamic time scales and the relative dynamic responses to step functions must afford him his basic clues as to the possible analytic descriptions. It would appear that these intermediate time scales pose very specific problems of being too slow for hydrodynamic fluid exchange, nervous, and equilibrium chemical reaction time, but too fast for thermal exchanges. This would appear to leave diffusion of mass products as the most promising element to provide appropriate time scales. An added word of caution lies in any attempt at a description of non-linear processes. The analytic solution must be relatively precise in order to give rise to the proper stability of the indicated limit cycles (3).

Testing Validity of Concept

A preliminary experiment was performed to test the validity of this concept of operational mode of the system. First glass thermometers were crudely inserted in the instep of a shoed and socked foot and in a lightly gloved hand. The subject was told to step up and down a small step at a faster or slower rate or move the hands up and down at a faster or slower rate in response to commands. The commands were based on rapid intermittent reading of the thermometers, choosing a temperature as reference at which the extremities were found under a normal sedentary activity level in the vasomotor region, the subject was given commands to exercise the extremities to hold this temperature constant (a switching band of nominally 0.1 deg C width was originally sought for the error level, but it was difficult to maintain level better than 0.5 C in this crude experiment). At the same time the air consumption level was measured.

For the first time, the system (i.e., the human) had the proper operating "feel" of a control system. It was "lively," the feedback of information (through the observer loop) was copious; adjustments of speed or power level were sensitive, the entire concept of a system that was "dragging its feet" in control disappeared, and perhaps most significantly of all, the feeling of dependence on psychological factors (the feeling of dependence on the width of a friction band, and the precise means by which signal was applied) disappeared. A better description lies in the usual feeling when an instrument or system is tested at input levels too close to its ultimate sensitivity, i.e., microvolt excitation of an instrument with a microvolt noise level. As soon as the inputs get large enough, the "fraction" level or "noise" level of the instrument begins to assume its proper perspective as a small fractional or percentage source of error. Whereas in previous experiments the state of mind of the subject was of considerable concern (was he relaxed, should he be instructed, what should he be told to do in all kinds of anticipated emergencies), and its effect incomprehensible (the similar questions in low-level excitation experiments are the thousands of meaningless questions regard the precise possible sources of noise), now it didn't matter. The subject was just too busy in high-level activity to be concerned with minor disturbance sources. In other words the directed signalling bedlam of a feedback loop in attempting to maintain a set point was found. At last, for example, there was the "feel" of a more sensitive operational measure

of a "comfort" level than the psychological response. From beginning drops of extremity temperatures of tenths of a degree, the observer could anticipate ultimate "discomfort" or "feeling of cold" much before the subject.

Thus while for the first time the stage setting for proper testing of clothes, and evaluation of the human as a complete system was achieved, many "minor" questions now raised themselves. Experimentally, for example, internal exercising by using the entire body seemed too "vigorous" (for example, the chest began to sweat when raising and lowering the body). Questions thus formed themselves as to whether the evaporative mechanism was a follower (say of a relaxation type), or an active controller; whether the "null" balance indicators of the extremities were lumped or distributed (that they are not localized appears obvious. However, this does not contribute information as to whether the null-balance system is distributed as a single equivalent lumped element, or multimoded); what the nature of the subsidiary control loops might be (that is whether null detection is one site, leads to engine action in a second site, involving convective mass transfer, or diffusive mass transfer of heat-exchange fluid, or whether the system is partially open in that part of the heat exchange is in a closed-conduit system going to one site, and part in an open-conduit system going to another site). Thus as a fielder's choice the first simple assumption to test was that the auxiliary loop was a closed-conduit system of a single lumped element in which the auxiliary circuit elements were all localized in the controlled site; i.e., a lumped sensor at the extremity could signal to turn on a lumped engine element at the extremity to control a lumped localized valve that controlled the "flow" of heat-exchange fluid. It is obvious that this literal model is nonsense. However, it permits rough characterization of the process and some understanding of what may be expected in dynamic response. More important it extends the count of the number of degrees of freedom to the minimal number that is required for the beginnings of a primitive model of the entire system. Thus now the body must have at least three zones as counted in the radial direction, and at least one equivalent independent three-zone side branch.

Internal Work Confined to Extremities

Thus a second crude experiment was performed in which the internal work was confined as far as possible only to the extremities. A

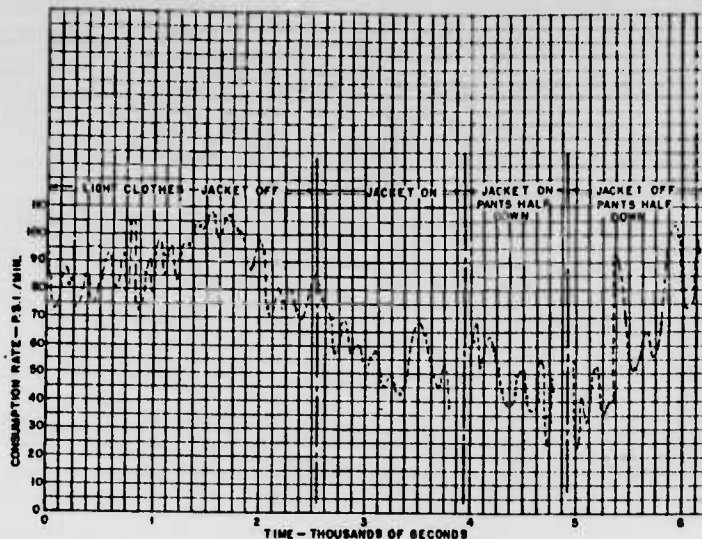


Fig. 4 Air consumption of a human subject at 15 C while engaged in just sufficient recoverable work with the hands and feet to keep those extremities at a "comfortable" temperature of 32 C.

bicycle was adapted to provide a life treadle for the hands with only slight forearm motion, by "immobilizing" the muscular structure above the elbows by creating a fixed resting point for the elbows; and to provide a treadle for the feet, in which the knee was essentially fixed in space, and, in the main, foot lift only used. Loading of a nondissipative sort was provided through "frictionless" pulleys attached to dead weights, in which change in weight was always made at a fixed height. Thus the excitation of these weights (i.e., raising and lowering them) satisfied the conditions of reversible quasi-static equilibrium in which no external work is done. Null measuring stations to provide error signals for the feedback loop were taken at the toe and finger. An additional monitoring station was taken on the chest. However, the controls were only for the hands and feet. A sensitive potentiometer and thermocouple were used to measure hand and foot temperatures. A schedule of measurement and weight adjustment of the order of 5 sec was chosen and attempts were made to maintain this schedule with only modest success. (As a rough summary it was found that the "valves" appear to have considerable dead time so that quite rapid control is needed to hang onto the set point). However, the over-all experiment appeared to work beautifully. As in the previous crude experiment, a step-function change in clothes level

was chosen for a signal change while at constant temperature. The previous experiment had already indicated the approximate transient time to allow before counting the dynamic equilibrium. In both experiments, the immediate and strikingly notable difference could be discerned that the load against which internal work must be done to maintain null balance in the extremities shifts largely, as was expected, with and without a jacket (i.e., the thermal load could be "seen" in the weight in the loading pans), and finally, in the latter experiment at last some notable changes in "metabolic" level (or air-consumption level) occurred.

Some preliminary data of this type are reported in Fig. 4. The data were taken by the same system used for the data in Fig. 2. Operating conditions were about 15 C. A lightly clothed young man was subjected to a step function in temperature to 15 C. After sufficient time had elapsed that it was reasonably certain that initial transients had decayed (approximately $2/3$ hr), he was then subjected to a step function of putting on a light jacket. The activity level to retain "comfort" dropped very quickly. However, it may be noted in Fig. 4 that the decay transient in air consumption (or metabolism) remained about the same, and about the same as in the passive mode of operation shown in Fig. 2. (A damped "oscillation" with a period in the 1000-sec range.) It is

thus quite reasonable that it is this periodic decay and the periodic sustained oscillatory cycle of this frequency that is most likely the main controller of thermoregulation in the body.

It may also be noted that air consumption (or metabolic change) is considerable. A second exploratory change of taking the pants down to half mast didn't appear to make much change. However, the subsequent removal of the jacket showed a transient that seemed to recover the earlier air consumption. Thus, even though the data at this time are limited, evidence for an operating mode of the human that tends to give equilibrium cycles and thus equilibrium states with regards to a set of clothes appears to have been achieved.

It is the intent at this time to construct an adequate automatic feedback apparatus and unquestionable "metabolic" apparatus for further exploration.

Conclusion

Thus in conclusion, it is believed that a basic model has been developed for measuring clothes. It may provisionally be considered a conductance model. It is based on the equation

$$(\bar{M} - \bar{L}) / (\bar{T}_s - T_0) = K$$

Here \bar{M} is the average metabolism obtained from an integrating period of the order of 3 hr; \bar{L} is the loss of power in stack products which do not pass through the clothes (i.e., breathing, face, other exposed parts); \bar{T}_s is to be operationally defined through the mode of operation of the system. It would appear to be a most meaningful quantity for active "comfort" operation. However, in any case it would have to be averaged over a period of time for equilibrium steady-state cycles to occur. It is the latter difficulty which probably would most quickly lend conviction to the need of a impedance law; i.e., resistance plus reactance, or conductance plus admittance. T_0 is the equivalent temperature of the external environment; i.e., it is to be derived in a manner similar to, but perhaps an extension of Gagge's concept of a mean operative temperature. K is a conductance which now is no longer necessarily constant or even single-valued by at least describable in an averaging sense. It is clear now that a priori models for K must be constructed in a dynamic fashion, that it must be constructed from dynamic treatment of a heat-transfer problem in which the dynamic laws of radiation, conduction, convection, storage capacitance, diffusion (say at least of water

vapor), and evaporation are treated. Thus, in principle, the conductance of clothes must be computed from a complete hydrodynamic field equation set.

Underlying physiological assumptions are contained not in this equation but in the dynamics of the fundamental equation from which this "result" may be considered derived. The instantaneous variations in the instantaneous dynamic equation reflects the specific physiological mechanism that are operative. However, the average in time, over equilibrium time, results in this equation.

The broad loose physiological mechanisms that have been alluded to are that in the cold, either a zone of "metabolic control" is achievable at constant comfort levels, or a zone of "axial conductance" control is achievable at constant average metabolic level; that these two cases are extremes; that all zones involve an active metabolic controller in the form of a heat engine which is "constantly oscillatory"; that the vasomotor region (if it exists as a true region) is most possibly properly described as a "radial conductance" control zone, in which the average metabolism is stationary over a finite-temperature range; but that is not yet completely understood how the higher temperature controller works. This area of uncertainty could be resolved possibly either by dynamic over-all weight measurements (the required sensitivity and frequency-response pose some difficulties), or microscopically of the basis of "valve mechanisms" by which water is made available at the skin surface dynamically. The important clues left for the physiologist is the rough order of magnitude of time scale at which various dynamic phenomena occur. The basic implication in this report is that an end must be put to static physiological models in studying human phenomena. The mechanisms must be described dynamically!

SUMMARY

A rational basis for a conductance model of clothing has been established. It derives from a phenomenological model of the physiological man as a thermoregulating system. The model of the system involves a liquid heat exchanger that permits radial and axial exchange of heat by forced convection or diffusion both in an inward and outward direction. This distributed heat exchanger, which may be regarded as a generalized hydrodynamic field, however, is partitioned by a sheath-like heat engine (the muscles) which thus establishes three

thermal zones; an internal well-regulated temperature zone, a transition zone in which heat is actively produced as a regulating function, and an exterior zone in which temperature is approximately regulated. The generalized hydrodynamic field, partially by virtue of the fact that an elongated body is being dealt with rather than a compact spherical body, is further partitioned by a distributed valve system to control the flux of heat-exchange fluid to the extremities. The heat exchange system is not a completely closed conduital system, so that some heat exchange fluid is available to the skin of the system through control valves for diffusive-evaporative heat flux. Thus the number of degrees of freedom of the system, and the potential number of controllers, include heat of combustion, radial conductance, axial conductance, mass flux of heat-exchange fluid to the skin of the system, storage (as measured by temperature) of thermal flux into the inner, outer, and peripheral zones. It is not completely clear as to what are the number of sensing elements that are used to generate error signals for feedback control. However, some novel points of view, with regard to sensing elements that have been suggested in the past, are that skin temperature is a slowly varying function of ambient temperature (at least in restricted extremity portions of the skin); with a relatively sharp break to ambient temperature at some critical ambient temperature. The break or possibly the slope of this curve at the break is used as a thermal sensing element for the control of axial conductance.

A second novel point of view is that extremity temperature is used as a null indicator of "comfort." While one feedback control loop involves shutting off of axial-fluid exchange, a second loop involves the system developing an activity level in which internal work is immediately degraded into heat in the muscle heat engine to maintain extremity temperature. The postulated existence of more than one operating mode of the system implies nonlinearity in controller and error-function detectors. This is borne out by the active continued dynamic response of the system at all times. The characteristic nature of this dynamic cyclic behavior has been demonstrated experimentally and shown to persist in all so-called zones of thermoregulation. Thus some significant modifications are necessary for the usual description of these zones. The harmonic content of the dynamic characteristics of the system is rich, but denumerable. In fact, approximately four major cycles at frequencies lower than the

breathing frequency have been demonstrated experimentally, and thus shown to contribute to the thermoregulation of the system. It would appear that thermodynamic equilibrium (i.e., a true steady state cycle) is established in the order of 3 1/2 hr. However, there are significant thermoregulating cycles down to the 100-sec level.

The integration of dynamic performance over an equilibrium time domain (i.e., 3 1/2 hr) permits the writing of an "equivalent static equilibrium" description of a heat balance in the form

$$\frac{\text{Net rate of heat production}}{\text{Temperature difference}} = \text{conductance}$$

or, mathematically

$$(M - L)/(T_s - T_o) = K$$

where

- M = rate of average heat production over the averaging period (i.e., the metabolism, assessable by oxygen and carbon dioxide consumption and production)
- L = rate of average flux of energy that does not pass through the clothes (i.e., heat losses from the lungs)
- T_o = an effective environmental operative temperature (this, in view of the dynamics suggested, requires an extension of the Gagge definition)
- T_s = mean skin temperature averaged over time for a particular mode of operation of the system (most comprehensibly, this mode should be a mode in which the system does the internal work necessary to maintain extremity temperatures. Whether the pure conductance-model description can be retained for other modes of operation of the system is moot and should be the subject of further experimental investigation)
- K = effective conductance of a set of clothes measured under the operational conditions specified. It may neither be constant, nor single valued

The method by which the conductance of a set of clothes might be computed theoretically begins to emerge conceptually. One must set up and solve a complete set of dynamic hydrodynamic field equations (suitably extended) subject to the dynamic boundary conditions determined by

the human system, and then integrated in time to produce the time-averaged conductance. Since an accurate description is not yet possible for the dynamic boundary conditions that obtain of the surface of the system, such computations are still moot. Either experimental data on the conductance of simple clothes systems are required, or a systematic analytic theory and experimental data on the dynamic thermoregulation of the human system are required to furnish data to build up and test clothing conductances.

Thus what is primarily needed is a dynamic analytic theory of the thermoregulation of the human system from the physiologist, experimental

data on clothes conductances from the clothing technologist, and a theory of clothes conductance from the physicist.

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COMPARISON OF RESPONSES TO 2800 MC AND 200 MC MICROWAVES OR
INCREASED ENVIRONMENTAL TEMPERATURE

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COMPARISON OF RESPONSES TO 2800 MC AND 200 MC MICROWAVES OR
INCREASED ENVIRONMENTAL TEMPERATURE

INTRODUCTION With the expanding use of microwaves at military and industrial installations, the biological effects of this form of energy is receiving increased attention. Investigation of potential hazards to man has necessitated a biomedical approach to this problem.

Numerous physiologic factors such as interspecies and interstrain variability; intraspecies age, sex, body size differences, or previous medication coupled with physical aspects of exposure such as frequency or power density result in marked alterations in the biologic response to microwave exposure. These factors must be considered before one can attempt to elucidate the effect of microwaves in the living intact mammal.

To date definitive information for man can be obtained only by extrapolation from animals along with comparison to meager human data. Man is an extremely complex organism with numerous interacting parts and systems. The various functions and reactions of the body are regulated by feedback and homeostatic or control mechanisms.

Because of this complexity and the variability of mammalian biological responses it is essential to investigate different species of animals under a variety of exposures before experimental results can be reliably extrapolated to man.

METHODS Dogs of small and medium breeds, albino rabbits and rats were exposed to 200 Mc continuous, 2800 Mc pulsed and continuous microwaves and in a room with controlled temperature and humidity.

Pulsed microwave exposures at 2800 Mc were carried out at the Verona Test Site. The methods of exposure have been described in previous reports.

The 200 Mc exposures were performed at the University of Buffalo using an end fire helical antenna in an anechoic chamber - 42 feet long x 10 feet wide x 9 feet high. Predetermined field intensity was 165 mw/cm².

Exposures at increased environmental temperature - 120° F, 50% humidity were carried out in the Environmental Temperature Laboratory at Griffiss Air Force Base.

A portable microwave generator was used in Rochester for the 2800 Mc continuous exposures.

Animals used in these experiments were toy fox terriers weighing approximately 4 kg, 8-20 kg mongrel dogs, 4 kg rabbits and 50 gm or 450 gm rats.

Thermal responses from 2800 Mc microwaves and hot room exposures were measured by continuous rectal temperature recording using a Yellow Springs thermistor. The thermistor probe was retained in a plexiglass shield inserted and fixed into the rectum of the animal. At 200 Mc microwaves, the thermistors seemed to pick up R-F and, therefore, it was found necessary to interrupt the exposures at regular intervals in order to obtain progressive rectal temperature readings by inserting the probe at each current break.

RESULTS

1. Thermal Response The thermal response in the dog exposed to 165 mw/cm^2 2800 Mc pulsed microwaves has been characterized and found to consist of three distinct phases. Phase I consists of the initial thermal response in which there is fairly rapid heating with a $2 - 3^\circ \text{ F}$ increase in body temperature within $1/2 - 1$ hour after onset of exposure. After this initial period the animal equilibrates and enters phase II - the period of thermal equilibrium. This period may last one hour during which time the temperature will cycle between 105 and 106° F . Phase III - period of thermal breakdown occurs when the temperature rises above 106° F , continues increasing rapidly until a critical temperature of 107° F or greater is reached at which time the animal may collapse and if exposure is not stopped death may ensue. The rabbit and the rat are extremely sensitive to this frequency (Fig. 1). A critical temperature is usually reached in 10 minutes in the rabbit and 20 minutes in the rat with no equilibration taking place. Body size does not seem to be a factor in the thermal response at this frequency. The rat and rabbit respond fairly similarly. A fox terrier weighing four kilograms, which is equivalent to that of the rabbit, responded the same as medium sized dogs ranging in weight from 8 to 20 kilograms.

At 200 Mc, 165 mw/cm^2 the dog equilibrates somewhat later than at 2800 Mc (Fig. 2), remains in equilibrium for a longer period of time on the average of 5 hours, before thermal breakdown is evident. This delay may be influenced by the frequent interruptions necessary during the exposure to facilitate temperature recordings. The four kilogram dog responds the same as the larger dog. The rabbit equilibrates slightly and critical temperature occurs within 30 minutes which is slower than at 2800 Mc. The rat shows some equilibration at $1/2$ hour with thermal breakdown absent at one hour.

Upon exposure to increased environmental temperature of 120° F , 50% humidity (Fig. 3), none of the animals equilibrate and critical temperature is reached between 30 minutes and one hour for all animals.

Body size may influence the thermal response within the same species in the hot room exposure. In this case, the small dog heated up more rapidly than the larger dog.

Variations in species response to these exposures are charted in Figures 4, 5, and 6. The dog displays a longer equilibrium period at 200 Mc than at 2800 Mc with lack of any equilibration at 120° F, 50% humidity (Fig. 4). The rabbit is more sensitive at 2800 Mc than at 200 Mc or the hot room (Fig. 5). The rat is most susceptible to the 2800 Mc with decreasing susceptibility in the hot room and lowest at 200 Mc (Fig. 6).

2. Influence of Pentobarbital Anaesthesia on the Thermal Response
Pentobarbital is being used in these studies as a tool in elucidating differences among species and exposure methods rather than as a means of restraint.

In the dog (Fig. 7), anaesthetization results in an increased thermal susceptibility to microwaves. This increased susceptibility was not noticed in the hot room. Whereas thermal equilibrium is achieved by the normal dog exposed to 2800 or 200 Mc microwaves, no such equilibration occurs under anaesthesia (Fig. 8). Thermal equilibrium does not develop in the dog when exposed to 120° F, with or without anaesthesia (Fig. 9). Time for reaching critical temperature in the anaesthetized dog, kept in the hot room, is the same as that for the normal dog.

In the rabbit and rat, exposed to 2800 Mc or the hot room, anaesthetization seems to delay the time for reaching a critical temperature. It should be noted that the anaesthetized animal's initial temperature was lower (Fig. 10, 11, 12). The anaesthetized rat appears to be more sensitive than the normal rat when exposed to 200 Mc.

A summary of the means of the thermal responses is included in Table I. This shows the extreme sensitivity of the rabbit to all forms of exposure with the rat intermediate.

Anaesthetization with pentobarbital seems to delay the approach to a critical temperature in the rabbit and rat exposed to 2800 Mc, but not in the rat subjected to 200 Mc. The anaesthetized dog seems to reach a critical temperature much more rapidly than the normal dog when exposed to either 2800 or 200 Mc. Anaesthetization does not seem to influence the time required for reaching critical temperature for any of the animals exposed in the "hot room."

Alteration in thermal response, induced by pentobarbital, may be explained on the basis of the pharmacologic action of this agent. The anaesthetic dose of pentobarbital used in these exposures, has been shown to depress the vasomotor center with resultant peripheral vasodilatation.

In the rabbit, the large surface area of the ears is said to function in heat loss by radiation and conduction. Vasodilatation

produced by pentobarbital no doubt results in a more efficient cooling mechanism, thereby extending the time for reaching a critical temperature in the case of both microwave and hot room exposure. In the rat, heat loss by radiation and conduction is quite likely also made more efficient with vasodilatation. The hairless, or thinly haired portions of the body such as the limbs become more effective thermal regulators.

The dog makes use of the evaporative surfaces of his pharyngeal and buccal cavities. Under normal circumstances during panting, the rapid movements of air through the mouth and upper respiratory passages facilitate cooling by increasing the loss of heat by convection from the mucous membranes of these parts. Although vasodilatation would improve thermal regulation, the anaesthetized dog does not pant, therefore, eliminating his most efficient thermal regulator.

These results indicate that vasomotor integrity is an important factor in regulating the thermal response to microwaves.

3. Relationship Between Rectal and Skin Temperature In several cases, temperature at the surface of the skin was measured and compared with simultaneously obtained rectal temperatures (Fig. 13). In this particular case, the side facing the horn was continuously monitored using a thermistor placed on the skin surface. At 2800 Mc the temperature difference between the skin of the side facing the horn, and the rectal temperature, gets smaller as radiation progresses for the first 30 minutes then reaches equilibrium while the rectal temperature increases.

"The Thermal Circulation Index" of three of these dogs was calculated (Fig. 14). Two of the dogs were exposed to 165 mw/cm^2 at 2800 Mc. On one, the skin temperature readings were obtained for the side that was facing the horn. On the other dog, the temperature was recorded for the side farthest from the horn. On the side exposed to the horn at 2800 Mc, the physiological thermal gradient, or difference between skin and rectal temperature, is small while the physiological thermal gradient for the surface on the far side of the horn is large.

At 200 Mc, 165 mw/cm^2 the one dog studied revealed essentially little change from its initial thermal circulating index. These differences in thermal circulation index are no doubt indicative of the depth of microwave energy penetration.

4. Response to Drugs of Known Pharmacologic Action As indicated previously, dogs exposed to microwaves while under pentobarbital anaesthesia show a much more rapid thermal response than normal dogs. The anaesthetized dogs do not reach the stage of thermal equilibrium.

In order to extend these observations and to determine how anaesthetization, sedation, or tranquilization affect the microwave response, four dogs picked at random were exposed to 165 mw/cm²-10 cm microwaves in a latin square experimental design, while under medication with Pentobarbital sodium, Morphine sulfate or Chlorpromazine. At least one week elapsed between exposures. Continuous rectal temperature recordings were obtained on each dog.

The data, have been analyzed and summarized (Table II). Thermal equilibrium is not reached with Pentobarbital. Temperature increase is more rapid with Morphine than in unmedicated dogs; thermal equilibrium is reached more rapidly and continues for a longer duration. Chlorpromazine surprisingly resulted in the longest equilibration, the dose, however, is quite low. Thermal breakdown under Chlorpromazine occurred after approximately the same equilibration time as with unmedicated dogs. Since thermal equilibrium is not achieved while under Pentobarbital medication, a critical temperature is reached much earlier in these dogs.

Analysis of variance for the first 30 minutes of exposure indicates that Pentobarbital results in a significant increase in the thermal response. The order of drug administration or previous microwave exposure did not influence the thermal response to weekly exposures.

Additional analysis of the time required to reach a 4° rise in temperature, revealed that Pentobarbital and Morphine significantly enhanced the rate of temperature increase when compared with unmedicated or Chlorpromazine treated dogs. There was no significant difference in response between chlorpromazine and controls although the former did decrease the time required for a 4° rise in temperature.

An analysis was made of cooling rates by comparing the time after the power was turned off for a 4° drop in temperature to occur. Only Pentobarbital required a significantly longer time to cool. Morphine was a close second but not statistically significant. This would definitely indicate an impairment of the thermal regulatory mechanism by pentobarbital, and possibly by morphine.

Further evidence of this interference was seen upon examination of the thermal curves. In each case where pentobarbital was used there was a one-half to one degree rise in temperature for 5 minutes after the exposure ended.

A further insight into the effect of these drugs on thermal regulation can be obtained by analysis of the cooling curves. In all cases temperatures returned to below initial. The mean time for reaching initial temperature was:

| | |
|----------------|---------------|
| Controls | - 41 minutes |
| Chlorpromazine | - 49 minutes |
| Morphine | - 1 1/2 hours |
| Nembutal | - 2 1/2 hours |

5. Hematologic Changes Hematologic examination was performed only in the dogs exposed to 2800 Mc and 200 Mc microwaves (Table III).

Comparison between white count changes, occurring among dogs exposed to 165 mw/cm² at 200 Mc and 2800 Mc, reveals an increase immediately after 200 Mc exposure as opposed to the 2800 Mc induced decrease. This may be due to the longer duration of exposure at 200 Mc, since similar findings were reported previously in dogs exposed to 2800 Mc where white cell increase may have been time dependent.

Differential changes. Evaluation of the differential white cell changes suggests a stress effect, which possibly is related to duration of exposure.

Hematocrit changes. Hematocrit changes for dogs, exposed to 200 Mc, were greater than with 2800 Mc which may also be explained by the longer periods of exposure.

6. Head Irradiation of the Rabbit The head of the rabbit was exposed to 2800 Mc continuous microwaves while the remainder of the body was shielded. In all cases the head was irradiated from the side.

The results are summarized in Table IV. Although these data are preliminary in nature, they are quite consistent.

From these exposures, it is learned that apparently the rabbit cannot tolerate more than 160 mw/cm² to the head for a period longer than 30 minutes and hyperpyrexia develops from head irradiation alone. Pupil dilatation of the eye on the exposed side may be indicative of brain stem damage. The eyes of the surviving rabbits were examined, at periodic intervals for two months, with no damage evident.

This program will continue in order to more clearly define time-intensity factors for survival and pathologic changes which might occur.

7. Response of Rats Exposed to Microwaves and X-Irradiation Rats were exposed, whole body, to both x-ray and 2800 Mc continuous microwaves (Table V).

Rats were arranged in six groups of five each. Group #1 was retained as controls. Group #2 was exposed to Microwaves 39 mw/cm²

for 38 minutes in the morning. In the afternoon Group #2, Group #3 and Group #4 were exposed to X-ray (700 r, 250 kvp, 18 r/min). Group #3 was exposed to microwave that same afternoon. Of the two remaining groups, Group #6 was exposed to microwave and X-ray simultaneously in the morning and Group #5 was exposed to microwave alone in the afternoon.

The rats exposed to microwave alone, at twenty-four hours post-exposure, showed a marked increase in WBC. Seven days afterwards, the WBC decreased and at fourteen days post there was a secondary increase. By the twenty-first day post-exposure, the WBC of these animals had returned to the normal range. The WBC and the hematocrits of the groups that received ionizing radiation followed a typical post irradiation response for the rat.

The mortalities that occurred in most of the groups were within the range expected for this dose of X-irradiation, except for the one animal in Group #6 that died twenty-four hours post-exposure. This death can be attributed to microwaves alone.

The mortality data were tested by Chi Square with the result that there was no significant difference in the mortality among the groups. Limitation to this test was the small number of animals in each group. Although there was an apparent difference in the primary drop of the WBC and in the recovery of the WBC in the groups that received microwave and X-ray, as compared with the group that received X-ray alone, this difference is not significant when tested by the students t-test.

Because of the limitations presented by the small number of animals used in each of the groups, this work should continue using groups consisting of larger numbers of animals.

DISCUSSION Numerous physiologic factors such as interspecies and interstrain variability, intraspecies age, sex, body size differences, or previous medication coupled with physical aspects of exposure such as frequency or power density result in marked alterations in the biologic response to microwave exposure. These factors must be considered before one can attempt to elucidate the effect of microwaves in the living, intact mammal.

For a first approximation of the biological effects of microwaves, characterization of the thermal response is essential. This has been done for three species of animals exposed to 200 Mc and 2800 Mc microwaves. In these two types of exposure the inherent thermal regulatory capacity and the degree of microwave penetration and absorption more than the physical factor of body size difference would appear to be the determining factor in the animals response to microwave exposure.

Premedication with agents which interfere with thermal regulation will tend to alter the homeostatic capability of the irradiated animal.

Although non-thermal effects have not been elucidated in the whole body exposed animal, head exposure of the rabbit would indicate that there may be an effect on the brain stem of such exposed animals.

With the completion of the phase of the study concerned with characterization of the thermal response, logical extension of the program towards comparison of microwave effects with those of increased environmental temperature, exposure to microwaves under different environmental temperature, and conditions of hydration is planned.

More extensive clinical observations including hematology and blood chemistry towards better understanding of the picture of apparent adrenal stress is contemplated.

Head exposure will be extended to the dog and anaesthesia will be used as a tool for further investigation of the neurologic component of microwave effect.

SUMMARY AND CONCLUSIONS Dogs, rabbits, and rats were exposed to 2800 Mc pulsed, continuous, 200 Mc continuous microwaves and in a room with controlled temperature of 120° F and 50% humidity.

Thermal response of these animals under the different conditions of exposure were characterized and analyzed.

The rabbit appears to be the most sensitive of the three species, with the rat intermediate and the dog least sensitive.

Critical temperature is reached more slowly at 200 Mc than at 2800 Mc.

The dog reaches thermal equilibrium at 2800 Mc and 200 Mc but not in the hot room. The rabbit fails to reach thermal equilibrium during any exposure. The rat reaches thermal equilibrium at 200 Mc but not at 2800 Mc or the hot room.

Anaesthetization of the dog results in an increased thermal susceptibility which is not evident in the rabbit or rat. Vasomotor integrity appears to be a critical factor in regulating the thermal response to microwaves.

The use of the Thermal Circulation Index appears to give a good indication of the depth of microwave penetration.

The thermal response of dogs exposed to microwaves while medicated with Pentobarbital, Morphine or Chlorpromazine reveals that both Pentobarbital and Morphine interfere with the animal's thermal regulatory mechanism.

Head exposure of rabbits in a field intensity, greater than 180 mw/cm² for 30 minutes, resulted in hyperpyrexia and death.

Periodic eye examination in all surviving animals has not revealed any cataract production.

ACKNOWLEDGEMENTS

The cooperation of the following is gratefully acknowledged:

Dr. Clinton Osborne of the University of Buffalo for permission to use the 200 megacycle generator.

Lewis Miller and Joseph Palmiter of Rome Air Development Center; William Quinlan, Walter Krasavage, J. Kevin Mahoney and Kathleen Scheer of the Department of Radiation Biology, University of Rochester for their assistance in carrying out the exposures and clinical tests.

THERMAL RESPONSE OF VARIOUS SPECIES OF ANIMALS TO 2800 Mc PULSED MICROWAVE

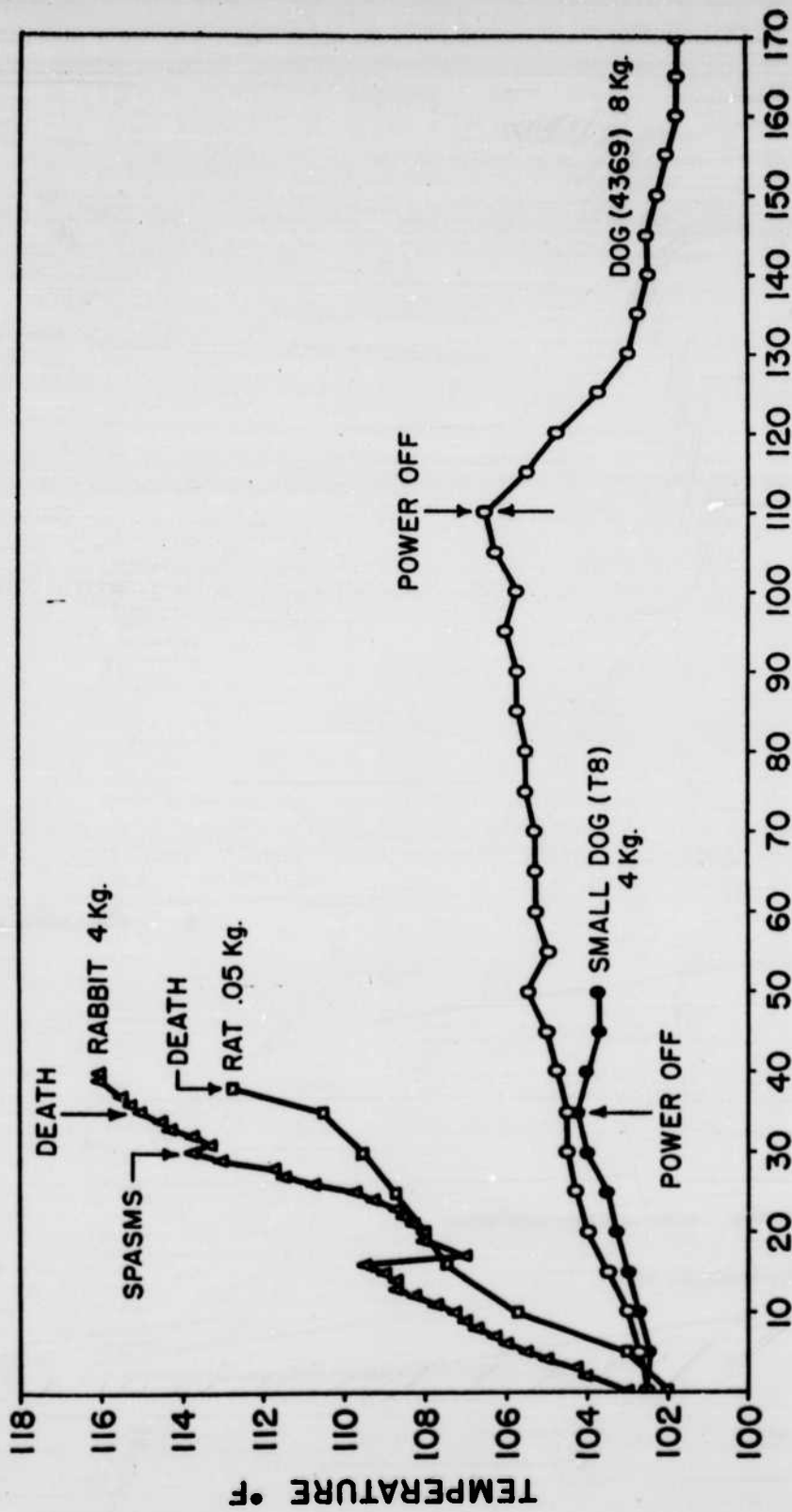


Fig 1
MINUTES

RESPONSE OF ANIMALS OF VARIOUS SIZE TO MICROWAVE EXPOSURE

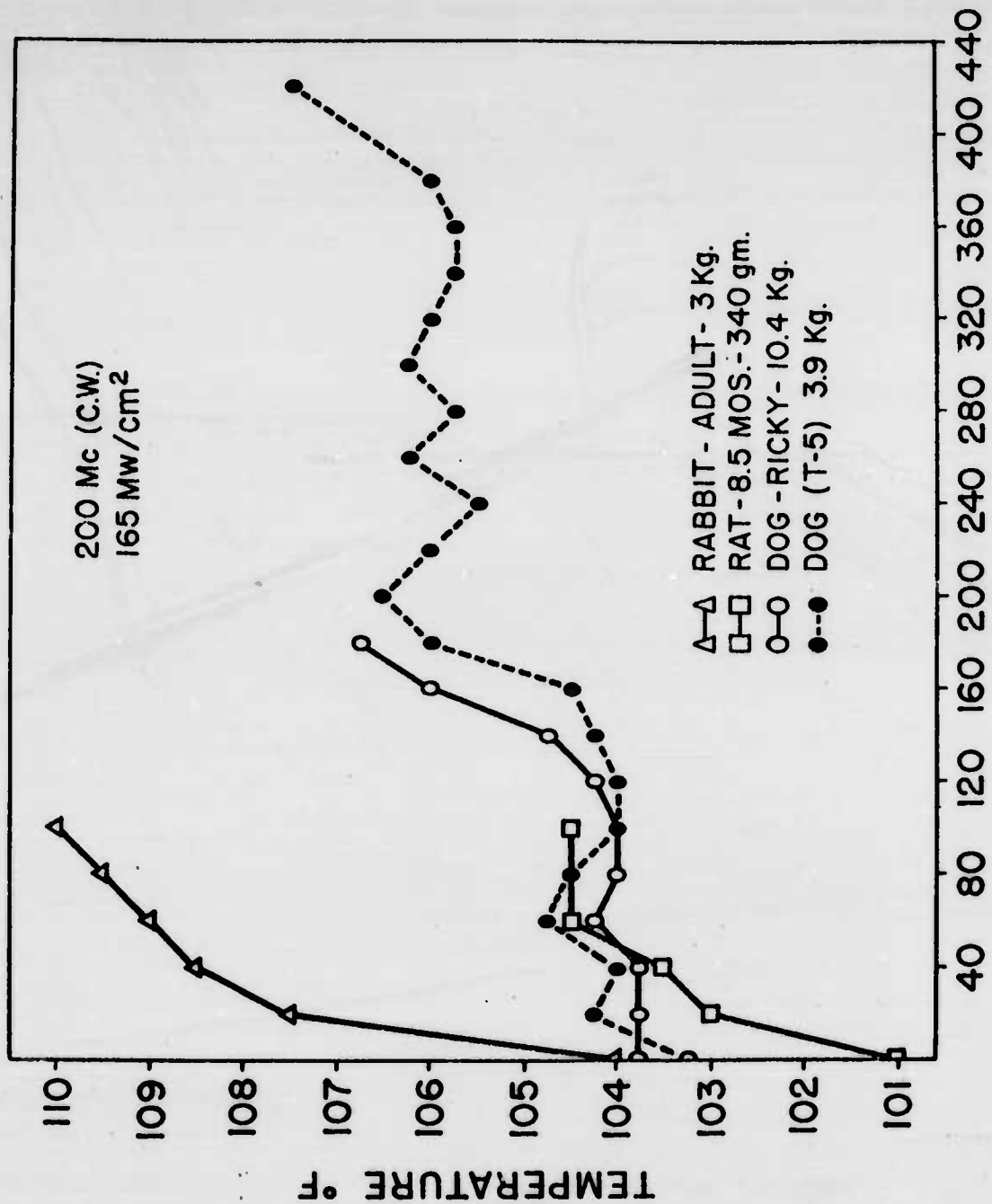


Fig 2 MINUTES

THERMAL RESPONSE OF
 VARIOUS SPECIES OF ANIMALS
 TO INCREASED ENVIRONMENTAL TEMPERATURE

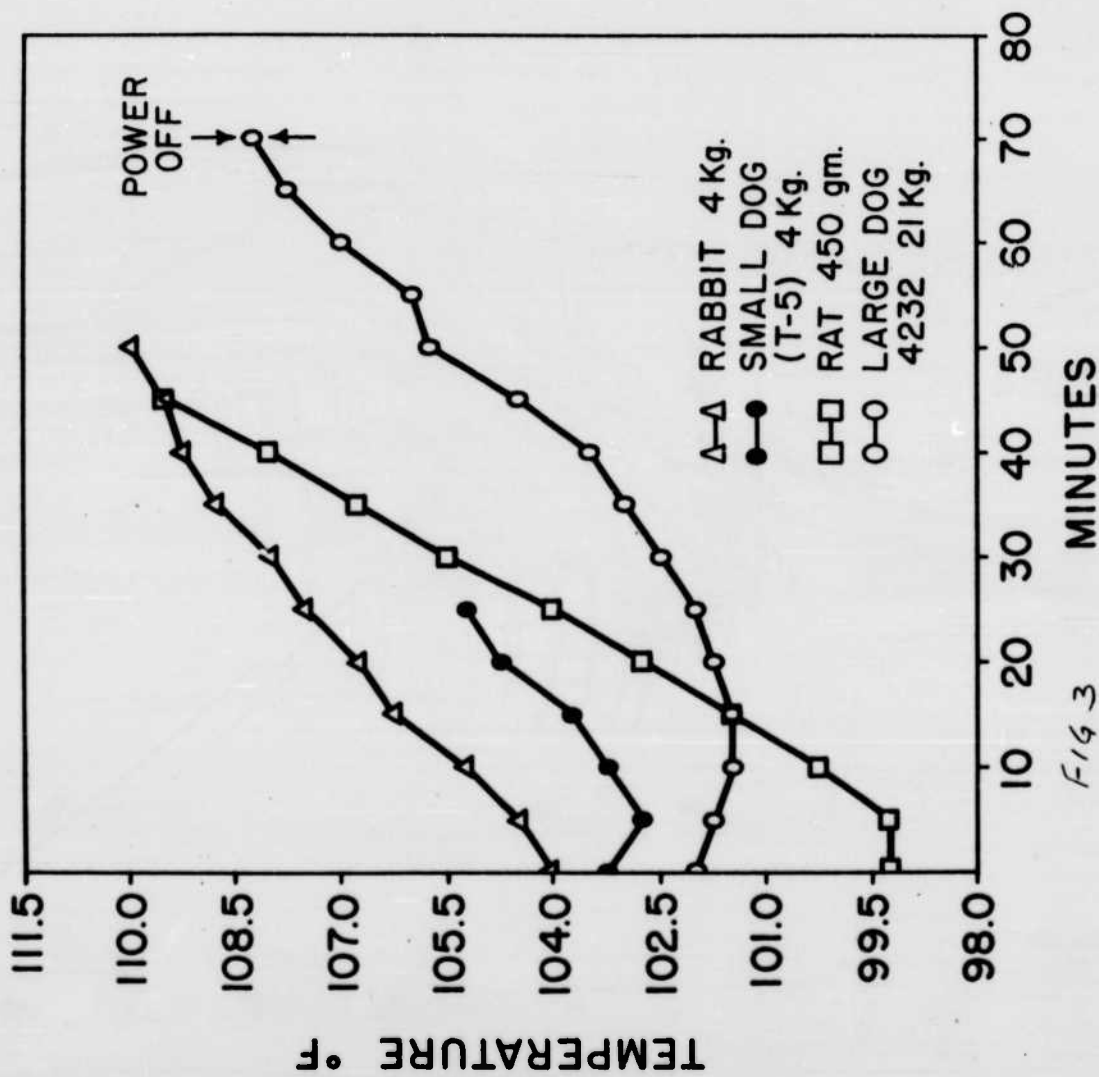


Fig 3 MINUTES

COMPARISON OF THERMAL RESPONSE OF NORMAL DOGS

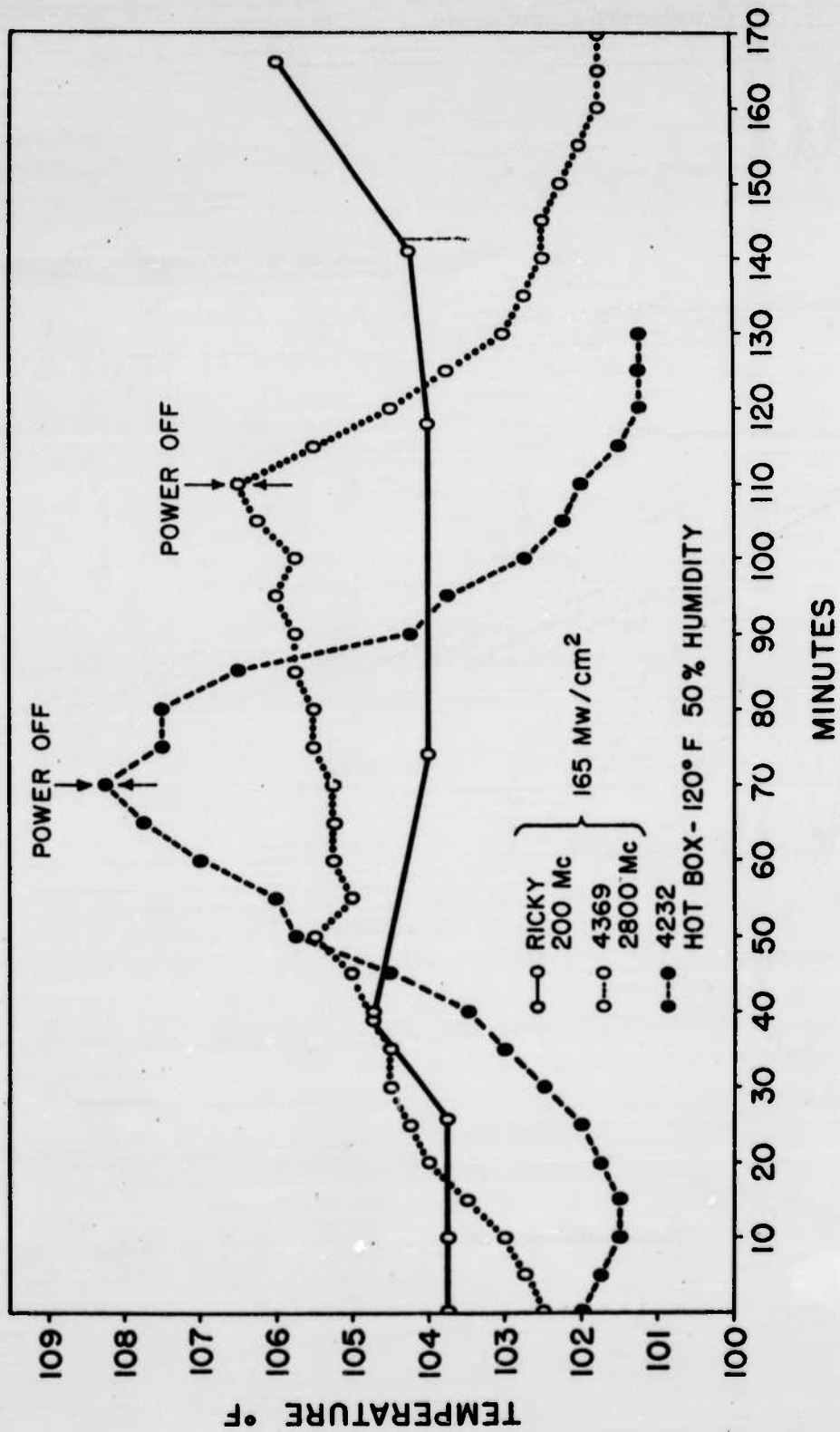


Fig-4

THERMAL RESPONSE OF NORMAL RABBITS

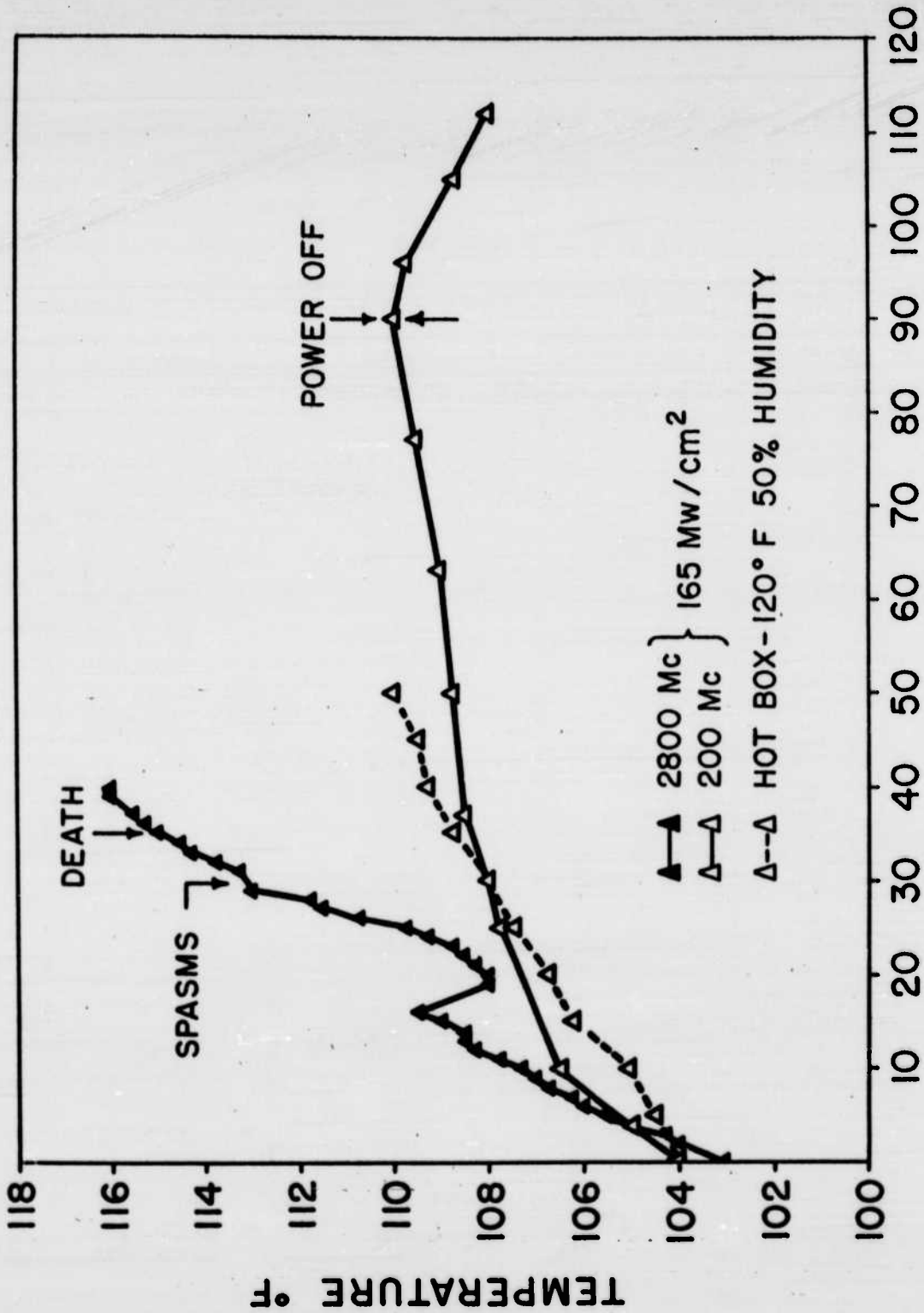


Fig 5 MINUTES

THERMAL RESPONSE OF NORMAL RATS

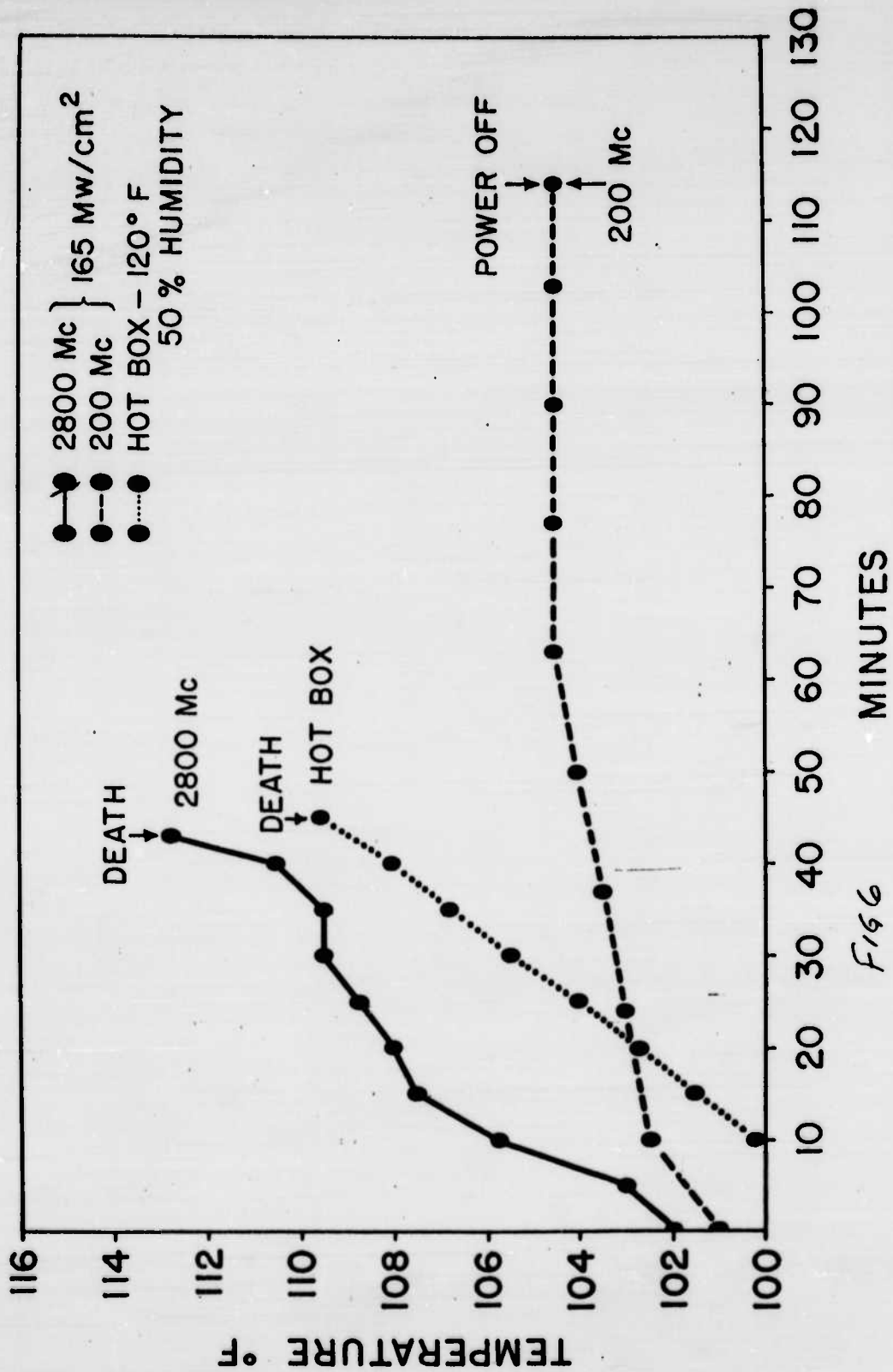


Fig 6

THERMAL RESPONSE TO MICROWAVE EXPOSURE

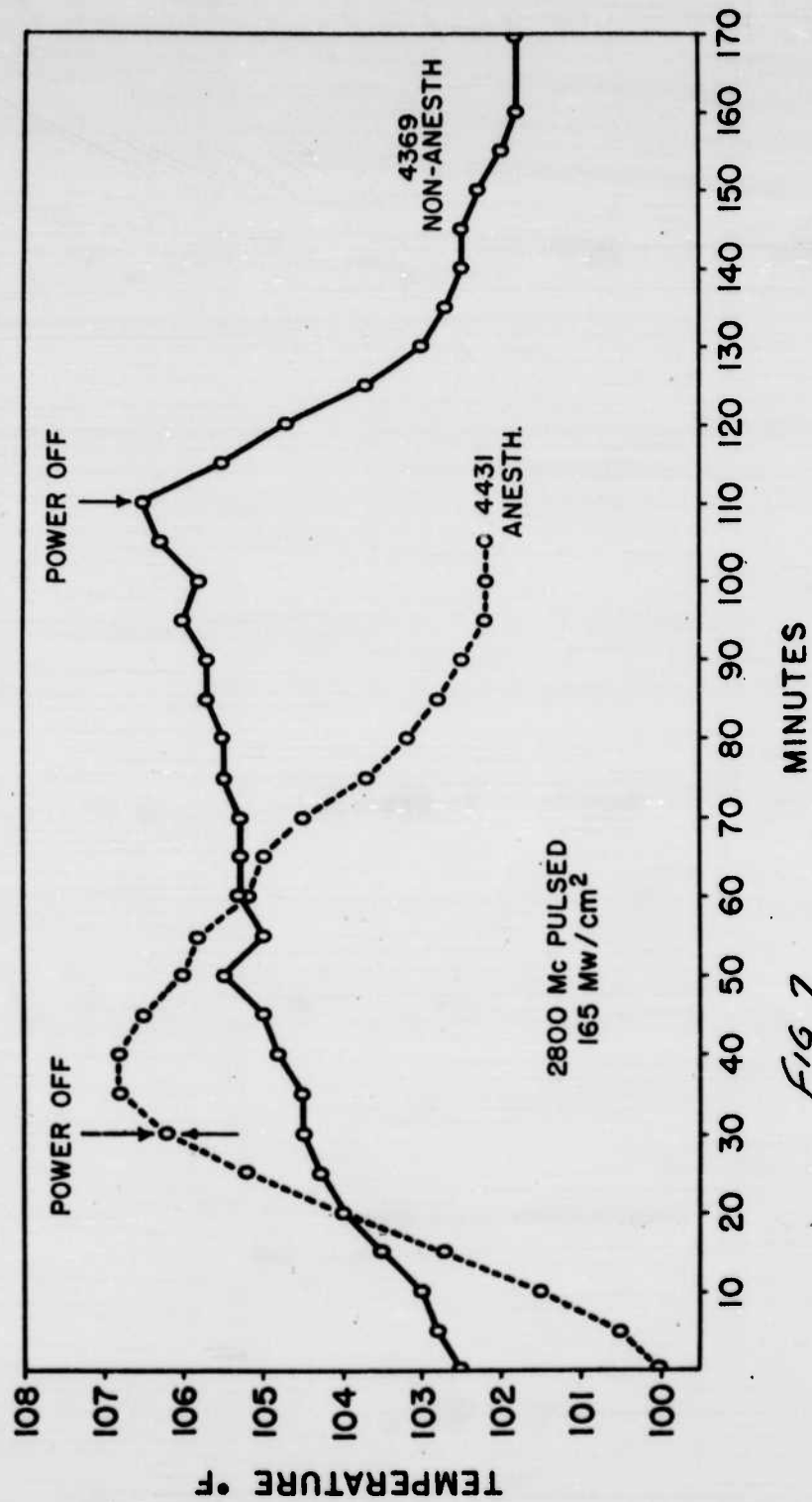
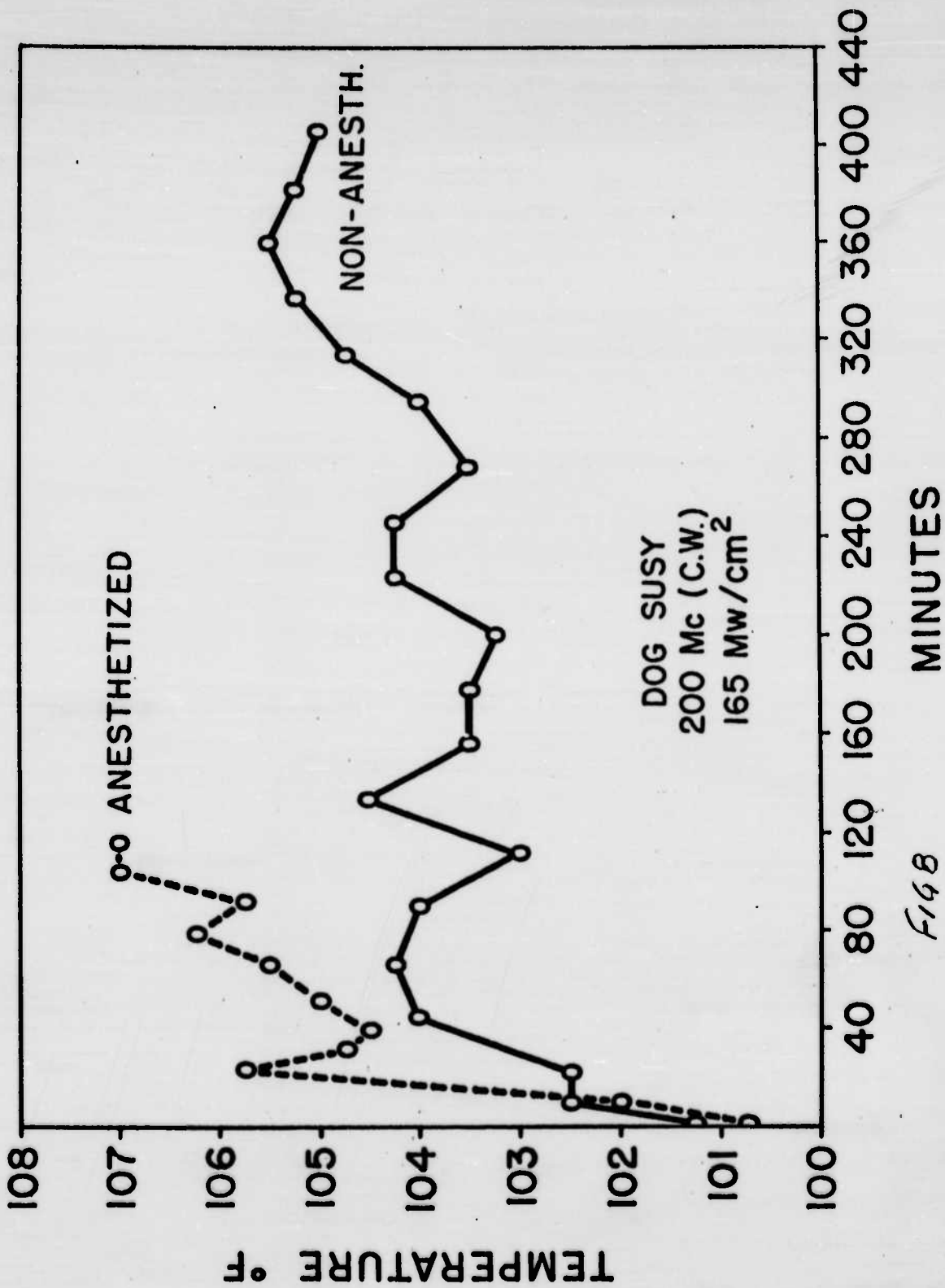


FIG 7

THERMAL RESPONSE TO MICROWAVE EXPOSURE



THERMAL RESPONSE OF DOG EXPOSED TO INCREASED ENVIRONMENTAL TEMPERATURE

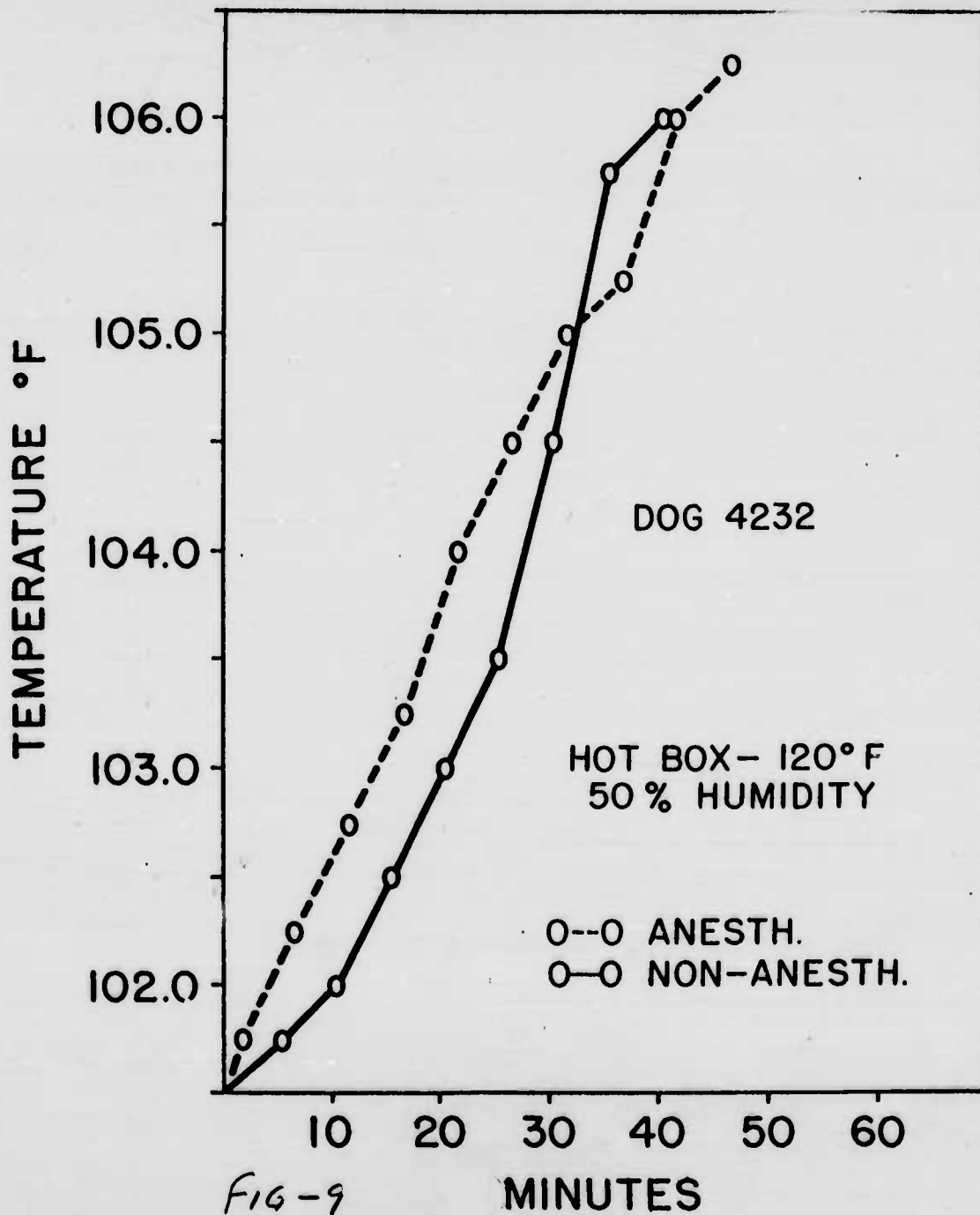


FIG-9

THERMAL RESPONSE TO 2800 Mc PULSED MICROWAVES
 165 Mw/cm²

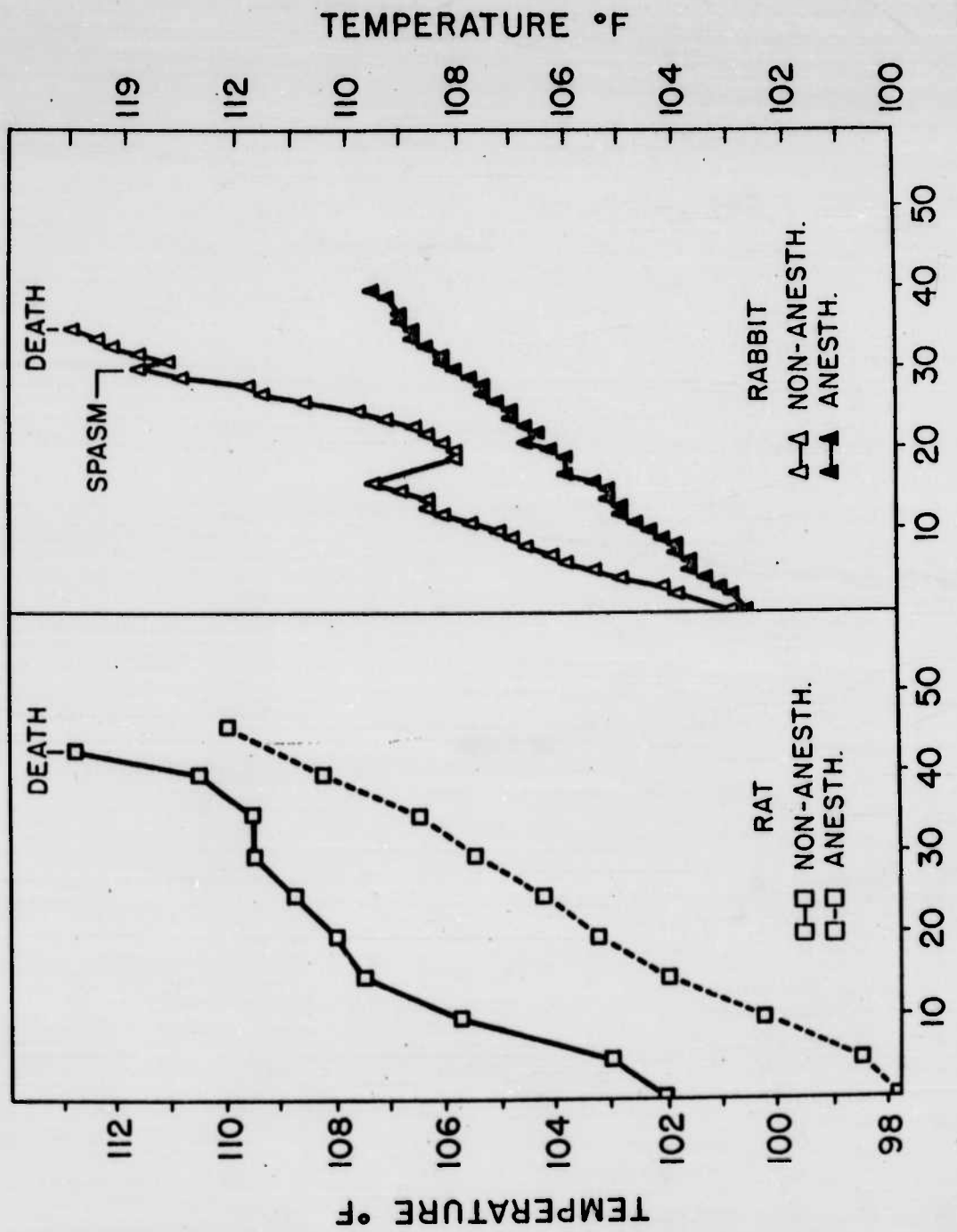


Fig-10 MINUTES

THERMAL RESPONSE TO MICROWAVE EXPOSURE

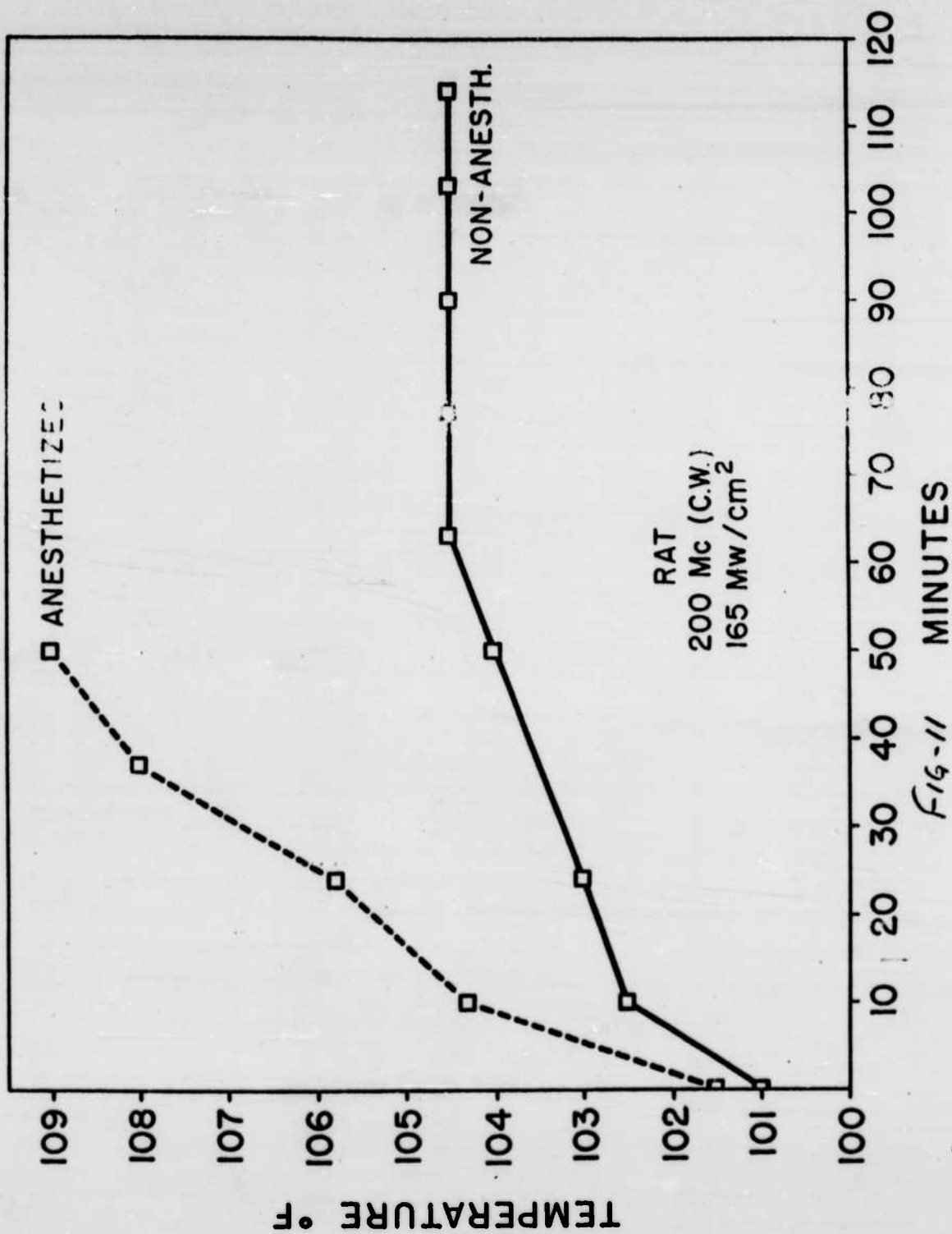


Fig-11 MINUTES

**THERMAL RESPONSE TO
INCREASED ENVIRONMENTAL TEMPERATURE
HOT BOX - 120°F 50% HUMIDITY**

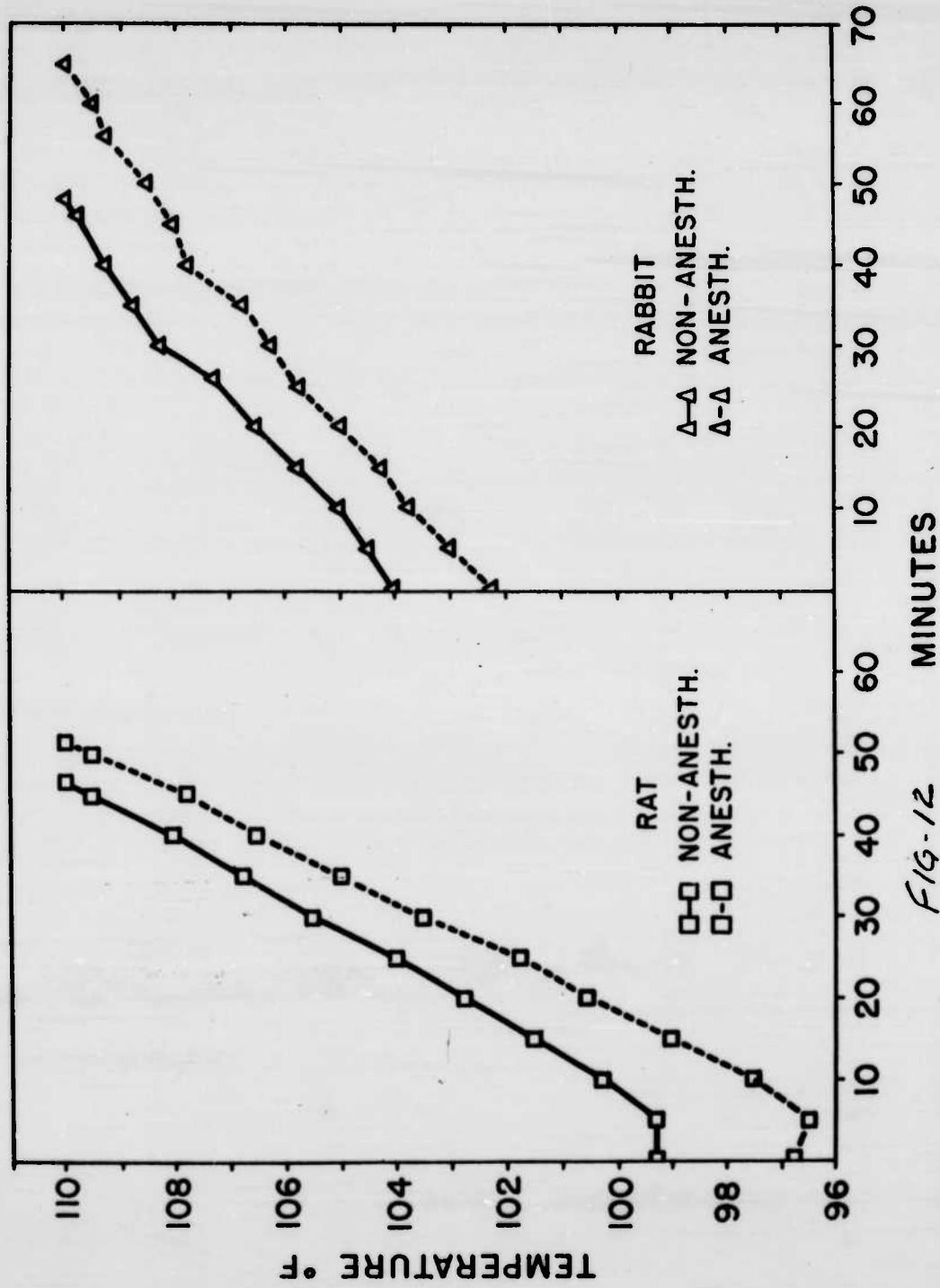


Fig-12

RELATIONSHIP BETWEEN RECTAL AND SKIN TEMPERATURE
DURING EXPOSURE TO 2800 Mc PULSED MICROWAVE

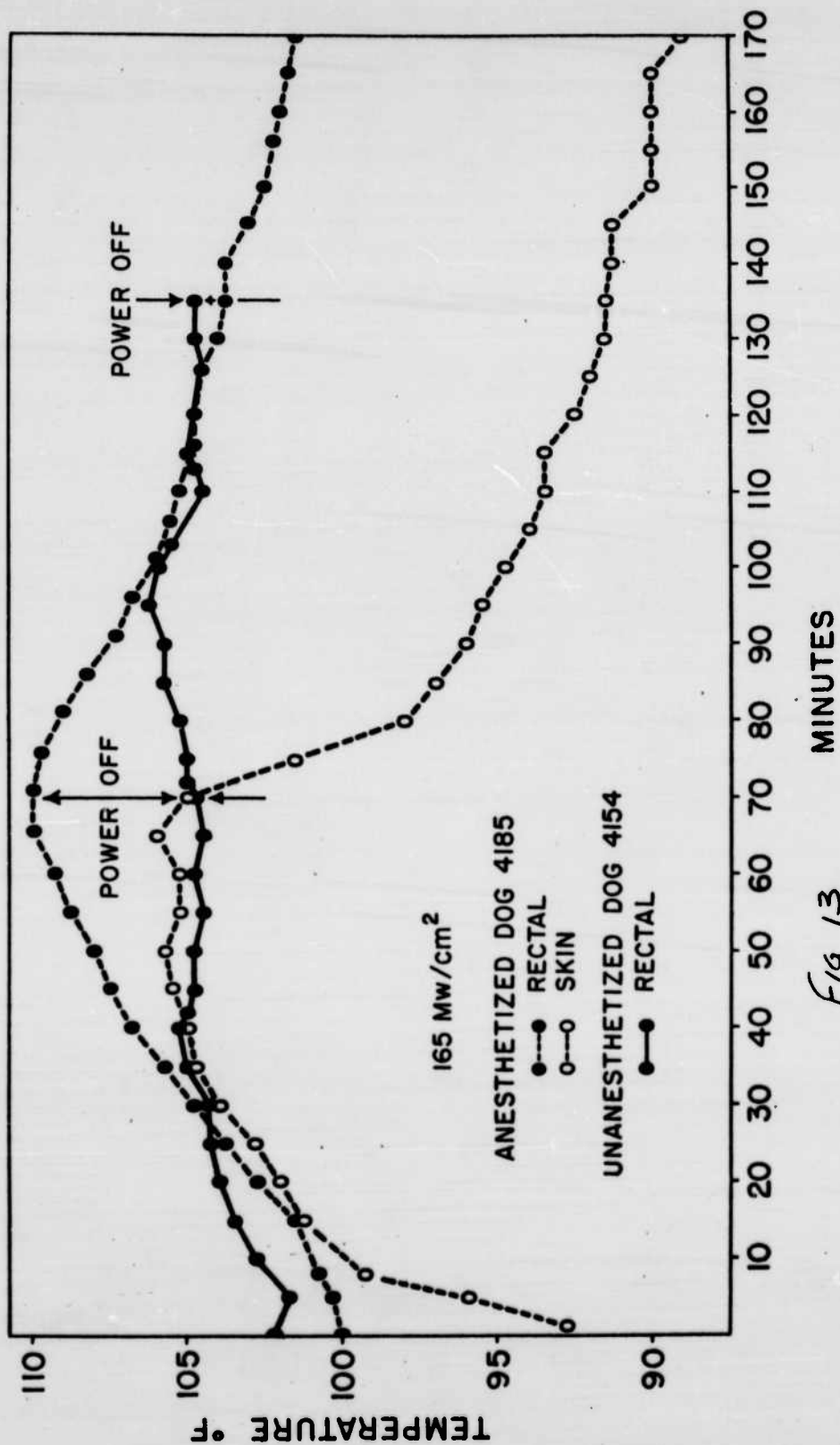


Fig 13 MINUTES

FIGURE 14 - THERMAL CIRCULATION INDEX FOR DOGS EXPOSED TO MICROWAVES

Field Intensity 165 mw/cm²

- - - - - - Dog 4185 - 2800 Mc - side facing horn
- - ○ - ○ - ○ Dog 4370 - 2800 Mc - side away from horn
- ⊙ - ⊙ - ⊙ Dog 4486 - 200 Mc - side facing horn

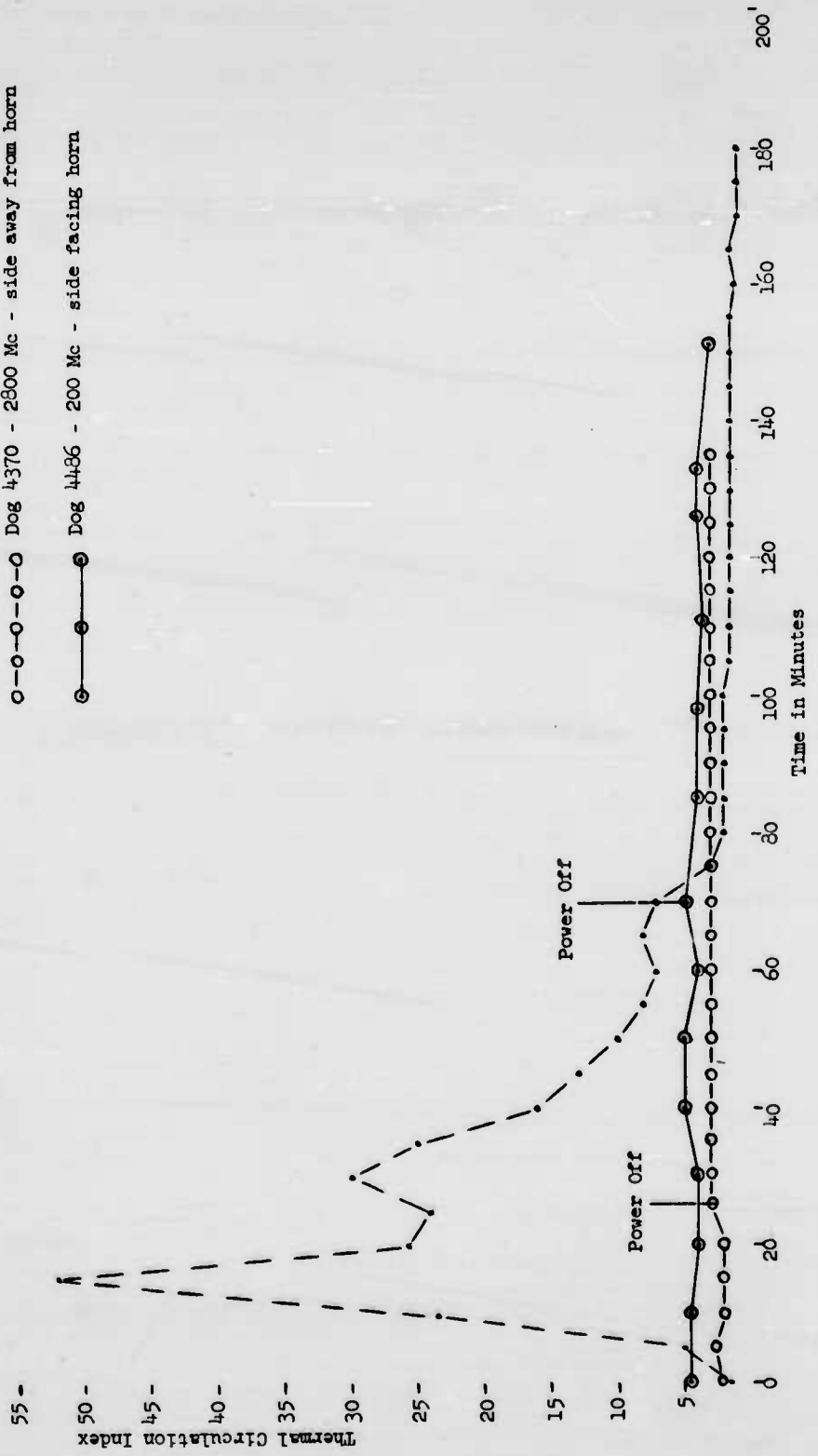


TABLE I - THERMAL RESPONSE OF ANIMALS EXPOSED TO MICROWAVES OR INCREASED ENVIRONMENTAL TEMPERATURE

(Time for Thermal Equilibrium)
(Time for Thermal Breakdown)

| Animal | Microwaves - 165 mw/cm ² | | | | Hot Room | |
|--------|-------------------------------------|--------------|-------------------|--------------|--------------------|--------------|
| | 2800 Mc pulsed | | 200 Mc continuous | | 120°F 50% Humidity | |
| | Normal | Anesthetized | Normal | Anesthetized | Normal | Anesthetized |
| Dog | *40/110 | 0/35 | 64/288 | 0/93 | 0/48 | 0/50 |
| Rabbit | 0/10 | 0/30 | 0/30 | 0/? | 0/30 | 0/45 |
| Rat | 0/22 | 0/40 | 30/55+ | 0/20 | 0/35 | 0/38 |

* Minutes

TABLE II - THERMAL RESPONSE OF DOGS EXPOSED TO MICROWAVES AFTER MEDICATION

| Drug | Dose | Onset of Exposure After Administration of Drug (minutes) | Thermal Equilibrium (minutes after onset of exposure) | Thermal Breakdown (minutes after onset of exposure) | Initial 30 Minute Heating Of Increase Mean \pm s.e. | Initial 30 Minute Cooling Of Decrease Mean \pm s.e. |
|----------------------|-----------------------------|--|---|---|---|---|
| Control | ----- | ----- | 36-154 | 154 | 1.31 \pm 0.47 | 3.75 \pm 0.30 |
| Pentobarbital Sodium | to Effect ca. 30 mg/kg i.v. | 0 | 0 | 36 | 4.44 \pm 0.47 | 2.25 \pm 0.40 |
| Chlorpromazine | 2 mgm/kg i.m. | 40 | 43-173 | 173 | 2.50 \pm 0.47 | 3.63 \pm 0.29 |
| Morphine Sulfate | 4 mgm/kg s.c. | 60 | 23-123 | 123 | 2.13 \pm 0.47 | 2.63 \pm 0.33 |

TABLE III - RESPONSE OF NORMAL DOGS TO MICROWAVES

| Frequency | Field Intensity | Duration (Minutes) | Number of Dogs | Temp. Change (°F) | WBC x 10 ³ | | HCT. | |
|------------------------|------------------------|-----------------------|----------------------|-------------------------|-----------------------|-------|------|------|
| | | | | | Pre | Post | Pre | Post |
| 200 Mc (Continuous) | 165 mw/cm ² | 300 | 7 | +2.9 | 10.70 | 15.54 | 46.0 | 52.0 |
| 2800 Mc (Pulsed) | 165 mw/cm ² | 120 | 15 | +3.1 | 15.23 | 12.84 | 46.0 | 48.5 |

TABLE IV - RESPONSE OF RABBITS TO HEAD EXPOSURE FROM 2800 MC CONTINUOUS MICROWAVES

| Rabbit No. | Distance From Horn (cm) | Field Intensity (v/cm^2) | Duration of Exposure (min) | Remarks |
|------------------------|-------------------------|------------------------------|----------------------------|--|
| M-38 | 1 | >1.0 | 30 | Excitement within 5 minutes, salivation, exhaustion, collapse, death. |
| M-40 | 5 | 0.700 | 15 | Excitement within 5 minutes, death. |
| M-41 | 5 | 0.700 | 25 | Temp. + 6°F, spasticity, death. |
| R-9 | 5 | 0.700 | 30 | Exposed eye-pupil dilated-burned. Opposite eye-pupil constricted. Death. |
| R-10 | 5 | 0.700 | 15 | Exposed eye-pupil normal. Opposite eye-pupil constricted. |
| R-11 | 5 | 0.700 | 15 | Exposed eye-burned and closed. Opposite eye-pupil constricted. |
| R-1 | 10 | 0.180 | 30 | Exposed eye-pupil dilated. Opposite eye-pupil constricted. Right foreleg paralyzed, death. |
| R-4 (Anaesthetized) | 10 | 0.180 | 30 | Exposed eye-pupil dilated. Opposite eye-pupil constricted. Temp. + 3°F, death. |
| R-6 | 11 | 0.160 | 30 | Exposed eye-pupil constricted. Opposite eye-pupil normal, survived. |
| R-7 | 11 | 0.160 | 30 | Exposed eye-pupil constricted. Ear on exposed side burned. Prostration. Temp. + 4.5°F, survived. |
| R-3 | 15 | 0.090 | 30 | Exposed eye-pupil constricted. Opposite eye-no change. Survived. |
| R-8 | 20 | 0.054 | 30 | No effect. Temp. + 3.75°F. |
| R-41 | 25 | 0.041 | 30 | No effect. |

TABLE V - RESPONSE AMONG RATS EXPOSED TO MICROWAVES AND X-RADIATION

| Group | Number of Animals In Group | Mean Rectal Temperature Of | Mortality- Days Post Exposure | 30 Day Mortality-% |
|--|-------------------------------|-------------------------------|----------------------------------|-----------------------|
| Controls | 5 | --- | ---- | 0% |
| Microwave plus X-Ray | 5 | 104 | 14, 11 days post | 40% |
| X-Ray plus Microwave | 5 | 108 | 21, 10 days post | 40% |
| X-Ray ¹ | 5 | --- | 10, 15, 16, 17 days post | 80% |
| Microwave ² | 5 | 107.5 | ---- | 0% |
| X-Ray and Microwave Simultaneously | 5 | 104.5 | 1, 12 days post | 40% |

1 - 700 r 250 kvp

2 - 2800 Mc continuous

39 mw/cm² - 38 minutes

STUDIES ON THE BIOLOGICAL EFFECTS OF MICROWAVE IRRADIATION
OF THE DOG AND RABBIT

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STUDIES ON THE BIOLOGICAL EFFECTS OF MICROWAVE IRRADIATION
OF THE DOG AND RABBIT

INTRODUCTION The current increasing use of microwave generators, the progressive development of greater power densities as well as the multiplicity of uses now being rediscovered for these energies demands immediate answers to possible hazards from such emanations. The major missions are (1) to detect and characterize the damaging effects of both acute and chronic nature caused by such exposures, (2) to establish, if possible, a lethal change in terms of timed exposure at specific power densities, and (3) to establish a tolerance or safe exposure level for individuals working with such generators similar to those already determined for noxious agents and ionizing radiations.

The above mission requires a biomedical approach with evaluation not only of the physiologic alterations which occur but also the related pathologic phenomena.

Selection of the dog as the experimental species of choice is not accidental. It was decided at the outset that (1) the selected animal should be more comparable to man in size and in possession of similar heat regulatory mechanisms, (2) the selected species should have well defined parameters of physiologic behavior in which observations on a single animal would be pertinent, (3) the exposures should be in free space and of a type possible in a hazardous situation in the human, and (4) because of the great variability in present physical measurement techniques, consistency in biological behavior would indicate presence of a uniform exposure. It is recognized that the experience in this laboratory with 15 or more years of problems in canine physiology and pathology could not be lightly disregarded.

In the following sections studies are reported on dogs exposed to 2880 megacycle electromagnetic irradiation from a AN/MPS-14 search radar adapted to an anechoic chamber designed for biologic experimentation. Exposures to energy levels of 100 and 165 mw/cm² for varying times were carried out. Clinical and physiological reactions are described. Characteristics of heat induction and recovery factors are analyzed on normal and anesthetized dogs. Certain responses suggest physiological adaptation to thermal energies. Changes in hemodynamics following various energies as measured by blood volume assay are given in addition to information on the behavior of the cellular elements. Certain specific pilot physiologic studies of comparative nature including red cell life, alteration of thyroid function and change in fat absorption are reported. Specific studies in the development of high frequency burns are reported. Investigations on combined exposure to electromagnetic and ionizing irradiation are analyzed. Comments concerning the hazardous nature of this energy are given.

METHODS This project is a cooperative effort between personnel of the Medical Division of the University of Rochester Atomic Energy Project and those of the Rome Air Development Center. At the

Rochester laboratory mongrel dogs carefully standardized as to age, sex and weight are kept for a holding period to insure good health. In view of the fluid changes which occur during and after exposure, these animals are kept on a constant food and water intake for a minimum of one week prior to exposure. They are then transported to the Verona site (115 miles) in a station wagon, rested overnight and exposed on the following day. Most animals are returned to Rochester the night of exposure except for those which are held at Verona for chronic or daily exposure. The distance of transport together with weather conditions sharply limits the number of exposures which can be conducted.

The source of r-f power is a radar set AN/MPS-14 operating at a frequency of 2880 megacycles and producing in excess of 2000 watts of average power. The pulse type emission utilizes a two-microsecond pulse width and a 360/sec pulse repetition frequency. The power densities available in the exposure chamber vary between the limits of 1.2 watts/cm² to less than 50 milliwatts/cm² depending upon distance from the antenna. In order to estimate the power distribution across the animal's body, contour lines of equal intensity have been measured. Analysis of the graphs indicates a relatively uniform field across the animal's body with the energy at the periphery differing by less than 20 per cent from that at the center.

To simplify the determination of the power density and aid in the interpretation of data, a microwave "free space" room was constructed for animal exposures. The exposure room, approximately 7' x 7' x 15', is lined with commercial microwave absorbing material. According to the manufacturer, a maximum of two per cent of the energy will be reflected from the surface of the absorber. Under these conditions, a "free field" for all practical purposes is produced.

A plexiglass cage was constructed to confine the animal during exposure. This was made of such a size that the limits of the cage were within the 80 per cent continuous exposure lines as measured in air by standard techniques. The material proved to have excellent dielectric properties and structural strength, with excellent transmission and tolerable distortion from diffraction of the radiation. During exposures it was noted that the animal preferred to remain at the outer limits of the field so that the dosage as indicated at each exposure level is undoubtedly on the high rather than low level. Rotation is not used. With the maximum penetration of ± 3 cm and the absorption of the major portion of the dose in this superficial layer, the total amount of exposed vs non-exposed tissue can not be stated with surety. However, since animals were free to turn to any desired position, and were observed to do so, it is considered that mechanical rotation is not required. The development of bilateral chest burns in specific animals is best explained by a uniform exposure to both sides of the thorax.

With the success of the initial plexiglass single cage, a double separated cage of the same overall size limit was constructed. Although increased reflection of radiation undoubtedly occurs, the advantage in certain exposure studies of simultaneously studying one animal together with his control may outweigh the defects of reflection and diffraction.

Dogs were exposed at two power densities (100 mw/cm² and 165mw/cm²) for different durations of time. Clinical and laboratory tests are performed before and after exposure. During exposure continuous observations of his response are recorded. Animals are placed in the cage for a 15-minute period prior to exposure during which rectal temperatures are made at five-minute intervals. If temperature is constant, exposure is started. Following exposure, rectal temperatures are recorded usually at 5, 15, 30, 45 and 60 minutes or continuously. Blood for hematological examination is obtained by jugular puncture within one minute after cessation of power. Following exposure the animal is observed for general appearance, alterations in behavior and gait, and desire for water. Superficial neurological examination sufficient to characterize motor or reflex changes is performed. Animals used in these studies include:

1. Normal healthy dogs of both sexes
2. Normal dogs exposed to 200 or 400 r whole body gamma radiation from a Cobalt-60 source four or 11 days prior to microwave exposure.
3. Dogs from groups of animals receiving x or gamma irradiation at the LD/50-90 level several months to 3 years prior to the microwave exposure. These animals are designated in this report as survivors.
4. Normal rabbits. Some of these animals are used for studies of antibody production and decay rates.

Exposures to both dogs and rabbits are reported as either high power (165 mw/cm²) or medium power (100 mw/cm²).

Laboratory methods are those used in routine clinical studies. Specific methodology for fat absorption, thyroid uptake, and red cell life is given in those special sections of the report where pertinent.

Pathological techniques include gross examination including opening of the skull, microscopic study with hematoxylin-eosin as standard and special connective tissue, fat and nerve tissue stains where indicated. Results will be reported in a subsequent publication.

RESULTS For clarity and complete reporting of experimental results, previously published data from ASTIA Document No. AD 21211C are being included.

Characterization of Thermal Response Pattern in the Dog Continuous thermistor recording of rectal temperature changes in the dog under conditions of exposure permits a careful study of the thermal

response pattern. At 165 mw/cm² (fig. 1) an initial period of heating occurs during which the rectal temperature increases two to three degrees F within 40 to 60 minutes after start of the exposure. During this time the animal shows open mouthed panting with extension of tongue, increased respiratory rate, restlessness and apprehension.

After this initial period of heating (40-60 minutes) a period of thermal equilibrium takes place. This persists for the next hour with the temperatures oscillating in a cyclic manner between 105 and 106° F. During this period the animal is more at ease with panting increasing and decreasing in a rhythmical fashion.

Next, changes occur more or less rapidly indicating an apparent breakdown of thermal regulation. A rapid increase in temperature occurs, the animal showing clinical signs of hyperpyrexia. Increased salivation, impaired locomotion, and excitability are noted which rapidly progress to acute distress and collapse with rectal temperatures measured between 107 and 109° F.

Within 15 minutes after removal from the cage, the surviving animal will stand but displays prominent weakness of hind quarters, staggering and incoordination. Dying animals remain in a state of collapse. Death which occurs within 30 minutes is followed by extremely rapid development of rigor mortis.

A motion picture has been made recording the specific signs and reactions described above.

Exposures to power levels of 100 mw/cm² (fig. 1) for periods of up to 6 hours (arbitrarily selected as a standard time) do not reach a lethal or even critical temperature. The initial period of heating is slight with the animal remaining in the phase of thermal equilibrium during the remainder of the exposure. Extension of the time beyond the 6 hour period has not been done. Undoubtedly with sufficient fluid loss and hemoconcentration, the phase of thermal breakdown can be reached at this exposure level.

The following tabular representation indicates the response of the dog to microwave exposure.

Response of the Dog During Whole Body
Exposure to Microwaves

1. Panting - rate increases with duration of exposure, some reach plateau, rate decreases and then increases.
2. Salivation - when present increases with duration of exposure.
3. Increased activity - appears agitated, moves around cage, then finally settles down in portion of cage furthest from center of field.
4. Responsiveness - attentive and responsive to sounds throughout exposure.

5. Vaso-dilation.
6. Akinesia.
7. Prostration.

Response of the Dog Immediately After Whole Body
Exposure to Microwaves

1. Disruption of equilibrium.
2. Increased rectal temperature.
3. Increased desire for water in most cases. Animals showing most marked response refuse water.

Detailed observations under fixed experimental conditions are described in Tables I and II. Findings are reported on 27 normal dogs, 11 dogs receiving 400 r of Co-60 ionizing irradiation plus subsequent microwaves and eight survivors receiving ionizing radiation from two months to several years previously. The results are reported in detail in ASTIA Document No. AD 212110 but will be briefly summarized here. Subsequent studies completely confirm the original findings.

The maximum temperature response of $+5.1^{\circ}$ F. occurs in the $165 \text{ mw/cm}^2/3 \text{ hr}$ dogs; the $100 \text{ mw/cm}^2/6 \text{ hr}$ animals showing a $+3^{\circ}$ elevation. Behavior of the irradiation survivors and acutely irradiated series was similar. Lack of the temperature response ($+1.4^{\circ}$ F) in the Co-60 plus $165 \text{ mw/cm}^2/2 \text{ hr}$ remains unexplained.

Attempted correlation with weight, sex, age or body surface area for the 2880 megacycle animals has produced negative results.

The most marked increase in hematocrit occurs in animals exposed for three hours at the 165 mw/cm^2 level. Changes at the 165 mw/cm^2 for two hours and the 100 mw/cm^2 for six hours are almost identical. Dilution toward pre-exposure levels and below immediately follows exposure and at the 24 hour period is complete.

Of interest is the post-exposure hematocrit reduction of 5-6% in the 400 r Co-60 plus microwave four days later. Since some specific vascular change is suspected to follow ionizing irradiation, this may be the first documented evidence. As indicated in Table II (one week after exposure) this reduction is exaggerated to a -19% and -21% respectively with control Co-60 at -10 and control microwave at -3.0 to -4.5%.

Leucocyte changes in the $100 \text{ mw/cm}^2/6 \text{ hrs}$ are negligible. At the 165 mw/cm^2 level, an initial decrease occurs at the two hour period; an increase at the three hour time may be associated with acute tissue damage. The gradual increase in all groups at the 24 hour level at which time the hematocrit has diluted often to below pre-exposure levels reflects major to minor tissue damage.

This is confirmed in a sense by the return to normal values in one week except in the $165 \text{ mw/cm}^2/3 \text{ hr}$ dogs and in the Co-60 irradiated animals. In the latter instance, the depression is typical of that seen in the Co-60 irradiated controls. Table III and IV show specific changes in hematocrit and leucocytes at all time periods.

Further studies of clotting time shown in Table V confirm earlier observations which indicated no change in either clotting time or clot retraction as a result of microwave exposure.

Table VI shows selected studies on certain typical animals. No changes in red cell fragility, blood sugar or NPN were noted. Blood viscosity measured at 25° C (equilibrated) parallels the increase in hematocrit which follows microwave exposure. This rapid test can substitute for the hematocrit in clinical use, or for emergency studies.

A rather startling and significant finding is noted in examination of the differential white cell changes in animals exposed to 2880 Mc energies at the two power levels and to 165 mw/cm^2 of 200 Mc continuous wave energy (Table VII).

At the $100 \text{ mw/cm}^2/6 \text{ hr}$ or 360 minutes, no change in the total leucocytes occurred. As an indication of possible injury, increase to 15,090 cells occurred after 24 hours. This was accompanied by the return of lymphocytes to pre-exposure levels. Of importance is the decrease of eosinophils from 558 to 220 cells with a later overproduction in 24 hours. With the parallel reduction in lymphocytes, this essentially performs a Thorne test of adrenal stress and constitutes a normal value.

It is noted that a similar reaction occurs with the 200 Mc. continuous wave energy although the series of animals is smaller. The eosinophil reaction is slightly greater (not statistical) and the lymphocyte reaction less.

With the $165 \text{ mw/cm}^2/139$ (30 to 180) min. exposure, the lack of eosinophil response and lymphocyte depression plus the low level at the 24 hour period would suggest a phase of adrenal exhaustion with persistent lack of recovery. Unfortunately examination of the blood of these animals at the 60 and 120 min. periods have not been carried out in sufficient numbers to determine whether the stress effect proceeds through to the exhaustion phase.

Burns A unique finding previously reported is the development of superficial burns on various portions of the body of the dog most common on both sides of the rib cage. These usually follow exposure to 165 mw/cm^2 and are well illustrated in the accompanying photographs and legends. A latent period of as much as five to six days following exposure may elapse, at which time the entire area sloughs away

leaving a deep clean wound identical in appearance with a third-degree burn. Prior to sloughing away, the central portion appeared to devitalize with development of a process suggesting a dry gangrene. Most burns occurred over the rib cages (bilateral) but others have developed on the neck and head, the latter two types being smaller in area. Healing without infection usually occurs in all except the survivor (ionizing irradiation) group where poor healing and considerable suppuration was observed. To date no scarring or keloid development is noted. Among the survivor dogs a delay in the appearance of the burns by as much as ten days is noted. In these animals healing even without infection was extremely slow. Pathological examination of burn site areas resembles those of a typical third-degree burn.

An observation made on one of the animals may be significant. This dog developed burns which healed within three weeks. Six weeks after initial exposure, this animal was again exposed with not only development of burns in a new site but a recurrence of burns in the original site.

Another observation of significance shows that dogs under pentobarbital anaesthesia, positioned in ventral recumbency with legs extended and perpendicular to the horn, develop burns in the loose portion of the skin between the trunk and front limbs and/or flank of the exposed side.

A greater incidence of burns follows exposure of dogs under anaesthesia. These "reverse third degree burns" (from the inside out) can be produced almost at will and with exposure times of as short as 30 minutes. The almost continuous rotation of the normal animal during microwave exposure essentially allows one side to cool while the other cooks. Under such conditions devitalization of a vascularly poor area cannot occur before the period of thermal breakdown of the animal is reached.

Factors known to be of importance in the production of these burns includes rate and/or time of heating, vascularization of the specific area, anaesthesia, and specific sensitivity of the individual animal. The possibility of standing wave production by reflection from rib cage, sacrum or skull is remote.

Effect of Previous Exposure to Ionizing Radiations on the Microwave Response Interest in the combined exposure of ionizing irradiation and microwave has been prompted by a number of factors. Electron tubes of microwave generators emit variable amounts of hard (+200 KV) x-rays often in the hazard range and in intensities of as much as 200 r per hour at one cm distance. Possible pathologic changes induced by thermal induction from microwave exposure may be synergized by associated ionizing reactions in the same tissue. The vibrational, rotational or polymerizing effect caused by microwaves on component

molecules may induce previously unobserved effects of either beneficial or damaging types. Finally the parallel use of microwave and ionizing radiation exposures may permit the first demonstration of a non-thermal reaction.

In Tables I and II preliminary results were given showing the effect of 400 r of Co-60 irradiation followed by microwave exposure four days later. Results as shown in Table VIII combine these initial results with the added animals necessary to make the groups more complete.

The results indicate that the thermal response of these animals is related to the energy absorbed. Previous exposure to ionizing radiation does not modify this response except in extremely high dosage of microwaves. In normal dogs a transient decrease in white cells occurs immediately after exposure. Within 24 hours a rebound occurs. This is followed in turn by a further decline at 48 hours. A gradual return to normal levels by the two week period occurs. In dogs surviving lethal amounts of radiation given two months to several years previously, the leucocytosis observed at 24 hours persists for several days. Animals exposed to Co-60 followed in four days by microwave irradiation do not demonstrate the leucocytic reaction. The depression of the white cells during the observation period parallels that in dogs given Co-60 irradiation alone.

A transient increase in hematocrit levels during exposure occurs in varying degree in all animals treated with microwave alone. In those exposed to serial ionizing and microwave radiations the hemoconcentration may persist to 24 hours with a progressive decline during the post-exposure period.

Analysis of the mortality data shows no difference in animals receiving Co-60 alone and those receiving Co-60 plus 100 mw/cm²/360 min. In dogs exposed to Co-60 plus 165 mw/cm²/120 min. a total mortality of 70% suggests a definite synergistic lethal effect.

Other combined ionizing and microwave exposure experiments are reported in the accompanying paper on comparative effects.

Residual Effects In observations on 100 animals extending over a period of 12 months in the early cases, residual effects have not been noted. In all cases unless immediate death with rapid rigor mortis occurred, recovery has been prompt and uneventful.

Periodic examination for cataracts by slit lamp has not shown any eye abnormalities. Estrus has been unchanged and pregnancy in one instance has been normal.

Clinical behavior has been normal in all animals to date. Nutritional status has been good and weights are stable.

Miscellaneous Effects A number of pilot studies of various types have been carried out on these animals under varying conditions of exposure. Sufficient observations for reporting have been made on (1) fat absorption, (2) red cell survival, and (3) reaction of antibody response. Other studies on thyroid function indicate a reaction of damage one week after exposure to $165 \text{ mw/cm}^2/3 \text{ hr}$ levels with gradual return to normal in 30 days. Other studies are reported as follows.

Alteration in Fat Absorption. In one normal dog exposed to $165 \text{ mw/cm}^2/2 \text{ hr}$ a marked delay in fat absorption occurred after 8 days with a return to normal levels at 20 days. The pattern after recovery continued to be disturbed with a flat response up to one hour after ingestion of the meal. (Fig. 2)

In the animals exposed to 400 r of Co-60, results indicate a slight delay with no apparent deficiency in six hour absorption up to 4 days post-exposure (Fig. 2). In survivor animals a variable response is noted. In general an increase in fat uptake occurs. Except in one animal a consistent depression of the one hour uptake is found. (Fig. 3)

In the serially exposed animals, response is variable although in this instance 1 - 4 days elapsed between Co-60 and microwave irradiations. These are shown in Fig. 4.

Studies of Red Cell Life. In one animal exposed to $165 \text{ mw/cm}^2/2 \text{ hrs}$ an increased loss of red cells over the normal control for this animal occurred. The change in this instance was a decrease to 18 and 20 against control values of 28 to 32 (initial apparent half times).

In animals exposed to Co-60 alone, initial apparent half times were 14.5 and 18 per ml whole blood. In those exposed to both Co-60 and microwave observations of 10, 11 and 18 were made on the three animals studied. The lower or more reduced times occurred with the higher radiation dosages.

The nature of the changing pattern in all types of exposure is shown in Fig. 5 which shows the per cent daily rate of loss per ml over that of normal aging. From this study it suggests that the combination of two injurious agents at the higher exposure levels makes for additivity in red cell decay over either alone.

Antibody Response. Determination of the antibody half life has been made in rabbits exposed to pulsed wave 2880 Mc at 100 mw/cm^2 until a critical rectal temperature is reached. This usually occurs within 30 minutes. Initial observations can be reported as follows.

A. Effect on antigenic stimulation. Thirty-six rabbits were used in the study and were divided into four groups according to the

time of the first injection of sheep RBC, as follows: (1) sheep RBC given I.V. two days before exposure to 30 minutes of microwave irradiation (100 mw/cm²), (2) antigen given I.V. 4 hours before exposure to microwave irradiation, (3) antigen given I.V. two days after exposure to microwave, and (4) controls, not exposed (Table IX).

Blood specimens were drawn on all animals daily after sheep RBC was given. The serum concentration of anti-sheep RBC hemolysins on these specimens was determined. For each group the following data are given - the peak titer, i.e. the number of days after antigenic stimulation when the maximum concentration of hemolysin was obtained for each animal, and the antibody half-life, i.e. the number of days required for the serum concentration to fall to 50 per cent of the maximum concentration in a given animal.

The peak titer is reached somewhat later in exposed animals, and maximum titers decline to half-life values somewhat later than normal controls. This suggests that following microwave exposure animals either (a) take up antigen more slowly, (b) form antibodies more slowly after they have absorbed antigen, or (c) formed antibodies are eliminated or metabolized more slowly than in normal controls.

B. Effect on disappearance rate of passively transferred antibodies. Nine rabbits exposed to microwave for an average of 30 minutes were given infusions of homologous sera, containing known concentrations of anti-sheep RBC hemolysins. The serum concentration of this antibody was then determined 10 minutes after infusion and once daily thereafter. Two rabbits, that had not been exposed to microwaves, were similarly given passive transfer of antibodies. The normal for the half-life of this particular antibody is known to average 2.5 days for one component and 3.5 days for the other (Table X).

It appears that passively transferred antibodies are more rapidly eliminated or metabolized in microwave exposed rabbits than in normal controls. This suggests that the difference in response of these animals to primary antigenic stimulation is probably due either to slow uptake of antigen or slow antibody production.

Observations on the Character of the Thermal Response Specific comment on the nature of the thermal response has been given in the first portion of this paper, describing specific periods of (a) initial heating, (b) thermal regulation, and (c) thermal breakdown leading to death or (d) recovery. In studying the behavior of the animal following serial exposure particular interest was directed toward the possibility of an additional factor - that of accommodation or training. Fig. 6 shows the typical reaction of a dog following 11 exposures for a total of 19.5 hours.

Nine exposures later over a total elapsed time of 14 days (total 51.25 hrs.) a definite accommodation had occurred.

Although the effect of barbiturates, tranquilizers and narcotics will be discussed in the accompanying comparative paper, it was decided to test the effect of chlorpromazine on the temperature center on this animal. (It must be added that this reported reaction is a typical example and has been noted in other dogs.) As noted in Fig. 6, the reaction to chlorpromazine results in a return of the temperature response to the pre-accommodation level.

Subsequent reactions to non-medicated exposure were surprising. While the chlorpromazine reaction occurred with the 21st exposure, even at the 23rd the temperature levels continued to be above that reached at maximum accommodation. The same reaction continued to be true, additional exposures being required to bring the temperature reaction to maximum accommodation levels. Repeat chlorpromazine or barbiturate (pentobarbital) action produce similar loss of this induced physiological memory. This recalls the controversial French theory of Mneme (physiologic memory) of the middle of the last century. There is little question that the temperature center is the repository for this memory inasmuch as the drugs possessing most effective response have major pharmacologic actions in the thermoregulatory area. It will be of interest if respiratory depressants (morphine), anti-vomiting agents and the like should have similar action. Such would suggest a multiple action of a single center rather than multiple central nervous control areas. Future experiments are being directed toward such studies.

Response to Repeat Short Exposures In order to evaluate the compensatory mechanism of temperature regulation in the dog, repeated exposures at 165 mw/cm^2 were carried out over a period of 5 hours. Results are shown in Fig. 7. The heating was cycled between 102 and 106° F. For the first three hours the animal behaved according to a fixed pattern with the time required for cooling to specified levels being approximately 50% that necessary for heating. After the third hour a gradual transition takes place with prolongation of the time required for cooling. The accompanying graph does not demonstrate this in a conspicuous fashion but if a divider is used on the chart, results toward the five hour time become striking. A series of studies is now being carried out toward characterization of this response in terms of thermal breakdown. Longer and higher initial exposures with maintenance of equilibrium produce the change much more rapidly than do small repeated events.

An excellent possible use of this technique is as a method of measurement of sensitive species and/or evaluation of possible circulatory abnormalities interfering with heat loss. An early study is being directed toward evaluation of the temperature sensitivity in

the radiation survivor, the rat and rabbit. Such studies will do much toward clarification of presently known mechanisms of thermal regulation.

DISCUSSION In this investigation of the nature of response to the potential hazard of microwave irradiation, care has been taken toward standardization of the animal prior to exposure, radiation under ideal experimental conditions and careful observation of post-exposure sequela. In all instances sufficient animals are used to characterize the responses which to date have been remarkably consistent and easily repeated. The very standard response and the ease of reproducibility from the outset indicates that thermal response leading toward failure of thermoregulatory center is the major hazard. Observations on animals receiving ionizing radiations suggest other factors which probably relate to vascular defect but remain unknown.

It is well known that lethality from thermal over exposure occurs as the result of failure of the thermoregulatory mechanism and may have its origin either peripherally or from primary central nervous system damage. Prior to death with rapidly mounting fever, a definite gradual hemoconcentration with resultant clinical picture of shock occurs. Animals which are adequately hydrated and fed prior to exposure reach this level with surprising uniformity.

To date - due to lack of an available balance of necessary sensitivity, experiments on fluid dose, which in the dog is primarily effected through the respiratory passages have not been attempted. It is planned to accurately determine this dose, maintain the loss by oral administration during the exposure and post-exposure periods. Other experiments on pre-exposure over-hydration, addition of electrolytes and the like should next be considered.

It is probable that study of the radiation survivors may be the most rewarding. Although the probable possession of a vascular defect as indicated by the reported experiments can explain most findings, a possible change in the central nervous system (heat regulatory areas) exists. It is not unlikely that the changes in temperature response which relate to age may be due to a mixed vascular-central nervous system reaction.

Other observations reported likewise show promise. The stress reaction on the adrenal and the reheating experiments could be modified toward specific clinical testing. The nature of temperature response to external factors should be compared with response to internally induced physical, chemical, or biological factors. It is necessary to assay the nature of the central nervous system response, the nature of the accommodation reaction and related neurophysiological activity of secondary nature.

To date no evidence of a non-thermal response or of injurious factors related to acute microwave exposure has been produced. All reactions

are either thermal in type or related to thermally induced injury. The mechanisms of production of such injury, the secondary manifestation and appropriate prophylactic and therapeutic measures demand urgent consideration.

SUMMARY Studies are reported on dogs exposed to 2880 megacycle pulsed electromagnetic irradiation from an AN/MP3-14 search radar adapted for biologic experimentation. Single and repeated exposures at energy levels of 100 and 165 mw/cm² for times up to 6 hours were carried out. Clinical reactions as observed in the animals are noted. Various physiological reactions are described. Characterization of thermal response into periods of initial heating, thermal equilibrium, thermal breakdown and death or recovery is made. Changes in hemodynamics as measured by blood assay are included with specific descriptions of behavior of cellular elements. Observations of eosinophil appearance and disappearance suggest a stress response at intermediate power (100 mw/cm²); with high power level, an adrenal exhaustion phenomenon may occur. Minor changes in fat absorption as measured by I-131 labelled fat are noted. Shortening of red cell life occurs in some animals at higher exposures. A response of injury in thyroid function is commented upon. Alteration of antibody production occurs. A prolongation of antibody life is observed. Passively transferred antibodies may be metabolized more rapidly in exposed animals. Studies on repeated exposure indicate development of adaptation or accommodation responses suggestive of physiologic memory. This response is completely wiped out by chlorpromazine therapy. On short repeated exposures, a possible development of thermal and vascular fatigue gradually occurs. Studies on serial exposures to ionizing and microwave energies are discussed. The gross and pathologic changes observed will be given in a future discussion.

TABLE I - RESPONSE OF DOGS TO MICROWAVE EXPOSURE - IMMEDIATE

| Power Level | 100 mw/cm ² | | 165 mw/cm ² | | | |
|------------------|------------------------|----------------|------------------------|-------------------|----------------|---------|
| | 6 Hours | | 2 Hours | | | 3 Hours |
| Previous History | Normal | Co-60 400 r | Normal | Co-60 Survivor | Co-60 400 r | Normal |
| Number of dogs | 10 | 5 | 10 | 8 | 6 | 7 |
| Temp. change ° F | + 3.0 | + 1.4 | + 3.4 | + 3.4 | + 3.8 | + 5.1 |
| WBC% (Total) | + 3.1 | + 3.8 | -18.2 | - 15.8 | -16.7 | +22.5 |
| Polys | +25.7 | | +14.0 | | | +22.2 |
| Lymphs | -49.4 | | -21.0 | | | -27.1 |
| Hct. | + 3.0 | - 5.0 | + 3.1 | + 9.9 | - 6.0 | +14.5 |
| Glucose %change | | | - 6.8 | + 4.3 | | |
| Hematuria | 2 | 3 | 0 | 3 | 1 | 1 |
| Akinesia | 2 | 1 | 3 | 0 | 2 | 6 |
| Death | 1 | 0 | 1 | 1 | 0 | 1 |

TABLE II - RESPONSE OF DOGS TO MICROWAVE EXPOSURE - DELAYED 1 WEEK

| Power Level | 100 mw/cm ² | | 165 mw/cm ² | | | | Control |
|-------------------------|------------------------|----------------|------------------------|-------------------|----------------|---------|----------------|
| Duration of Exposure | 6 Hours | | 2 Hours | | | 3 Hours | |
| Previous History | Normal | Co-60 400 r | Normal | Co-60 Survivor | Co-60 400 r | Normal | Co-60 400 r |
| Number of Dogs | 10 | 5 | 10 | 8 | 6 | 7 | 6 |
| WBC % (Total) | + 3.2 | -78.5 | - 1.8 | +12.4 | -86.1 | -23.1 | -68.2 |
| Polys | | | | | | +15.6 | |
| Lymphs | | | | | | -21.7 | |
| Hct. % change | - 3.1 | -19.7 | - 4.5 | - 5.5 | -21.0 | - 3.9 | -10.1 |
| Burns | 1 | | 0 | 3 | 1 | 1 | |
| Pain Abdominal or Joint | 1 | | 0 | 1 | 0 | 1 | |
| Weight change | 0 | | 0 | | | 0 | |
| Death | 0 | 2 | 0 | 1 | 6 | 0 | 1 |

TABLE III - HEMATOLOGIC CHANGES IN DOGS EXPOSED TO MICROWAVES

| Time After Exposure | % Change Hematocrit | | | | |
|---------------------|---------------------|---------------------------------|---------------------------------|---|---------------------------------|
| | Sham | 100 mw/cm ² 6 hr. | 165 mw/cm ² 2 hr. | Survivor 165 mw/cm ² 2 hr. | 165 mw/cm ² 3 hr. |
| 0 | - 4.6 | ♦ 3.0 | + 3.1 | + 9.9 | +14.5 |
| 15 min. | - 8.4 | ♦ 3.3 | + 0.6 | + 4.4 | +14.0 |
| 24 hr. | -10.2 | - 3.9 | - 7.5 | + 0.9 | + 2.3 |
| 28 hr. | --- | - 2.7 | - 5.7 | - 1.7 | +10.7 |
| 1 wk. | - 4.2 | - 3.1 | - 4.5 | - 5.5 | - 3.9 |

TABLE IV - HEMATOLOGIC CHANGES IN DOGS EXPOSED TO MICROWAVES

| Time After Exposure | % Change WBC | | | | |
|---------------------|--------------|---------------------------------|---------------------------------|--|---------------------------------|
| | Sham | 100 mw/cm ² 6 hr. | 165 mw/cm ² 2 hr. | Survivors 165 mw/cm ² 2 hr. | 165 mw/cm ² 3 hr. |
| 0 | - 3.7 | + 3.1 | -18.2 | -15.8 | +22.5 |
| 15 min. | --- | --- | --- | --- | --- |
| 24 hr. | +10.2 | +80.3 | +15.3 | +77.3 | +96.3 |
| 28 hr. | --- | +10.4 | - 3.1 | +28.4 | +30.6 |
| 1 wk. | + 5.1 | + 3.2 | - 1.8 | +12.4 | -23.1 |

TABLE V - HEMATOLOGICAL STUDIES ON DOGS EXPOSED TO MICROWAVES

2800 Mc

| Exposure mw/cm ² | Time (min) | Dog | Clotting Time (Mins) | | | | | | Clot Retraction | |
|--------------------------------------|---------------|------|----------------------|--------|--------|---------------|--------|--------|-----------------|---------------|
| | | | Pre-Exposure | | | Post-Exposure | | | Pre-Exposure | Post-Exposure |
| | | | Tube 1 | Tube 2 | Tube 3 | Tube 1 | Tube 2 | Tube 3 | | |
| 100 | 360 | 4303 | 4 | 6 | 8 | 4 | 7 | 8 | <1 Hr. | <1 Hr. |
| 165 | 70 | 4185 | 5 | 6 | 6 | 4 | 6 | 7 | -- | -- |
| 165 | 110 | 4207 | 5 | 7 | 9 | 3 | 5 | 7 | <1 Hr. | <1 Hr. |
| 165 | 110 | 4369 | 3 | 5 | 8 | 3 | 5 | 7 | <1 Hr. | <1 Hr. |
| 165 | 120 | 4456 | 4 | 4 | 4 | 5 | 4 | 5 | -- | -- |
| 165 | 120 | 4154 | 5 | 6 | 7 | 5 | 5 | 7 | -- | -- |
| 15 Previous for Total of 34½ Hrs. | | 4432 | 4 | 6 | 9 | 3 | 5 | 7 | -- | -- |
| 14 Previous for Total of 27½ Hrs. | | 4370 | 2 | 4 | 6 | 5 | 7 | 8 | -- | -- |
| 165 | 180 | 4367 | 5 | 5 | 6 | 3 | 4 | 3 | -- | -- |
| 165 | 200 | 4380 | 3 | 6 | 9 | 3 | 4 | 5 | <1 Hr. | <1 Hr. |
| 165 | 226 | 4427 | 4 | 4 | 4 | 3 | 3 | 3 | -- | -- |

TABLE VI - HEMATOLOGICAL STUDIES ON DOGS EXPOSED TO MICROWAVES

2800 Mc

| Exposure mw/cm ² | Time (min) | Dog | Red Cell Fragility | | Hct. | | Viscosity Blood Sugar (25°C) mg/100ml | | NPN mg/100ml | | | | | |
|--------------------------------|---------------|------|-------------------------|---------------------------|-------|-------|--|------|-----------------|------|------|------|-------|-------|
| | | | Pre-Exposure Initial | Post-Exposure Complete | Pre | Post | Pre | Post | Pre | Post | | | | |
| 100 | 360 | 4303 | 0.48 | 0.32 | <0.48 | 0.32 | 49.0 | 44.5 | --- | --- | --- | --- | | |
| 165 | 110 | 4207 | 0.48 | 0.32 | 0.48 | 0.32 | 44.0 | 46.5 | --- | --- | --- | --- | | |
| 165 | 110 | 4369 | 0.48 | 0.36 | 0.48 | 0.32 | 44.5 | 51.5 | --- | --- | --- | --- | | |
| 165 | 120 | 4456 | <0.48 | <0.32 | <0.48 | <0.32 | 38.0 | 43.0 | 4.9 | 6.0 | 80.5 | 83.9 | 36.75 | 36.75 |
| 165 | 180 | 4367 | <0.48 | 0.32 | <0.48 | 0.32 | 36.0 | 40.5 | 4.8 | 5.3 | 81.5 | 80.5 | 36.75 | 42.4 |

TABLE VII - MEAN HEMATOLOGICAL CHANGES IN NORMAL DOGS EXPOSED TO MICROWAVES

| Mc | Field Intensity | Exposure (mins) | No. Of Dogs | Determinations | WBC | Polys | Lymphocytes | Eosinophiles |
|-------|------------------------|-----------------|-------------|----------------|--------|--------|-------------|--------------|
| 2800 | 100 mw/cm ² | 360 | 12 | Initial | 12,710 | 8,875 | 3,092 | 558 |
| ----- | --- | --- | 11 | Post | 12,620 | 10,580 | 1,490 | 220 |
| | | | | 1 Day P | 15,090 | 10,680 | 3,174 | 722 |
| 2800 | 165 mw/cm ² | (30-180) | 14 | Initial | 12,820 | 8,566 | 3,223 | 617 |
| ----- | --- | --- | 6 | Post | 13,240 | 10,186 | 2,228 | 617 |
| | | | | 1 Day P | 15,320 | 11,665 | 2,951 | 293 |
| 200 | 165 mw/cm ² | (140-480) | 4 | Initial | 9,900 | 6,414 | 2,482 | 808 |
| | | | | Post | 14,310 | 11,817 | 1,698 | 267 |

TABLE VIII - RESPONSE OF DOGS EXPOSED TO MICROWAVES FOUR DAYS AFTER IONIZING RADIATION

| Ionizing Radiation (Co60) | Microwaves 2800 Mc Pulsed | Duration (Minutes) | Number of Dogs | Temp. Change (°F) | WBC x 10 ³ | | HCT. | | % Mortality 30 Days |
|---------------------------------|---------------------------------|-----------------------|----------------------|-------------------------|-----------------------|-------|------|------|------------------------|
| | | | | | Pre | Post | Pre | Post | |
| 400 r | -- | --- | 11 | --- | 6.00 | -- | 46.0 | -- | 36 |
| -- | 100 mw/cm ² | 360 | 10 | +2.40 | 13.24 | 13.74 | 48.0 | 49.5 | 10 |
| 400 r | 100 mw/cm ² | 360 | 10 | +2.30 | 5.43 | 5.22 | 46.0 | 47.0 | 30 |
| -- | 165 mw/cm ² | 120 | 15 | +3.10 | 15.23 | 12.84 | 46.0 | 49.5 | 7 |
| 400 r | 165 mw/cm ² | 120 | 10 | +3.23 | 5.25 | 4.27 | 45.0 | 48.0 | 70 |

TABLE IX - EFFECT OF EXPOSURE ON PRIMARY ANTIGEN STIMULUS (RABBITS)

(2800 Mc at 100 mw/cm²)

| Time of Administration | Number of Animals | Peak Titer (Days) | Half-Life of Antibody (Days) |
|---------------------------|-------------------|-------------------|------------------------------|
| 2 Days Prior to Exposure | 9 | 7.6 | 6.3 |
| 4 Hours Prior to Exposure | 9 | 9.0 | 7.7 |
| 2 Days After Exposure | 8 | 9.4 | 7.9 |
| Controls | 7 | 7.8 | 3.9 |

Antigen dosage - 1.0 ml of 10% sheep RBC in Saline.

TABLE X - EFFECT OF EXPOSURE ON DISAPPEARANCE RATE OF PASSIVELY TRANSFERRED ANTIBODIES

(2800 Mc at 100 mw/cm²)

| Group | Number of Animals | Half-Life (Days) |
|----------|-------------------|------------------|
| Exposed | 9 | 1.8 |
| Controls | 8 | 3.5 |

Range of dosage of passively transferred antibodies - 30,000 - 100,000 Units.

Antibody Unit - Arbitrarily defined as volume in ml of a serum which hemolyses 50% of a standard 2.5% saline suspension sheep RBC.

THERMAL RESPONSE OF THE DOG
 EXPOSED TO DIFFERENT POWER LEVELS OF
 2800 Mc PULSED MICROWAVES

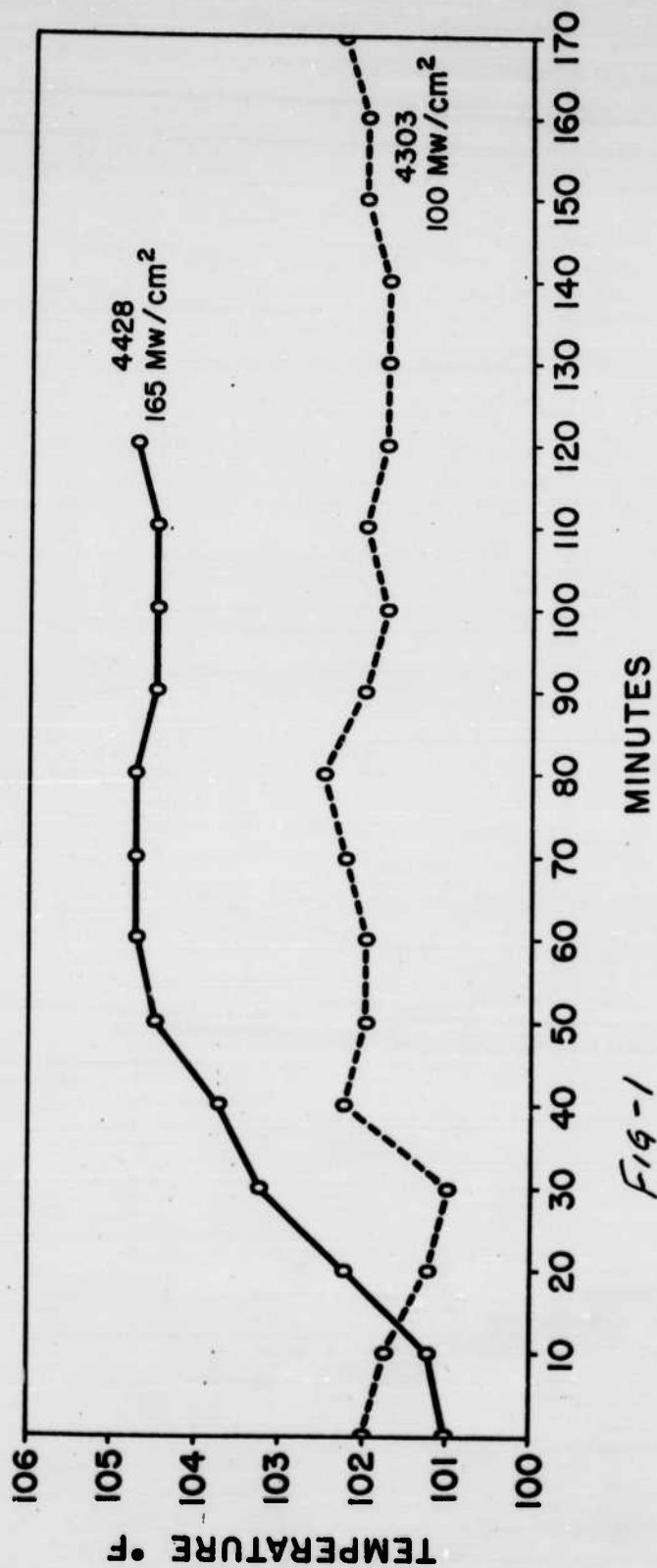
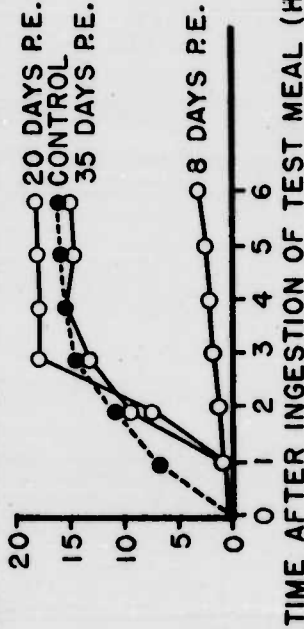


Fig-1

% OF THE I131 LABELED FAT TEST MEAL
RECOVERED IN BLOOD / TOTAL BLOOD

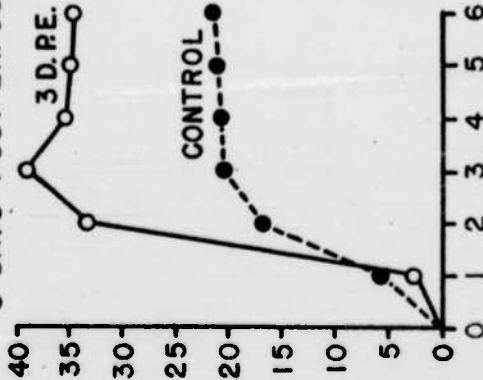
CHANGES IN FAT ABSORPTION PATTERN IN NORMAL DOG
AFTER EXPOSURE TO MICROWAVES

4369-AGE 2 YRS.-2 HRS. 165 MW/CM² MICROWAVES

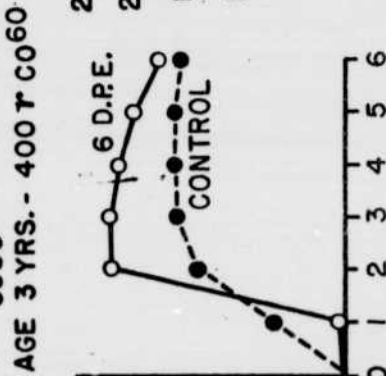


CHANGES IN FAT ABSORPTION PATTERN IN NORMAL DOGS
AFTER EXPOSURE TO Co⁶⁰

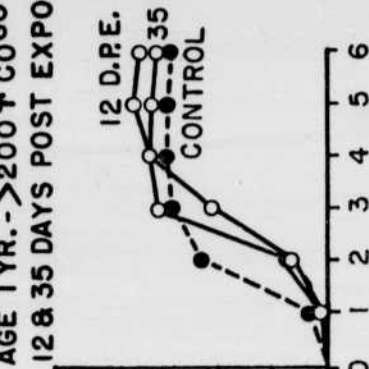
4247-AGE 3 YRS.- 400T Co⁶⁰
3 DAYS POST EXPOSURE



3933
6 DAYS POST EXPOSURE



4351
AGE 1 YR.->200 T Co⁶⁰



4351
AGE 1 YR.->200 T Co⁶⁰
12 & 35 DAYS POST EXPOSURE

Fig-2 TIME AFTER INGESTION (HOURS)

CHANGES IN FAT ABSORPTION PATTERN IN SURVIVOR DOGS
AFTER EXPOSURE TO MICROWAVES - 2880 MC

% OF THE I131 LABELED FAT TEST MEAL
RECOVERED IN BLOOD / TOTAL BLOOD VOLUME

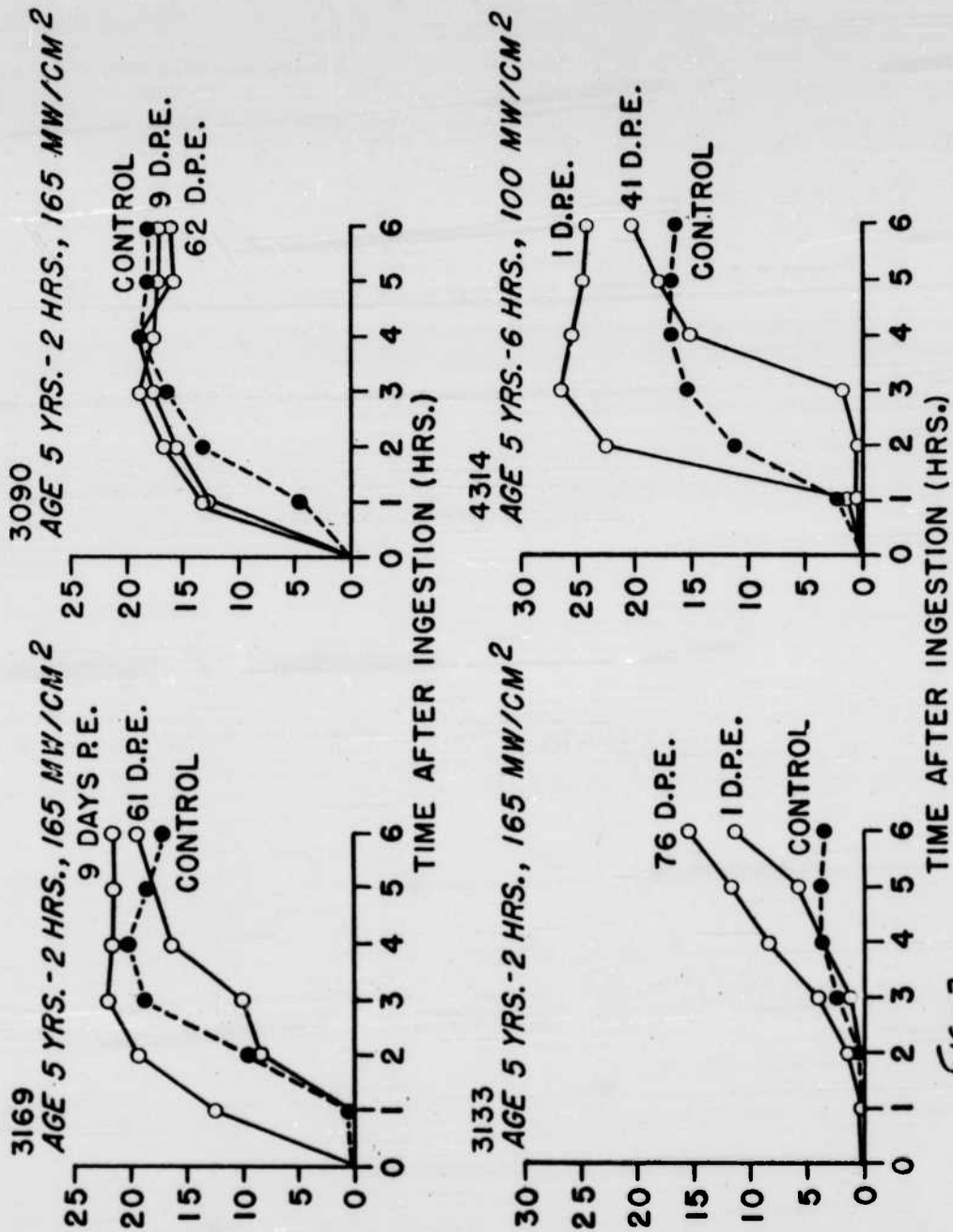


Fig-3

% OF THE I¹³¹ LABELED FAT TEST MEAL
RECOVERED IN BLOOD / TOTAL BLOOD VOLUME

CHANGES IN FAT ABSORPTION PATTERN IN NORMAL DOGS
AFTER EXPOSURE TO BOTH CO⁶⁰ AND MICROWAVES

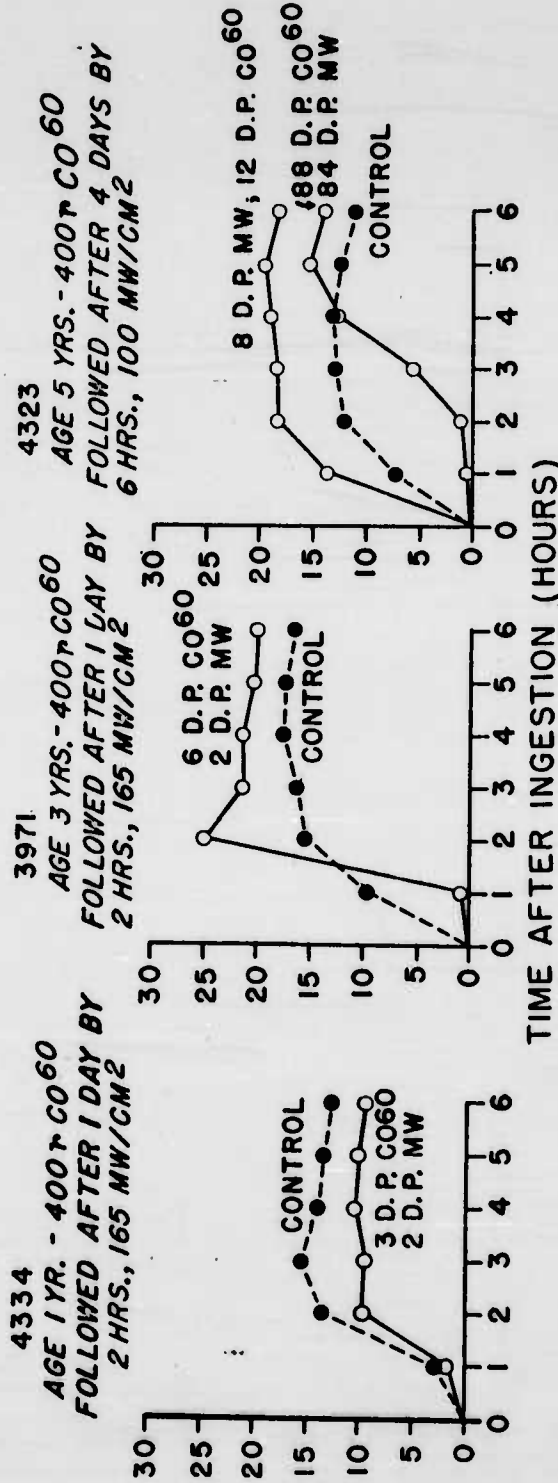


Fig-4

PERCENT DAILY RATE OF LOSS PER ML. RED CELLS
IN EXCESS OF THAT DUE TO NORMAL SENESCENCE

(APPARENT ELUTION RATE / ML. RED CELLS)

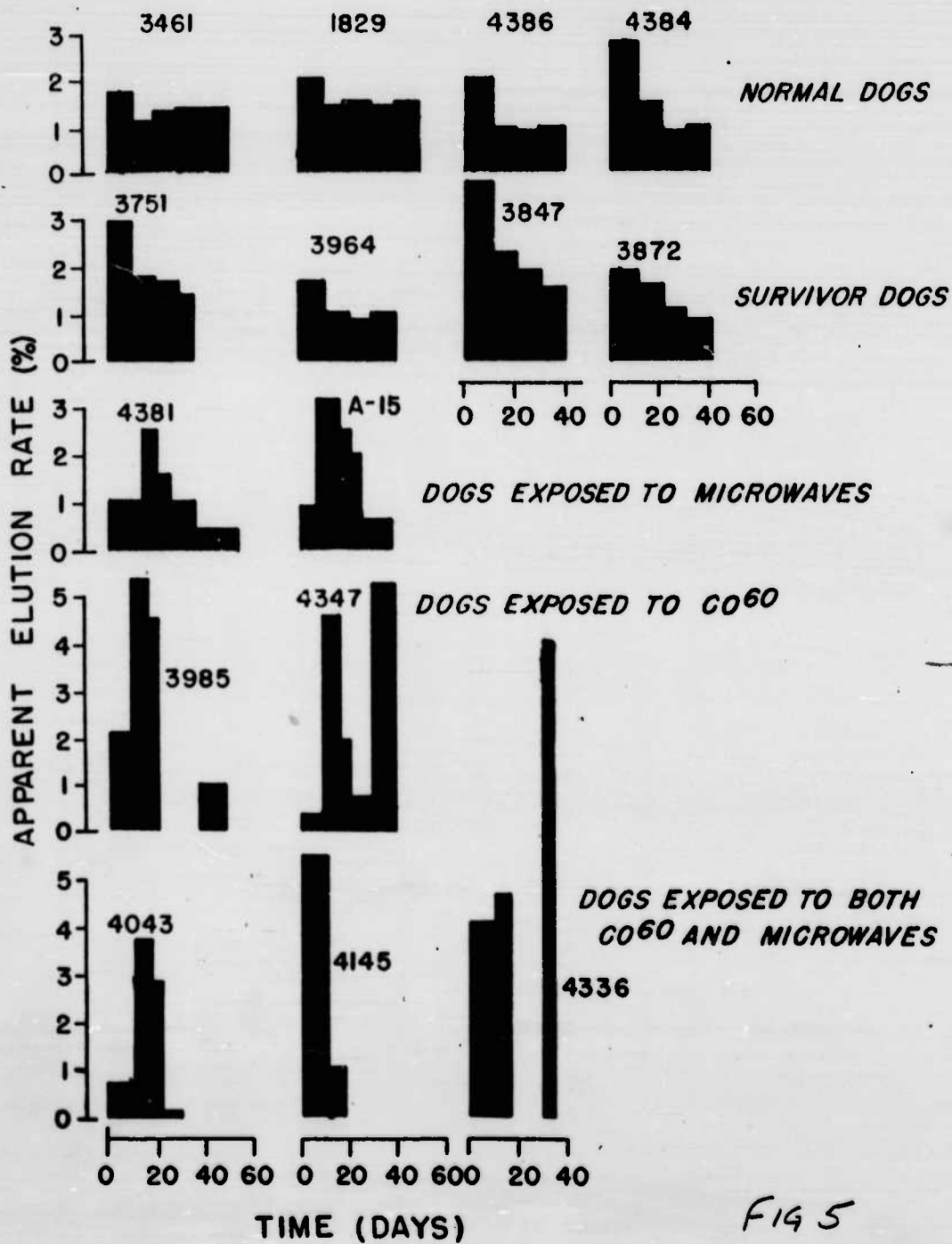


FIG 5

THERMAL RESPONSE TO DAILY EXPOSURES OF MICROWAVE

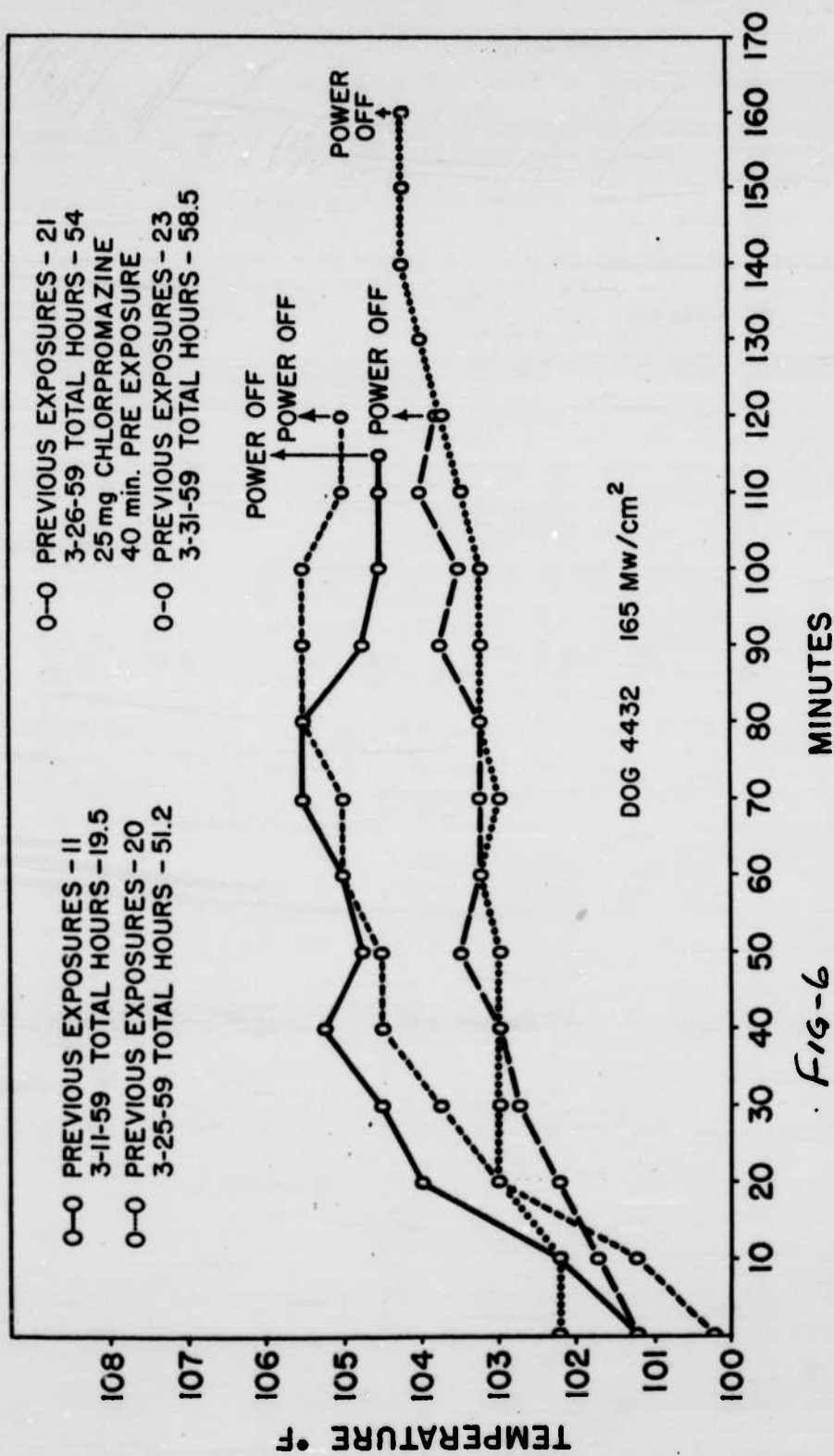
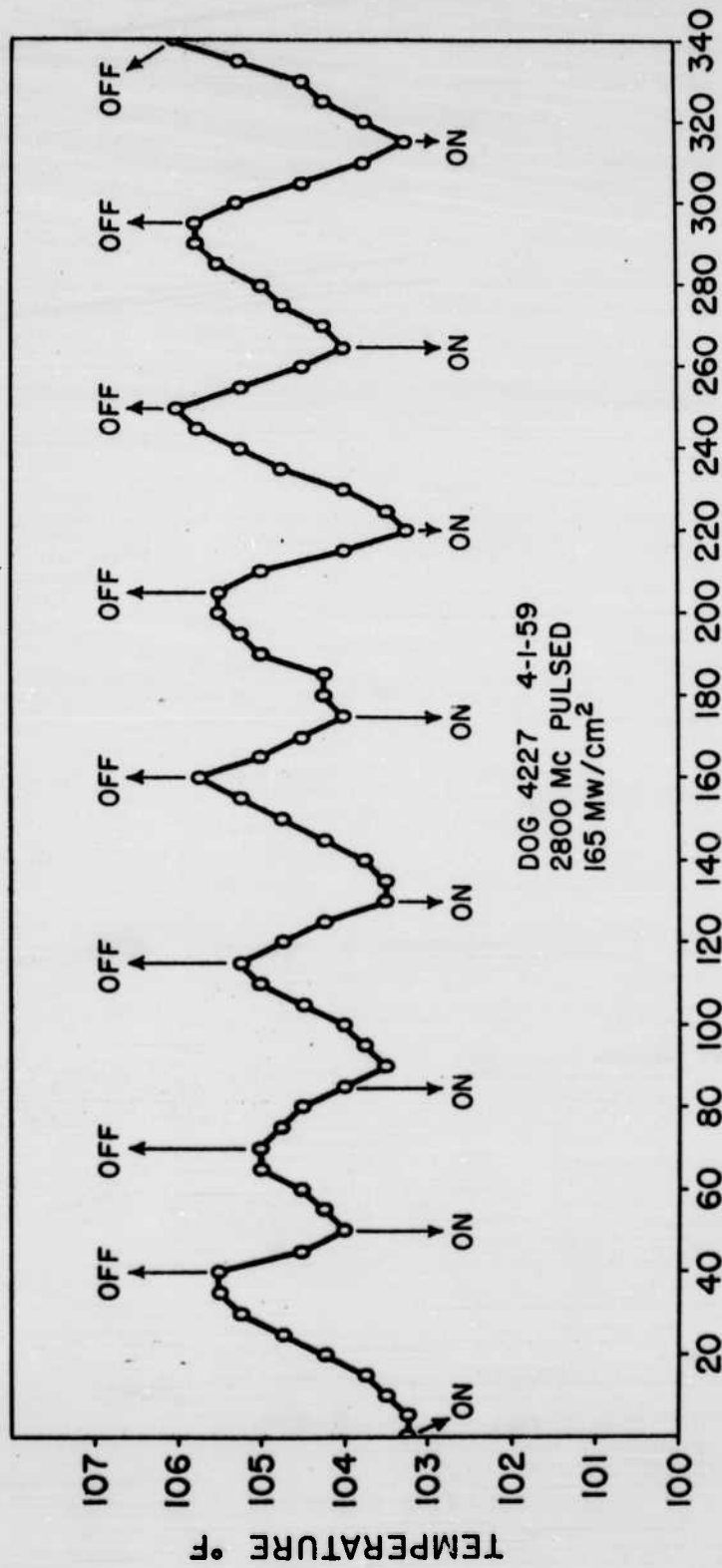


Fig-6

THE RESPONSE OF THE DOG TO
REPEATED SHORT MICROWAVE EXPOSURES

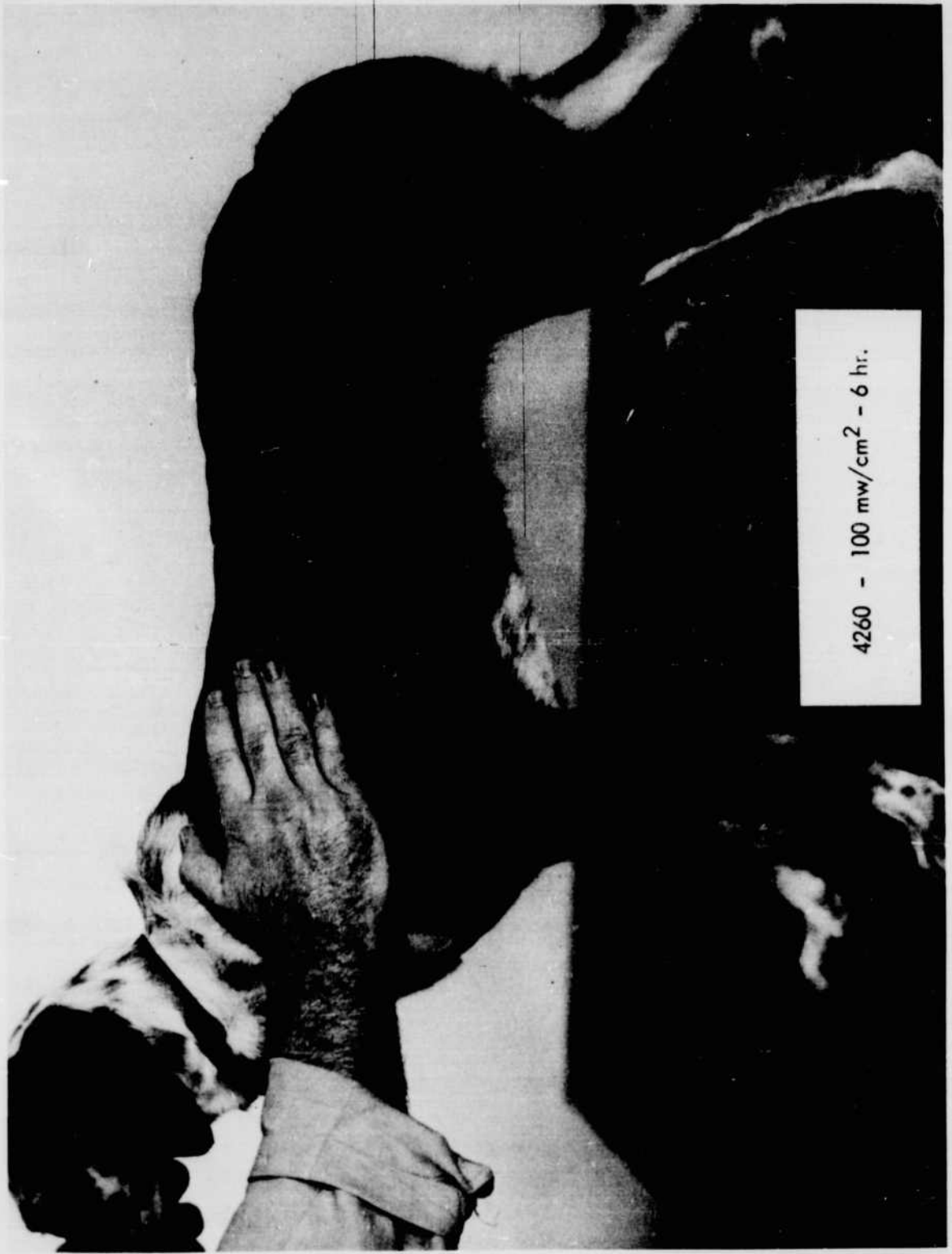


MINUTES

Fig. 7



Photograph 1 4260 Normal - 100 mw/cm² - 6 hours - right side.
Photographed 6 days after exposure.

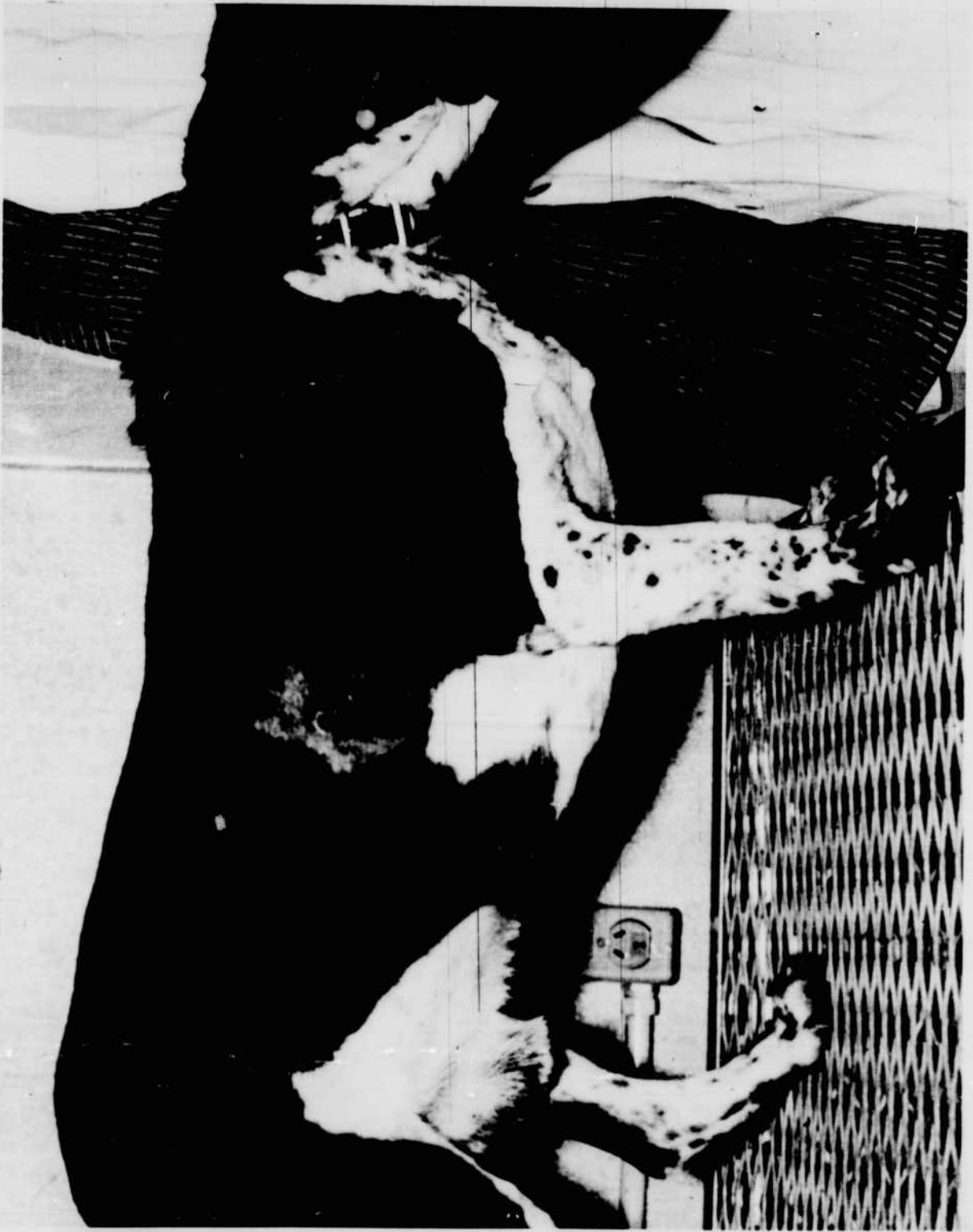


4260 - 100 mw/cm² - 6 hr. Photographed 6 days after exposure.

Photograph 2 4260



Photograph 3 4260 Normal - 100 mw/cm² - 6 hours - close up -
right side. Photographed 6 days after
exposure.

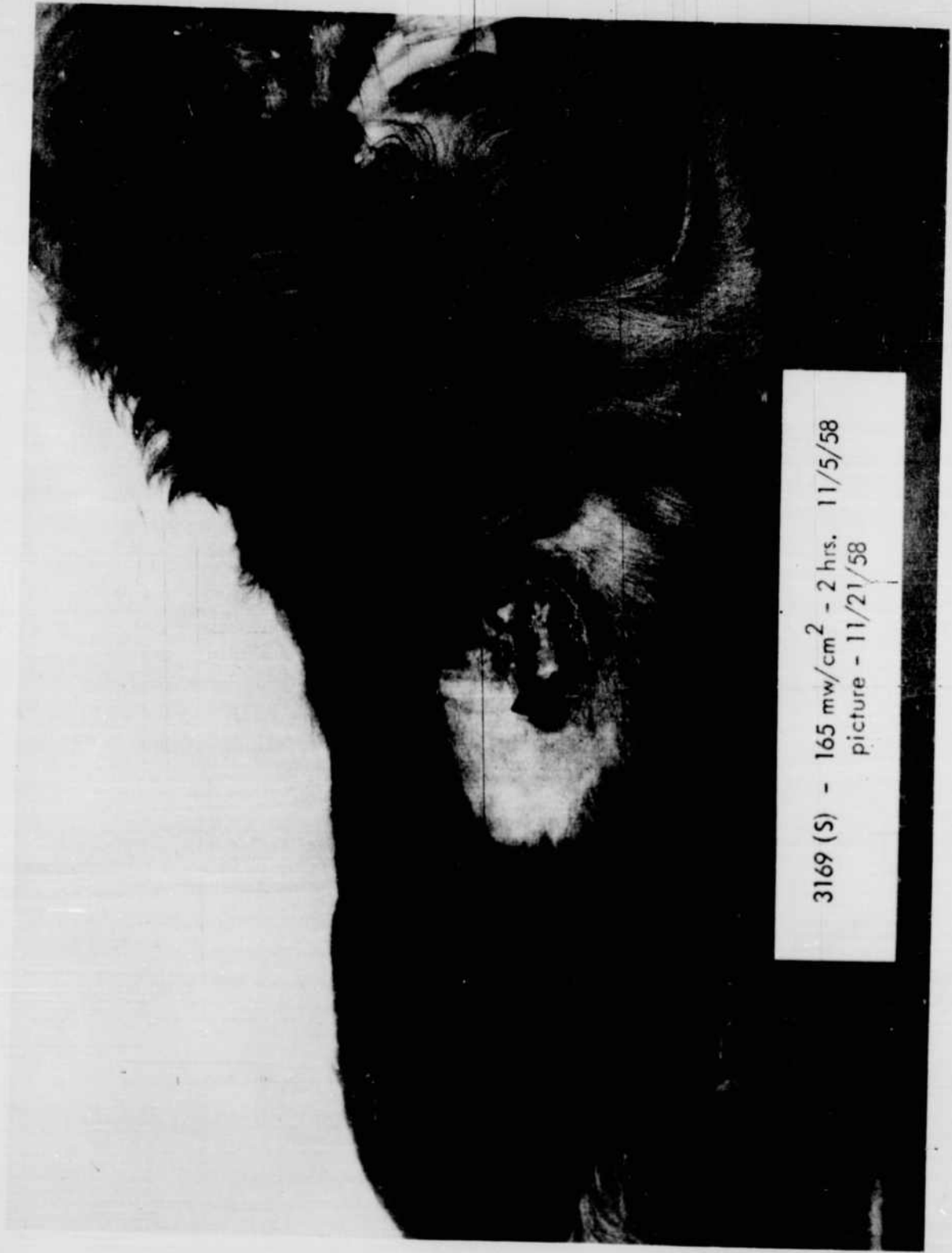


Photograph 4 4207 Normal - 165 mw/cm² - 2 hours - right side -
healing lesion 6 weeks after exposure.

275



Photograph 5 4207 Normal - 165 mw/cm² - 2 hours - left side -
healing lesion 6 weeks after exposure.



3169 (S) - 165 mw/cm² - 2 hrs. 11/5/58
picture - 11/21/58

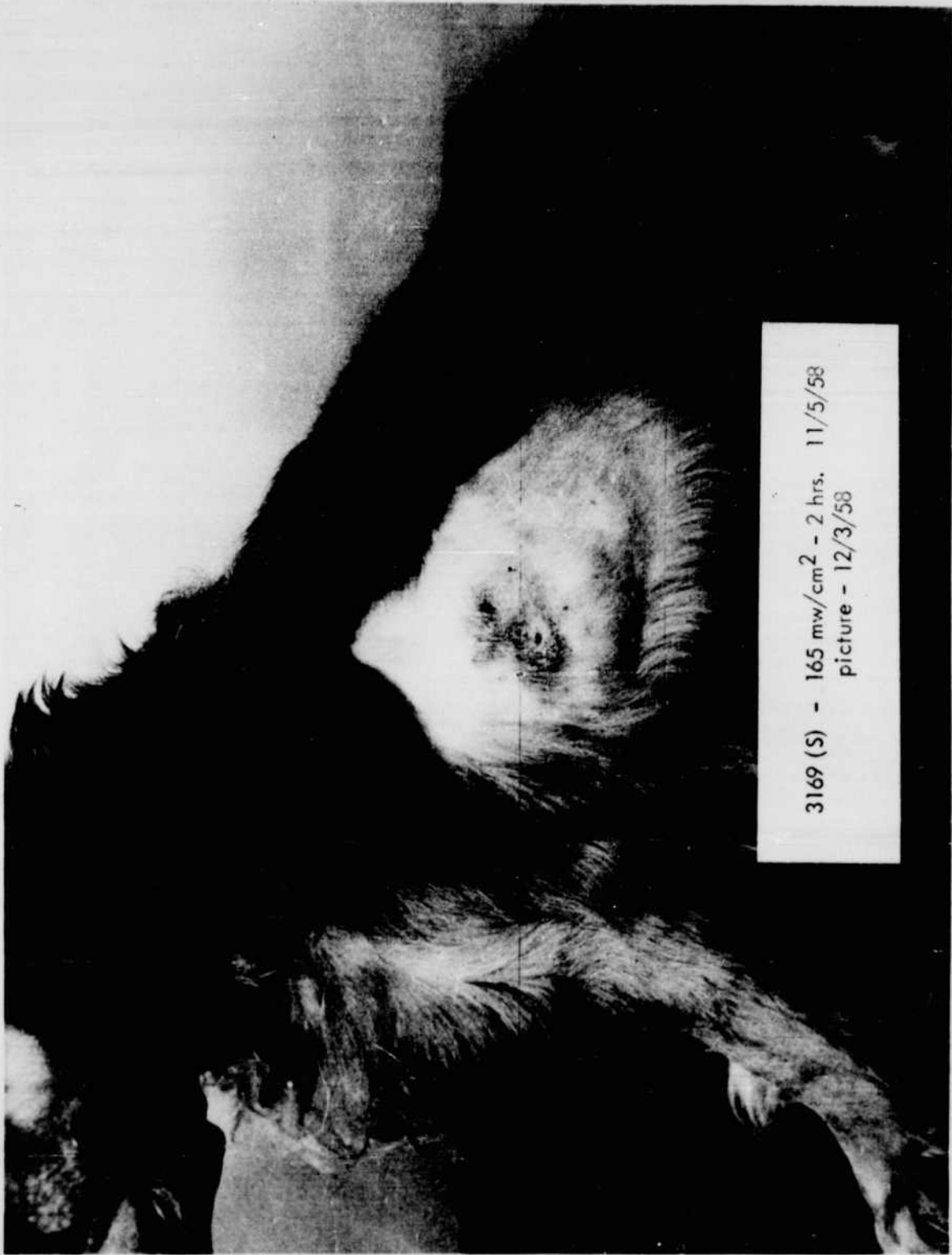
Photograph 6 3169 Survivor - 165 mw/cm² - 2 hours - right side -
2 weeks after exposure.



228:

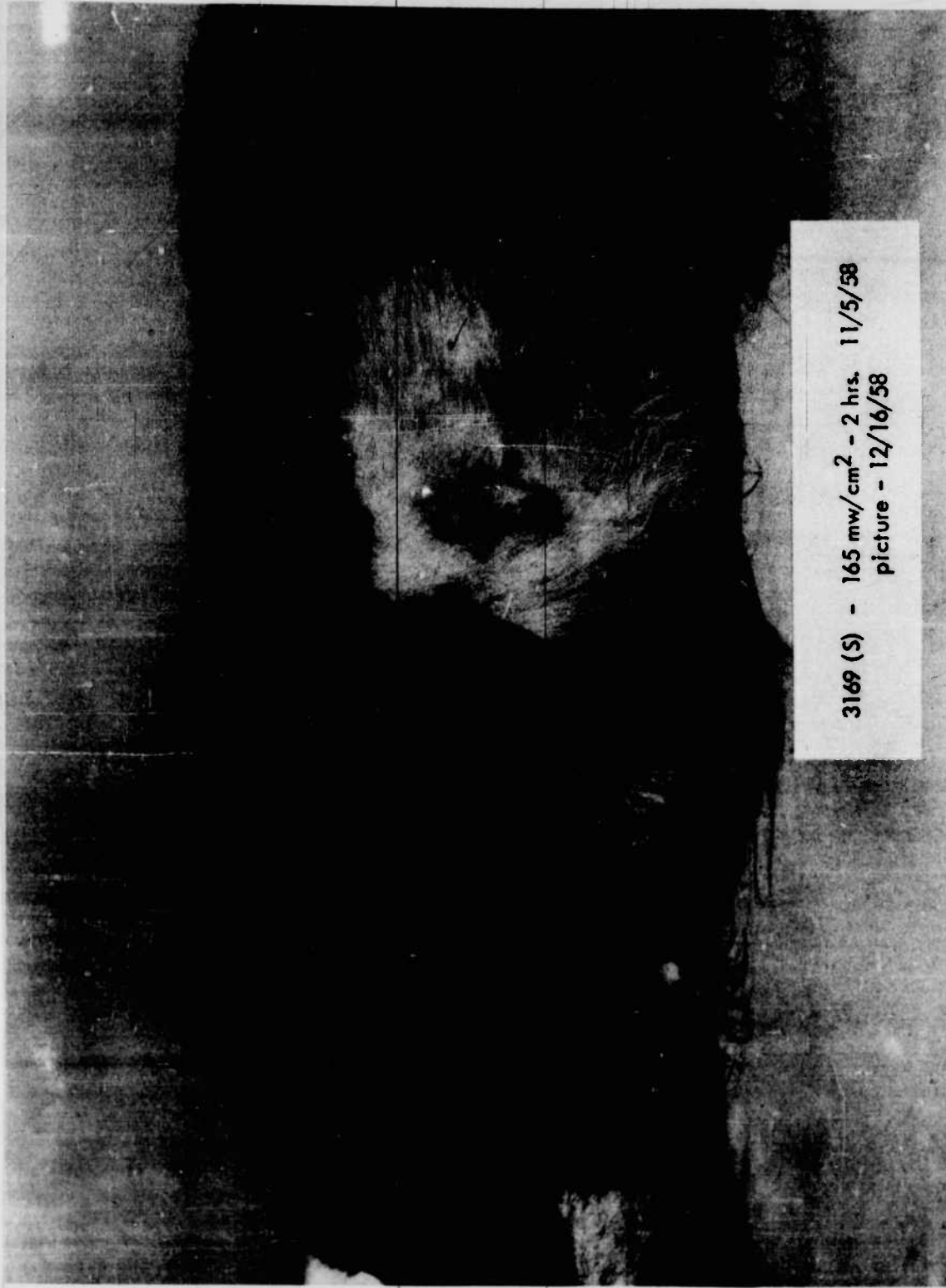
3169 (S) - 165 mw/cm² - 2 hrs. 11/5/58
picture - 11/21/58

Photograph 7 3169 Survivor - 165 mw/cm² - 2 hours - left side -
2 weeks after exposure.



3169 (S) - 165 mw/cm² - 2 hrs. 11/5/58
picture - 12/3/58

Photograph 3 3169 Survivor - 165 mw/cm² - 2 hours - left side -
4 weeks after exposure.



3169 (S) - 165 mw/cm² - 2 hrs. 11/5/58
picture - 12/16/58

Photograph 9 3169 Survivor - 165 mw/cm² - 2 hours - left side -
6 weeks after exposure.



Photograph 10 4227 Anaesthetized - 165 mw/cm² - 30 minutes -
front - 10 days after exposure.



232

Photograph 11 4227 Anaesthetized - 165 mw/cm² - 30 minutes -
flank - 10 days after exposure.



Photograph 12 4431 Anaesthetized - 165 mw/cm² - 30 minutes -
3 days after exposure.



234

Photograph 13 4431 Anaesthetized - 165 mw/cm² - 30 minutes -
3 days after exposure.



Photograph 14 4431 Anaesthetized - 165 mw/cm² - 30 minutes
7 days after exposure.



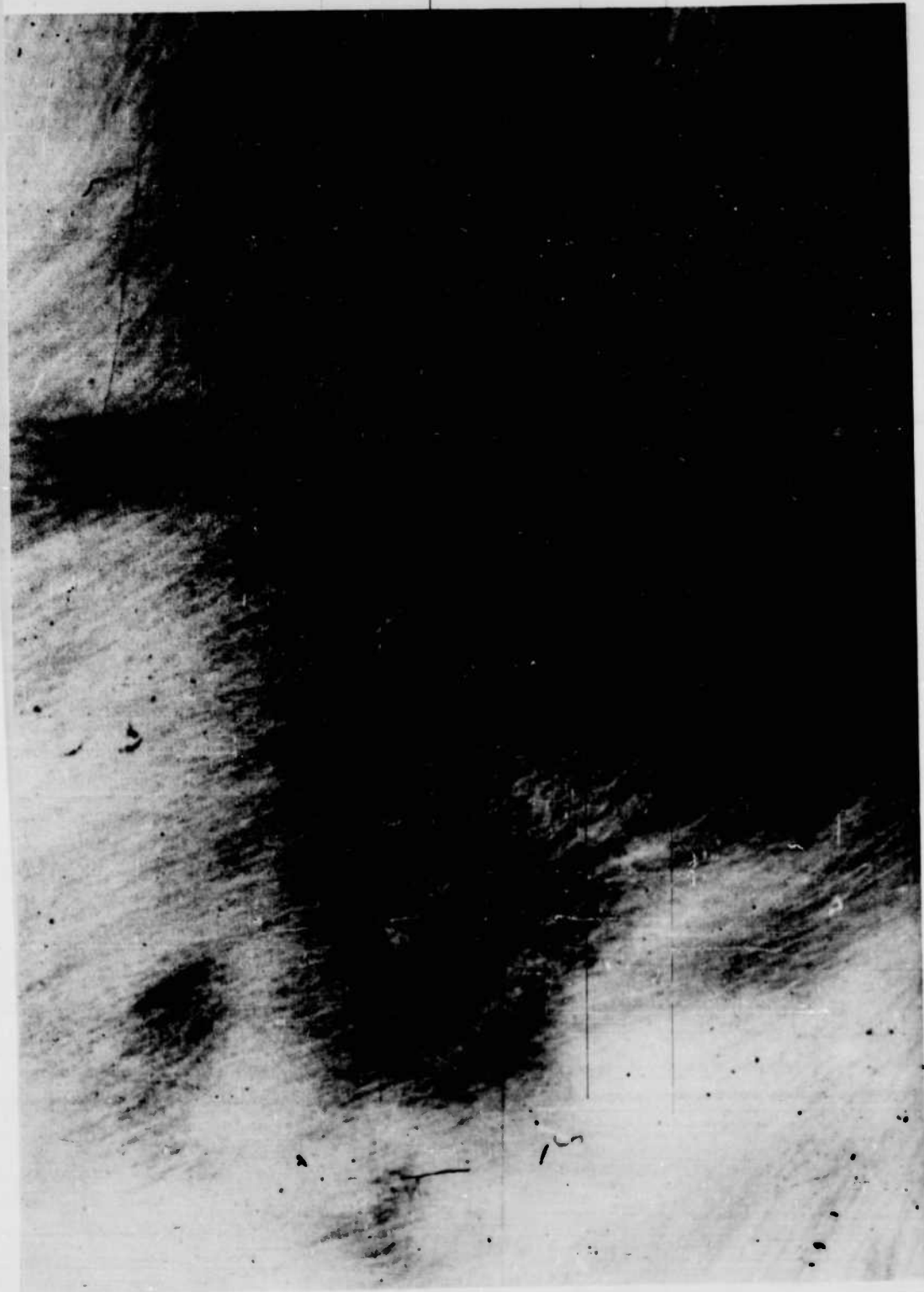
Photograph 15 4431 Anaesthetized - 165 mw/cm² - 30 minutes -
10 days after exposure.

236



237

Photograph 16 4431 Anaesthetized - 165 mw/cm² - 30 minutes -
14 days after exposure.



Exposed to 165 mw/cm^2 - burn (right) occurred and healed within 3 weeks. Six weeks later dog re-exposed. Burn reoccurred in original site and second burn (left) appeared. Photographed 2 days after second exposure.

Photograph 17 A-15

EYE STUDY SURVEY

Dr. Lee A. Clark, Jr.
Rome Air Development Center

Summary

Detail slit lamp examinations were performed on 23 people who work with microwave equipment at Griffiss AFB, N. Y. Fifty-two people were used as a control group. The survey, being on a limited number of individuals, cannot give conclusive information but the data presented is suggestive of increased incidence of abnormal findings in the suspect group.

When Colonel Knauf was Base Surgeon at Griffiss AFB, site of the Rome Air Development Center, his interest in gathering basic information on the biological effects of microwaves was paralleled by his desire to fulfill his responsibilities in the field of preventive medicine. He saw early the need of survey studies on personnel using microwave equipment. In the literature at that time there were reports on the cataractogenic effect of microwave; therefore, he had a number of people at Griffiss who worked with microwaves undergo detail eye examinations. He felt that the results of those examinations were significant, but admitted there were no controls, thus preventing scientific evaluation of his suspicion.

In order to gather more valid data he arranged for Major Frank W. Jones, an ophthalmologist, to examine 23 people who worked with microwave equipment. This examination included the use of the slit lamp. Fifty-two people were used as controls; they were secretaries, bookkeepers, etc. When Major Jones examined these people he did not know which were suspects and which were controls. I shall present the data from Dr. Jones' survey.

There is one point I must present as a preface to the referenced survey. Microwave generators also produce ionizing radiation which is known to be cataractogenic. I cannot say how much or to what type ionizing energy these people were exposed.

I will not attempt to review the literature on microwave cataracts, but will only say that most of the opacities experimentally produced have been in the posterior cortex of the lens. However, this may be because most of the experimental studies have been at one frequency - 2450 mc.

The lens is about 1.5 cm in diameter and 3.5 mm thick; it is biconvex. It is topographically divided into the capsule, cortex, adult nucleus, and fetal nucleus. If one studies the lens systematically, from the anterior to the posterior, he encounters in this order the anterior capsule, anterior cortex, anterior adult nucleus, fetal nucleus, posterior adult nucleus, posterior cortex, and posterior capsule. The slit lamp is an instrument that enables one to study the lens under magnification, layer by layer.

The findings reported do not significantly interfere with vision; they are to be distinguished from cataracts, which actually hinder the function of the eye. Each eye is reported separately. There were 52 control persons and 23 suspects given a total of 104 control eyes and 45 suspect eyes (one man had an old eye injury).

Figure 1 is a tabulation of the entire data. I have divided it into seven parts for discussion.

Figure 2 shows the findings in the anterior capsule of the lens. There seems to be in the younger two age groups an increased incidence of pigmented opacities. In the older groups the findings are too infrequent for comment. This is true throughout for non-pigmented opacities.

In Figure 3 are the anterior cortex findings. Here, there is an increased percentage of positive findings of nearly every type in the suspect group.

Figure 4 shows the incidence of opacities and cloudiness in the anterior adult nucleus; they are somewhat less prevalent in the suspect group.

In Figure 5 the findings by Dr. Jones in the fetal nucleus are given. Considering the entire group, the incidence of all type abnormalities is low, excepting probably the non-pigmented opacities in the control group in the 4th decade, which is 40.9%.

The posterior adult nucleus findings show essentially no difference between the suspect and control group. Figure 6 shows the data.

Post cortex observations are given in Figure 7. Here, as stated earlier, is where in experimental animals the effects have been most often seen. The incidence of opacities is consistently high in the suspect groups, being greater than 70% in the 3rd, 4th, and 5th decades. This is probably the most significant point in Dr. Jones' observations. The incidence of vacuoles, increased light reflex, spoking, and increased radial marking do not present the same striking difference between suspects and controls.

In Figure 8 the findings in the post capsule are given. Excepting an incidence of 20% opacities in the youngest control group as compared with 2.8% in the controls, there is no essential difference in the control and suspect groups.

I have presented the lenticular findings in Dr. Jones' survey. The data is not sufficient to make definite conclusions but it is very suggestive in that people whose work includes intimate association with microwave generators have a higher incidence of abnormal lenticular findings than persons not working with such equipment.

FIGURE 1

Under 30

30-39

40-49

| Exam | Under 30 | | 30-39 | | 40-49 | |
|--------------------|-----------------|----------------|----------------|----------------|----------------|---------------|
| | Controls 104 | Suspects 45 | Controls 74 | Suspects 48 | Controls 54 | Suspects 8 |
| Ant Capsule | | | | | | |
| Pgmt Opacities | 10 - 9.7% | 10 - 22.2% | 8 - 10.8% | 12 - 25.0% | 2 - 3.7% | - |
| Non-Pgmt Opac. | 4 - 3.8% | 1 - 2.2% | 3 - 4.0% | 1 - 2.0% | 4 - 7.4% | 1 - 12.5% |
| Ant Cortex | | | | | | |
| Opacities | 35 - 33.6% | 22 - 48.8% | 25 - 33.7% | 26 - 54.1% | 29 - 53.7% | 7 - 87.5% |
| Incr. Lite Reflex | 19 - 18.2% | 10 - 22.2% | 34 - 45.9% | 24 - 50.0% | 20 - 37.0% | 6 - 75.0% |
| Incr. Radial Mark | 12 - 11.5% | 2 - 4.4% | 34 - 45.9% | 10 - 20.8% | 45 - 83.3% | 2 - 25.0% |
| Spoking | 3 - 2.8% | 4 - 8.8% | - | 7 - 14.5% | 3 - 5.5% | - |
| Ant Adult Nucleus | | | | | | |
| Opacities | 39 - 37.5% | 4 - 8.8% | 32 - 43.2% | 15 - 31.2% | 12 - 22.2% | - |
| Cloudiness | - | - | - | - | - | - |
| Fetal Nucleus | | | | | | |
| Incr. Sut. Mark | 12 - 11.5% | 2 - 4.4% | 9 - 12.1% | 7 - 14.5% | 11 - 20.3% | - |
| Pgmt Opacities | 1 - 0.9% | - | - | - | - | - |
| Non-Pgmt. Opac. | 29 - 27.8% | 4 - 8.2% | 30 - 40.5% | 12 - 25.0% | 9 - 16.6% | 1 - 12.5% |
| Cloudiness | - | - | - | - | 2 - 3.7% | 2 - 25.0% |
| Post Cortex | | | | | | |
| Opacities | 30 - 28.8% | 32 - 71.1% | 19 - 25.6% | 35 - 72.9% | 32 - 59.2% | 8 - 100.0% |
| Vacuoles | 1 - 0.9% | - | 2 - 2.7% | - | 1 - 1.8% | - |
| Incr. Lite Reflex | 17 - 16.3% | 11 - 24.4% | 30 - 40.5% | 26 - 54.1% | 27 - 56.0% | 6 - 75.0% |
| Spoking | 1 - 0.9% | 4 - 8.8% | - | 6 - 12.5% | - | - |
| Incr. Rad. Mark | 3 - 2.8% | 2 - 4.4% | 1 - 1.3% | - | 3 - 5.5% | - |
| Post Capsule | | | | | | |
| Opacities | 3 - 2.8% | 9 - 20.0% | 4 - 5.4% | 3 - 6.3% | 1 - 1.8% | - |
| Incr. Granularity | - | 5 - 11.1% | 1 - 1.3% | 2 - 4.1% | - | - |
| Mittendorf Dot | 4 - 3.8% | 3 - 6.6% | 1 - 1.3% | - | 1 - 1.8% | - |
| Vacuoles | 1 - 0.9% | - | - | 3 - 6.3% | 1 - 1.8% | - |
| Post Adult Nucleus | | | | | | |
| Opacities | 8 - 7.6% | 2 - 4.4% | 7 - 9.4% | 7 - 14.5% | 1 - 1.8% | - |
| Cloudiness | - | - | - | - | - | - |

FIGURE 1 (cont'd)

| Exam | 50 - 59 | | Over 60 | |
|--------------------|----------------|---------------|---------------|----------|
| | Controls 18 | Suspects 6 | Controls 8 | Suspects |
| Ant Capsule | | | | |
| Pgmt Opacities | 1 - 5.5% | - | 1 - 12.5% | - |
| Non-Pgmt Opac. | - | 1 - 16.6% | - | - |
| Ant Cortex | | | | |
| Opacities | 5 - 27.7% | - | 6 - 75.0% | - |
| Incr. Lite Reflex | 10 - 55.5% | 6 - 100.0% | 4 - 56.0% | - |
| Incr. Radial Mark | 18 - 100.0% | 2 - 33.3% | 6 - 75.0% | - |
| Spoking | 2 - 11.1% | - | 1 - 18.5% | - |
| Ant Adult Nucleus | | | | |
| Opacities | 11 - 61.1% | - | 5 - 62.5% | - |
| Cloudiness | - | 2 - 33.3% | - | - |
| Fetal Nucleus | | | | |
| Incr. Sut. Mark | 2 - 11.1% | - | - | - |
| Pgmt Opacities | - | - | - | - |
| Non-Pgmt Opac. | 10 - 55.5% | - | 3 - 37.5% | - |
| Cloudiness | 2 - 11.1% | 4 - 66.7% | 4 - 50.0% | - |
| Post Cortex | | | | |
| Opacities | 5 - 27.7% | 2 - 33.3% | 5 - 62.5% | - |
| Vacuoles | 4 - 22.2% | - | - | - |
| Incr. Lite Reflex | 10 - 55.5% | 6 - 100.0% | 5 - 62.5% | - |
| Spoking | - | - | - | - |
| Incr. Rad. Mark | 1 - 5.5% | - | - | - |
| Post Capsule | | | | |
| Opacities | - | - | - | - |
| Incr. Granularity | 2 - 11.1% | - | - | - |
| Mittendorf Dot | - | - | - | - |
| Vacuoles | - | 2 - 33.3% | - | - |
| Post Adult Nucleus | | | | |
| Opacities | 1 - 5.5% | 2 - 33.3% | 3 - 37.5% | - |
| Cloudiness | - | - | - | - |

NEW MICROWAVE DOSIMETRY AND THE PHYSIOLOGIC NEED

by

Alfred W. Richardson, Ph.D.*

INTRODUCTION

Recognition should be given to Dr. Theodore Cooper, Mr. Walter Ciszczon and Miss Teresa Pinakatt for their aid in the execution of the physiological portions of these studies and in the testing procedures.

PERTINENT PHYSIOLOGICAL STUDIES

Excessive exposure of animals to microwaves produces many physiologic alterations in the homeokinetic body systems, and if the exposure is sufficient pathologic changes are produced. These effects have been well established by experimental evidence in numerous laboratories. The brief review here will include only selected findings in our laboratory that reveal the need for adequate field dosimetry to protect human personnel.

Although we have been able to demonstrate a multitude of physiologic and pathologic changes in animals using field densities of 25 mw/cm^2 and greater, none of our observed findings to date refute the concept that 10 mw/cm^2 is a permissible microwave dosage.

Body Distribution of Hyperthermia

Apparently the brain is the key site of critical hyperpyrexia when an animal is subjected to microwave-induced hyperthermia. Where 60 albino rats were irradiated to the lethal point using selected (masked) exposures of the head, the chest, the abdomen, and the whole animal, the critical midbrain temperature was 40.5°C . (plus or minus 0.5°C). In these studies at the lethal point of exposure the rectal temperature, usually assumed to represent the "core" temperature of the body, varied from 41 to 48°C .

Direct head exposure is not the most effective way to increase the temperature in the brain, because the skull presents a reflecting barrier. The brain temperature is most effectively elevated by exposure of the chest above the midline, where the brain is heated by blood convection. This finding is in keeping with the hemodynamics of the body where all the blood must be recirculated through the lungs and heart, and 30% is circulated through the liver to be reheated and passed back to the lungs. The brain received 14% of the left cardiac output, all of which has minimal heat loss in passage to the brain. With 4% of the cardiac output going into coronary circulation, microwave irradiation of the chest reheats

about half of the blood of the body in a semi-closed circuit, and all of the blood of the body when it returns to the chest cage.

When the abdominal area is selectively heated by masking, the lungs become a site of heat dissipation rather than heat induction. In our studies with abdominal exposure the rectal temperature increased to 47-48°C. coincident with the time that the brain reached the critical temperature of 40.5°C. With exposure of the whole body of the animals the rectal temperature was 45.5°C., and with chest exposure 41°C., when the brain temperature reached 40.5°C. It is of interest that the consistent critical temperature in the brain was 5°C. less than the temperature rectally, with whole body exposure.

Whereas these findings demonstrate the nebulous aspect of rectal temperatures to be employed for the prediction of brain temperature, the error is on the safe side, i.e., even with wide fluctuations the rectal temperature tends to be higher. However, where human personnel are involved with microwave exposure, rectal thermometry is remarkably impractical, and oral thermometry has dubious practicality. Convenient microwave or radar dosimetry presents a better solution to the problem.

A NEWLY DEVELOPED RADAR DOSIMETER

The original type CP Richardson microwave dosimeter responded as an analogue to the temperature rise in human tissues when exposed to microwaves. It used an electrolytic gelatin capsule for the sensing element. This device has been altered to include automatic temperature compensation, but will not be reported here.

The new dosimeter, type P, to be reported here does not require special temperature compensation, and responds to radar per se. A photograph of the finished model is shown in Figure 1. It is smaller than a king size pack of cigarettes and about the same shape. It is not responsive to cw microwaves, or other cw rf energy, but it responds to radar over a wide band to cover all current frequencies and pulse conditions. It features 360° pickup, a high but variable sensitivity, and responds with both a visual and auditory signal at the output.

Figure 2 shows a basic diagram of the instrument. The dotted lines indicate the shielded area. At the input are three resistors to be arranged in three right angle vectors bent to form a sphere. The composite signal pickup leads to a diode and special RC arrangement which erases the carrier signal and transmits the pulse wave to the basic amplifier depicted by a block diagram. A gain control is provided. The amplifier output leads to a miniature transformer across which there is a phone connection for a miniature earphone. The radar pulse can be heard as a tone frequency, and tests have indicated that the ear can differentiate one transmitter from another. A difference in pulse frequency obviously can be discriminated.

Resistors R1 and R2 along with the condenser at the input serve to integrate the highly peaked square wave into a modified saw tooth wave, so as to diminish the height and increase the width. This makes the signal more audible and eliminates loading at the input. Capacitor C5 across the gain control aids also in shaping the wave. At the output transformer secondary, the AC wave is rectified to DC and recorded on a miniature 50 to 200 microampere panel meter.

The basic amplifier is shown in Figure 3, and is revealed to be a four stage transistor amplifier of classic stable feedback design. The amplifier used in the model described is a commercial compact postage stamp size, that can be chosen to cover various frequency ranges for pulse detection. This one covers 30 to 10,000 pps with a "flat" response (almost negligible loss).

Tests of this dosimeter in our laboratory have demonstrated a sensitivity of detectible response from a few microwatts per cm^2 up to a fraction of a watt per cm^2 . At the highest gain setting we had no field meter with sufficient sensitivity to calibrate with precision, but a pulsed Maxson generator at 3 watts average output could be readily detected at over 100 feet in the side fringe area of the field of the director.

This is not a dosimeter designed for accumulated field energy as was the original CP model. It quantitatively measures field density per se. However, where average fields of rotating antennae are involved, the meter can be altered to average the field by increasing the capacitance at the output section in Figure 2, and adding a small resistor in series at each side of the top of the condenser.

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ABSTRACT

Physiologic findings indicate that the brain temperature is the critical thermic site in microwave-induced hyperpyrexia of the body of the albino rat. Selected conditions of exposure suggest that the rectal temperature may have a widely variable relationship to the critical brain temperature.

With the recognized need for dosimetry for personnel protection around radar, a newly designed radar dosimeter is described which is not temperature sensitive, but which is extremely sensitive to pulsed microwaves. It is a compact miniature size, and offers both visual and auditory response.



FIGURE #1

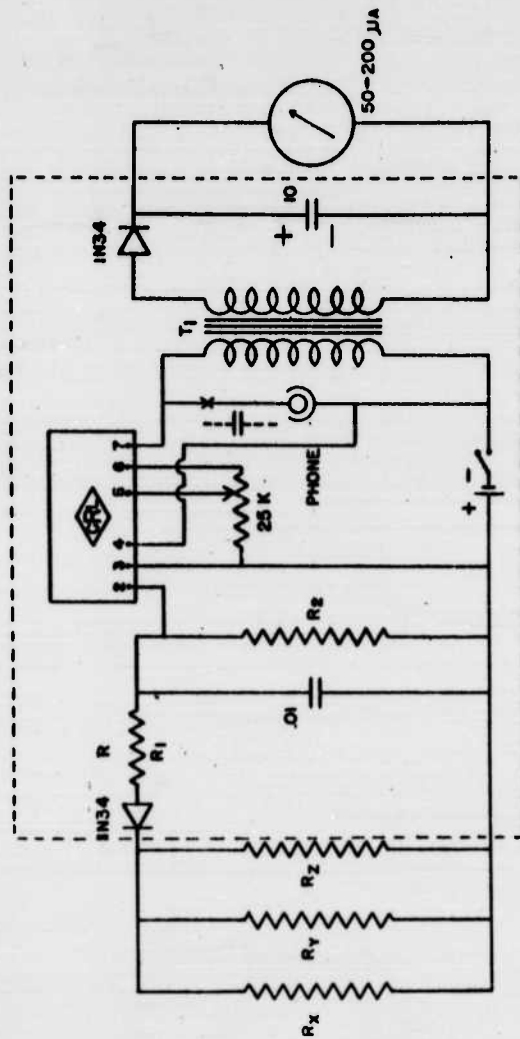
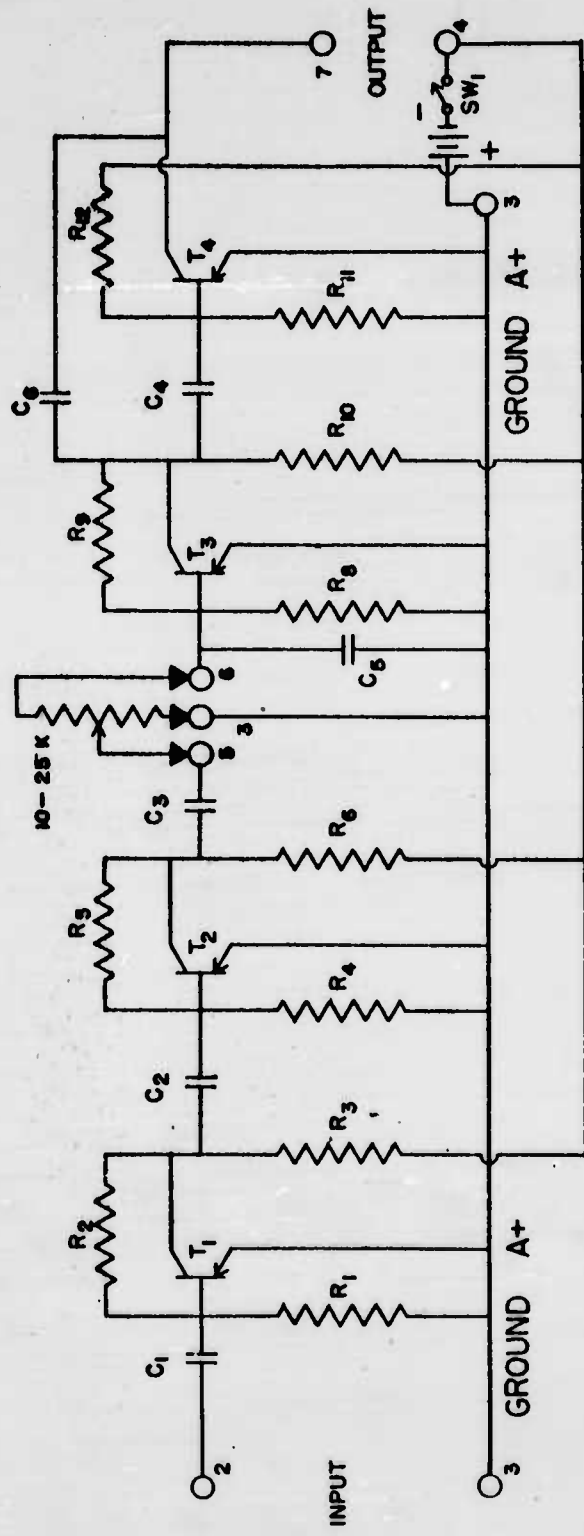


FIGURE #2



BASIC AMPLIFIER

FIGURE #3

A PRELIMINARY INVESTIGATION OF THE APPLICATIONS OF
MAGNETIC RESONANCE ABSORPTION SPECTROSCOPY TO
THE STUDY OF THE EFFECTS OF MICROWAVES ON
BIOLOGICAL MATERIALS*

by

George Pish, William H. Storey, Jr., Frank Truby, William Rollwitz
Southwest Research Institute, San Antonio, Texas

ABSTRACT

Electron paramagnetic resonance (EPR) methods were found applicable to show changes induced by microwave radiation upon the eyes of rabbits. The EPR signals obtained from ionizing radiation induced free electrons in the posterior cortex tissue of the lens show a simple singlet structure with the differences between normal and opaque lenses giving a doublet structure. Also, observation of dilute solutions of lens tissue shows the effects of microwave damage. The free radicals induced in lenses exposed to microwave radiation have a longer life than those from normal lenses.

Differences in protein structure between normal rabbit eye lenses and rabbit eye lenses in which cataracts have been induced by microwave irradiation have been investigated by high resolution nuclear magnetic resonance (NMR). NMR spectra from the proteins in rabbit eye lenses are given and are interpreted grossly. Differences in the NMR spectra between some of the cataract-bearing lenses and their non-cataract controls are pointed out, and the meanings of the differences are suggested. The effects of the presence of moisture on the action of trifluoroacetic acid on proteins is considered as a possible explanation for the time dependence of the data. In addition, a table is given showing the dry weights of the samples, together with weight loss upon drying.

Recommendations are presented for the continuation of the EPR and NMR investigations.

*The data presented are taken from the RADC-TR-59-81 report. The program was supported under RADC-AF 30(602)-1843 Contract.

INTRODUCTION

In this investigation magnetic resonance methods were applied in order to determine whether differences could be observed between spectra from lenses with microwave radiation induced opacity and those from normal lenses. Both electron paramagnetic resonance (EPR) and nuclear magnetic resonance (NMR) methods were used. Dr. Russell L. Carpenter's group at Tufts University prepared the lenses used in these experiments. The samples were prepared and observed in pairs from the same rabbit. One lens of each pair had a cataract induced by 2450 megacycle microwaves, while the other member had been left unirradiated for use as a control. For EPR studies only, the posterior cortex samples were supplied. However, whole lenses were supplied for NMR studies. All samples had been packed in dry ice after excision and shipped to SwRI immediately in a dry ice package.

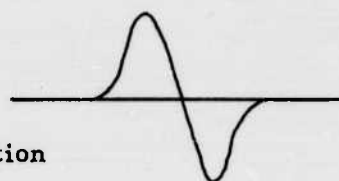
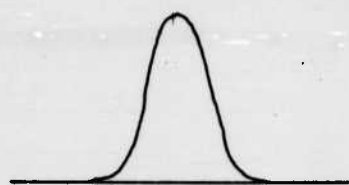
EPR STUDIES

Since there were no measurable electron resonance signals from free radicals occurring naturally in the lenses, unpaired electrons to be used as observers for any changes that may have occurred were introduced by ionizing radiation. These unpaired electrons were trapped in some position in the structure and kept from recombining by keeping the samples at or below -180°C at all times, except when plotting a temperature decay curve.

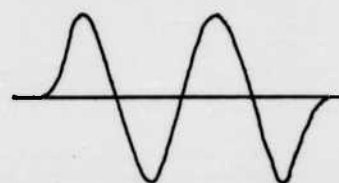
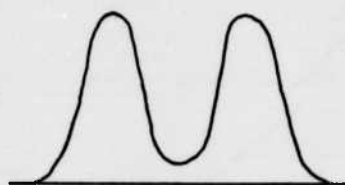
Induced unpaired electrons are very sensitive to changes in structure near the point at which they lodge after having been generated by the gamma rays. If the changes induced by opacities caused a change in the position or number of hydrogen atoms, there will be a change in the interaction between the hydrogen nucleus and the unpaired electron. The effect of a single hydrogen nucleus is shown in Figure 1. The absorption curve is the plot of the energy absorbed by the unpaired electrons as the magnetic field causing them to absorb energy is varied. The instrumentation required to record the signal draws the derivative curve instead of the absorption curve. With no interaction, the curve is very simple. With total interaction, two distinct absorption curves are obtained; with only partial interaction, a central curve with two "wings" is obtained. Total interaction is where all of the unpaired electrons are acted upon by hydrogens, and partial interaction is where only part are interacting. The derivative curve is the only one drawn by the instrument.

Absorption Curve

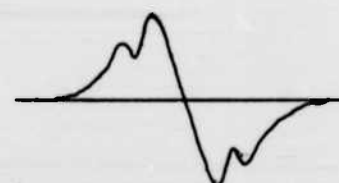
Derivative Curve



a. No Interaction



b. Total Single Hydrogen Interaction



c. Partial Single Hydrogen Interaction

Figure 1. Hydrogen Interaction with the Free Electron Absorption

Figure 2 shows a comparison between the derivative curves from a normal lens (Sample 14A) and from an opaque lens (Sample 14). The partial interactions are seen at points I and II.

Table I shows the kind of samples received and the differences obtained by EPR measurement. In the first series (Group 1) of samples investigated, some were whole lenses. Since the whole lens could not be "seen" by the instrument, but the cortex could, only the cortex samples were used. Freeze drying seems to give the more consistent results since all of the samples of Group 1, when freeze dried, showed differences. The samples of Group 2 for each sample number had a correspondingly numbered normal lens from the same rabbit designated 1A, 2A, 5A, etc. The samples of Group 2 were all quick frozen instead of freeze dried. There were no differences noted except between Samples 14 and 14A, as shown in Figure 2 before. The lack of any differences may be due to the following:

1. The presence of insufficient opaque material to give any differences. Sample 14 had the most extensive opacity although not an order of magnitude greater than Samples 5, 7, and 9.
2. The lodging of the unpaired electron in a position insensitive to the changes caused by an opacity.

Further controlled experiments of quick freezing and freeze drying must be undertaken to obtain sufficient information for definite conclusions.

A sample of lens dissolved in trifluoroacetic acid was obtained from the NMR experiments, quick frozen, and irradiated. Sample 10 was opaque and Sample 10A was clear. The derivative curves obtained are shown in Figure 3. The differences between the curves are readily apparent at points I and II. The derivative curves for these dissolved samples are different in shape from the undissolved samples because of the interactions of the hydrogen nuclei in the trifluoroacetic acid.

The EPR measurements have shown the following results:

1. It is possible to obtain by EPR measurements an indication of the changes caused by a microwave irradiation opacity in a rabbit lens. The change is not indicated in all lenses measured with and without opacities.

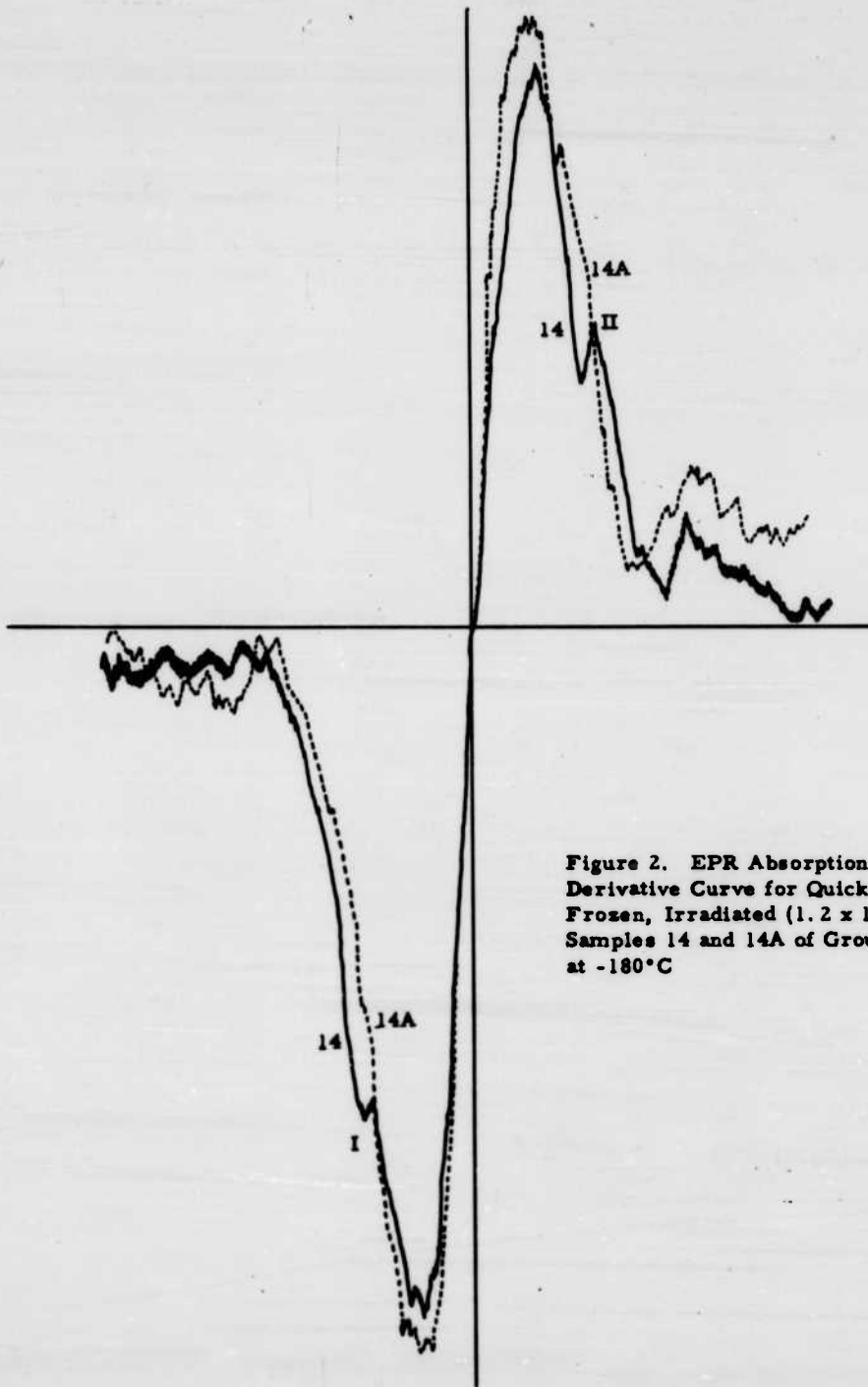


Figure 2. EPR Absorption
Derivative Curve for Quick
Frozen, Irradiated (1.2×10^6 rad)
Samples 14 and 14A of Group 2
at -180°C

TABLE I. RESULTS OBTAINED BY EPR METHODS

| Sample Group | Sample No. | Description | Microwave Radiation Power | Irradiation Time | EPR Measurement Results |
|----------------------|------------|--|---------------------------|------------------|-------------------------------------|
| 1 Freeze Dried | 4 | Normal lens cortex | 0 | 0 | No interaction |
| | 6 | Normal lens cortex | 0 | 0 | No interaction |
| | 8 | Cortex with large diffuse cataract | 250 mw/cm ² | 15 min | Partial single hydrogen interaction |
| | 11 | Cortex with extensive opacity | 250 mw/cm ² | 15 min | Partial single hydrogen interaction |
| 2 Quick Frozen | 1 | Posterior cortex removed immediately after exposure | 250 mw/cm ² | 15 min | No interaction |
| | 2 | Posterior cortex removed immediately after exposure | 250 mw/cm ² | 15 min | No interaction |
| | 5 | Posterior cortex of lens with extensive posterior opacity showing granules, vesicles, and fibers | 250 mw/cm ² | 15 min | No interaction |
| | 6 | Posterior cortex removed immediately after exposure | 250 mw/cm ² | 15 min | No interaction |
| | 7 | Posterior cortex with extensive opacity | 250 mw/cm ² | 15 min | No interaction |
| | 9 | Posterior cortex with extensive opacity | 250 mw/cm ² | 15 min | No interaction |

TABLE I. RESULTS OBTAINED BY EPR METHODS (Cont'd)

| Sample Group | Sample No. | Description | Microwave Radiation Power | Irradiation Time | EPR Measurements Results |
|--------------|------------|---|---------------------------|------------------|-------------------------------------|
| Quick Frozen | 10 | Whole lens with hyper-mature cataract dissolved in trifluoroacetic acid | 250 mw/cm ² | 15 min | Partial single hydrogen interaction |
| | 12 | Posterior cortex of right eye lens removed immediately after exposure | 250 mw/cm ² | 15 min | No interaction |
| | 14 | Posterior cortex of right eye lens with extensive opacity | 250 mw/cm ² | 15 min | Partial single hydrogen interaction |

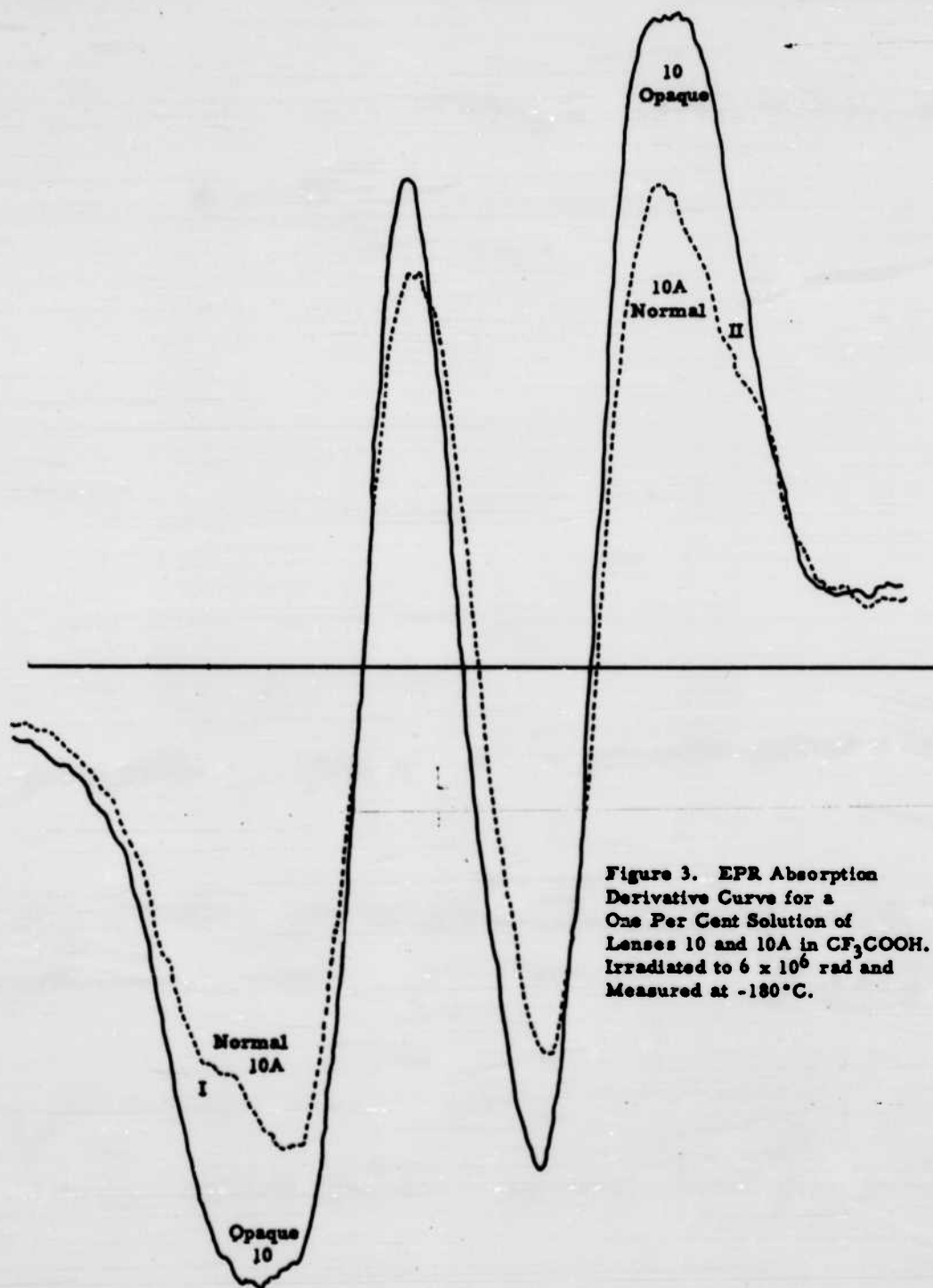


Figure 3. EPR Absorption Derivative Curve for a One Per Cent Solution of Lenses 10 and 10A in CF_3COOH . Irradiated to 6×10^6 rad and Measured at -180°C .

2. In many opacities, the concentration of the opacity may be too small to give an indication with the relatively low signal/noise ratio equipment employed.
3. Instrumentation with a much higher signal/noise ratio should be used.
4. No changes were indicated in lenses removed immediately after microwave exposure.
5. The curves obtained from normal lens tissue alone show a simple singlet structure.
6. The curves obtained from opaque lens tissue alone show a singlet structure with a superimposed doublet structure indicating a single hydrogen interaction with the unpaired electron.
7. One per cent solutions of lens tissue showed a more readily defined difference between normal and opaque lenses.
8. Free radicals in lens tissue in solution decay completely within 10 minutes at -95°C .
9. Solutions of lens tissue show the same shape for both the normal and opaque lenses with the amplitude of the hydrogen interaction lines being larger in the opaque lens curve.
10. Solutions of lens tissue have unpaired electron positions so as to give an interaction with two hydrogens which produces three points of change in the curve.

NMR STUDIES

Upon receipt of the lenses for NMR studies at this laboratory, they were weighed, lyophilized under forepump pressure at -20°C to apparently constant weight, and then reweighed. Weight losses expressed as a per cent of the dry weight of the samples ranged from 141% to 179%, except for those samples which were listed as having hypermature cataracts. Per cent weight losses of samples containing hypermature cataracts ranged from 266% to 388%. Dry weights of those samples containing hypermature cataracts tended to be less than their controls. Table II summarizes the weights of the individual samples. The lyophilized samples were dissolved in trifluoroacetic acid for NMR observation.

TABLE II. DRY WEIGHT AND PER CENT WEIGHT LOSS OF
 SAMPLES DURING LYOPHILIZATION (GROUP II)

| Sample No. | Hypermaturation Cataract ? | Weight of Dry Sample (gm) | Per Cent Weight Loss (Wt Loss x 100/Dry Wt) |
|------------|----------------------------------|---------------------------------|--|
| 3A | | .134 | 157 |
| 3 | yes | .058 | 388 |
| 4A | | .152 | 150 |
| 4 | yes | .117 | 266 |
| 8A | | .152 | 146 |
| 8 | no | .152 | 141 |
| 10A | | .119 | 162 |
| 10 | yes | .135* | 274 |
| 11A | | .119 | 163 |
| 11 | yes | .099 | 337 |
| 13A | | .119 | 148 |
| 13 | no | .124 | 168 |
| 15A | | .133 | 148 |
| 15 | no | .132 | 160 |
| 16A | | .129 | 164 |
| 16 | no | .115 | 157 |
| 18A | | .141 | 159 |
| 18 | no | .129 | 179 |
| 19A | | .115 | 158 |
| 19 | no | .118 | 165 |

*This lens was damaged and some of it was smeared over and firmly adhered to the side of the container in which it was shipped. Only 0.061 gm was noted as having been transferred to a weighing bottle for dissolving. This seems too high a loss, and the more questionable figure is the one given in the table.

Figure 4 shows a typical spectrum from one of the control samples. The tetramethylsilane reference peak and one of its 60 cycle sidebands is shown to the far right of the spectrum. The use of tetramethylsilane as an internal reference has been investigated by Tiers, of the Minnesota Mining and Manufacturing Company. Following Tiers, a value of 10.0 ppm has been assigned to the position of the tetramethylsilane peak, shifts from this position being expressed as parts per million of the applied magnetic field. Bovey and Tiers, both of the Minnesota Mining and Manufacturing Company, have obtained spectra of the majority of amino acids and some of their peptides, and the interpretation of this spectrum is based on their work. Several of the spectral bands can be assigned with considerable certainty. Four of these are the CONH band at 2 ppm, the alpha-H band at 5 ppm, the band from methyl groups in leucine, valine, and possibly isoleucine at 9 ppm, and the band from aryl groups in such amino acids as phenylalanine and tyrosine at about 2.8 ppm. If methionine were present, we would expect to find the methylene groups and the methyl group in the region indicated. Several possibilities exist for the other spectral features, some of which are indicated, but these bands cannot be assigned to particular amino acids with certainty. The sharp little peak at about 4 ppm is the central peak of a triplet due to an impurity, probably difluoroacetic acid in the trifluoroacetic acid. The other members of the triplet are barely visible 53 cycles/second on either side of this central peak.

In Table III are summarized the results of the investigation. The samples were observed over a period of approximately three weeks in the order shown. The first two samples observed were left over, partly by accident, from an earlier group of samples which we have labeled Group 1. We wish to call attention here to the following facts:

1. The first samples that were observed did show significant differences in their spectra.
2. In observations of the later samples, no differences could be detected between spectra from the cataractous lens samples and their controls.
3. Changes in spectra from the same sample over a period of several days became rather reproducible from sample to sample for the later samples. The spectra from Samples 11 and 11A do not fit in either category.

The following figures will illustrate what we term significant differences and the sort of changes in the spectra with time that we observed.

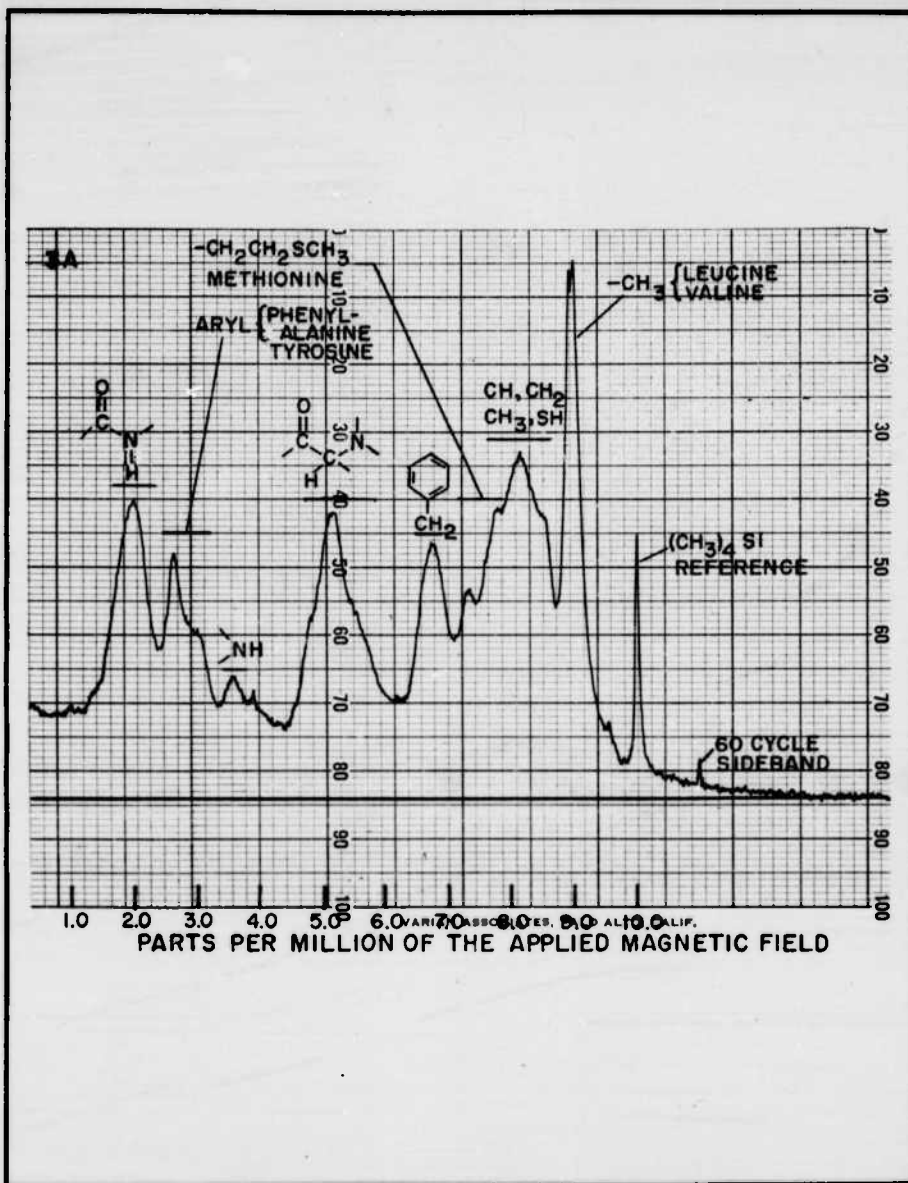


FIG. 4. PARTIAL ASSIGNMENT OF PEAKS
IN RABBIT LENS SPECTRA

TABLE III. SUMMARY OF NMR RESULTS FOR RABBIT LENSES INVESTIGATED

| Sample Pair | Change with Time? | Significant Differences in Spectra? | | |
|-------------------------|-------------------|-------------------------------------|-------------------------------|-------------------------------|
| | | What Spectral Region? (ppm) | Immediately After Dissolving? | After Elapse of Several Days? |
| Group 1: 2 (control) | } yes | general | yes | |
| 10 (cataract) | | | | 7.6 - 7.8 |
| Group 2: 3 | no | | yes | { 5.6 8.0 - 8.6 |
| 4 | no | | yes | 7.6 - 7.8 |
| 8 | yes | 7.6 - 7.8 | yes | 7.6 - 7.8 |
| 10 | yes | { 5.8 - 6.0 7.6 - 7.8 | yes | { 5.8 - 6.0 7.6 - 7.8 |
| 11 | no | | no | |
| 13 | yes | 5.8 - 6.3 | no | |
| 15 | yes | 5.8 - 6.0 | no | |
| 16 | yes | 5.8 - 6.3 | no | |
| 18 | yes | 5.8 - 6.3 | no | |
| 19A | } yes | 5.8 - 6.3 | } no | |
| 19 | | | | |

Figure 5 shows spectra from Samples 2 and 10, Group 1, Sample 2 being the control and Sample 10 being a cataractous lens. These lenses were not from the same rabbit. The principal feature of interest in these two spectra is that the spectrum from Sample 10 displays a sharp peak of high amplitude in the neighborhood of 7.7 ppm, whereas the corresponding peak in the spectrum from Sample 2 is of lesser amplitude. Sample 2 gelled in a storage vessel before any more observations could be made. However, we were able to follow Sample 10 for two more days.

Figure 6 shows the extensive changes that are taking place in the spectrum. The peak at 7.7 ppm seems to be diminishing in amplitude with time, whereas other peaks in this spectrum are becoming more pronounced. Possibly hydrolysis of this sample is taking place and the sharp peaks we are observing are due to free peptides of relatively low molecular weight in solution. This is the only lens sample in which such extensive changes were observed.

Figure 7 shows spectra from Samples 4 and 4A. Again we see in the neighborhood of 7.7 ppm a sharp peak of high amplitude in the spectrum from Sample 4, but not from Sample 4A.

Figure 8 shows spectra from Samples 10 and 10A in which the most dramatic difference in the region of about 7.7 ppm was observed. The peak in the spectra from Sample 10 is of great enough amplitude that sidebands on this peak are clearly seen. This peak is present in small amplitude in the spectra from Sample 10A. In the later spectrum from Sample 10A, we note the growth of a peak at about 5.9 ppm. The later spectrum from Sample 10 does not contain this peak.

Figure 9 shows spectra from Samples 16 and 16A, which demonstrate a behavior typical of the group of lenses that were observed at the end of the investigation. The first spectra show several small peaks in the region from about 5.8 to 6.3 ppm. The small peaks almost disappeared, giving rise to a pronounced single peak at about 5.9 ppm in the later spectra. This behavior was always observed at either the first or the second or possibly the third exposure to the atmosphere during observation of the sample. It was as if the growth behavior depended upon exposure to the atmosphere, since the samples were run in unsealed tubes. During storage the samples were at least partially sealed in weighing bottles.

Returning to Table III, several points should be made. First, we cannot identify the peaks in either of the regions where differences or

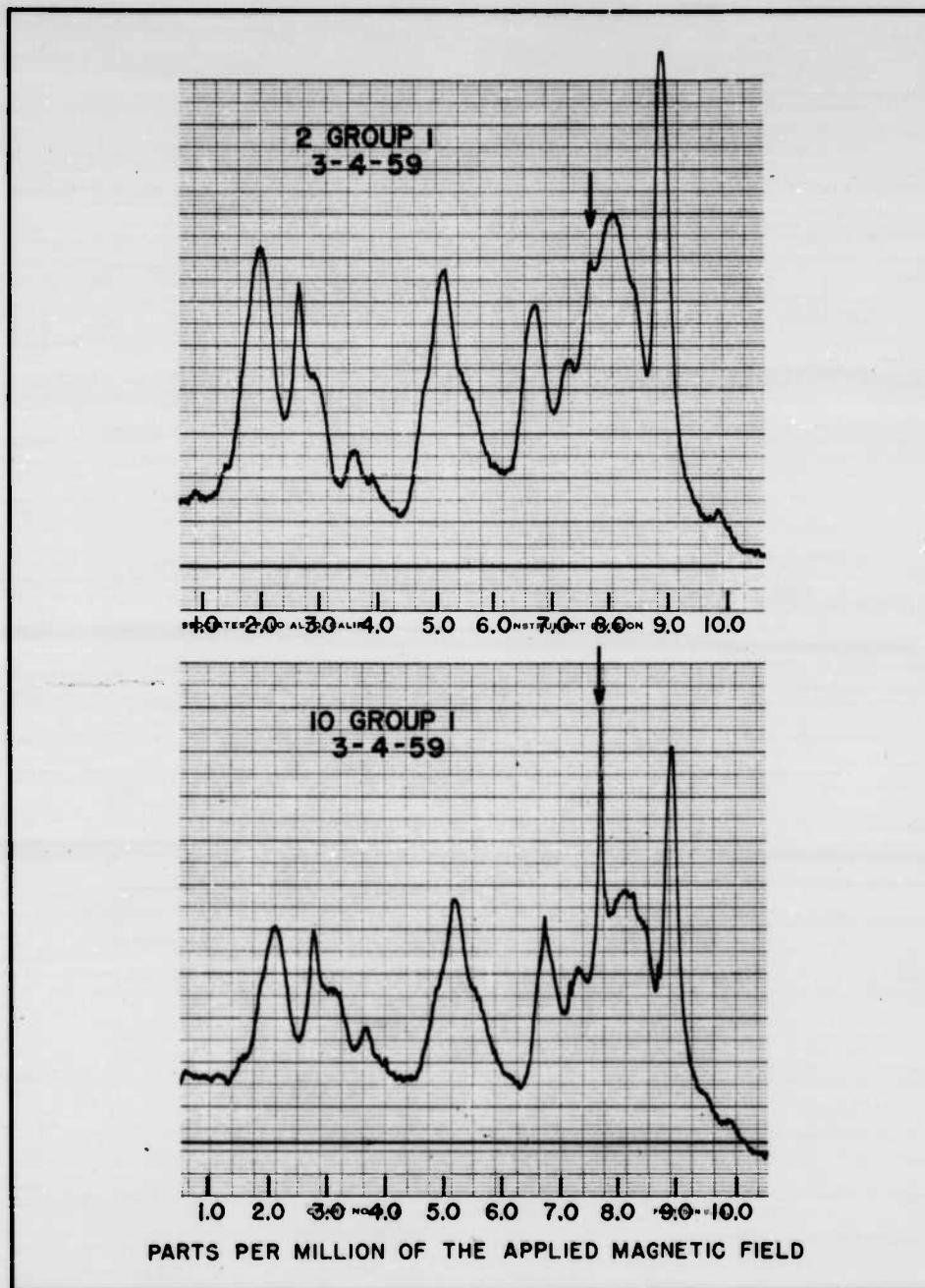


FIG. 5. NMR SPECTRA FROM NORMAL AND CATARACTOUS RABBIT EYE LENSES

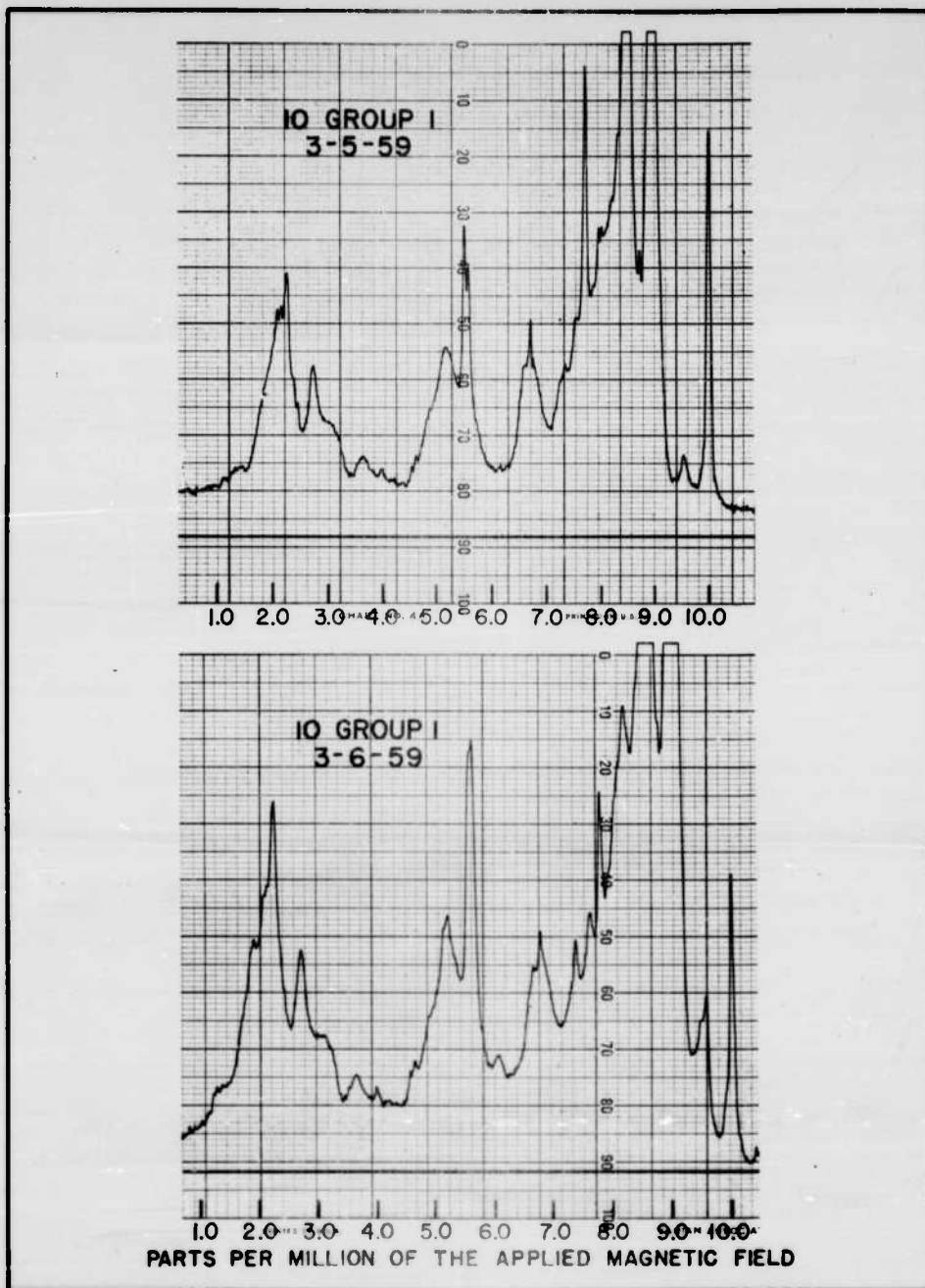


FIG. 6. NSR SPECTRA FROM NORMAL AND CATARACTOUS RABBIT EYE LENSES

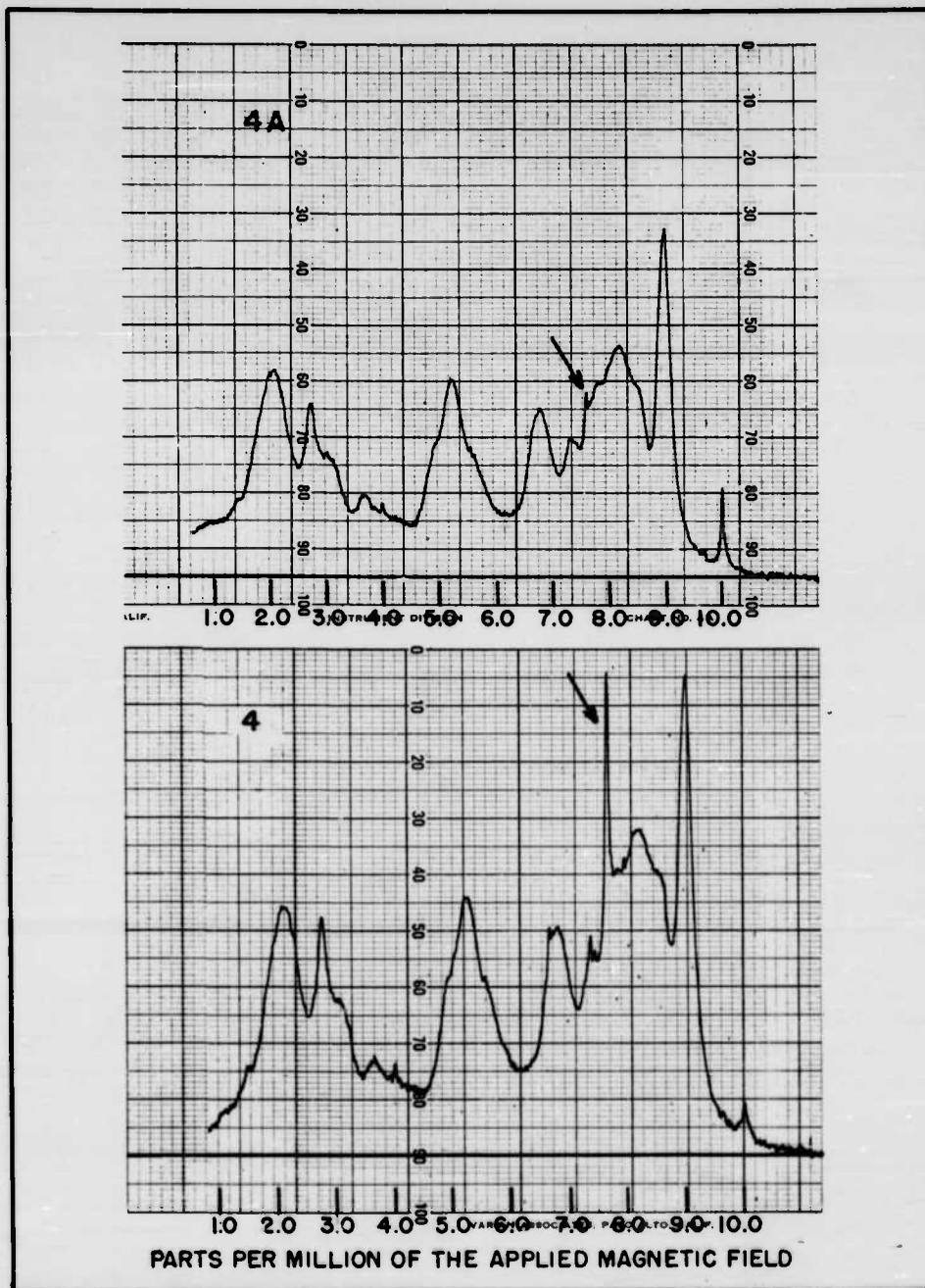


FIG. 7. NSR SPECTRA FROM NORMAL AND CATARACTOUS RABBIT EYE LENSES

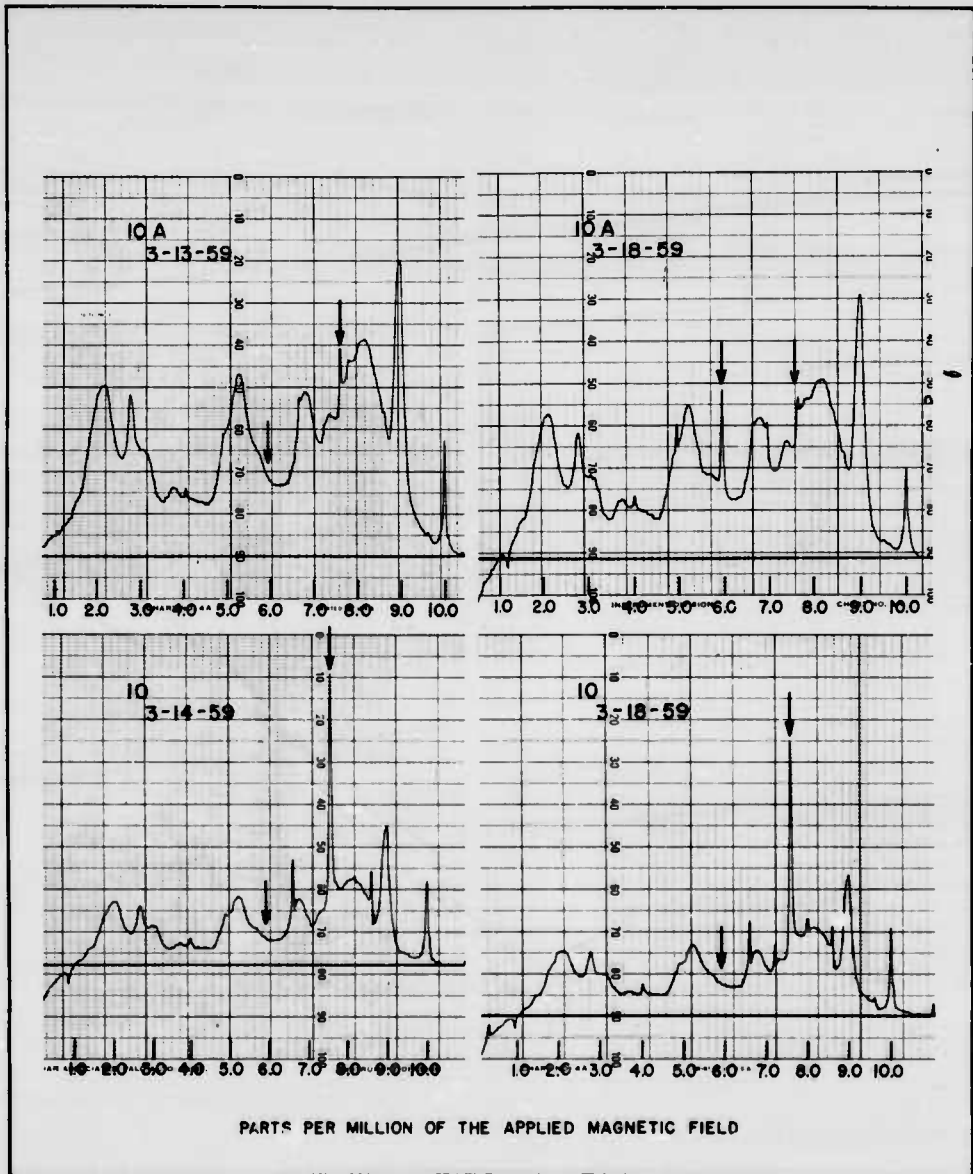


FIG. 8. NSR SPECTRA FROM NORMAL AND CATARACTOUS RABBIT EYE LENSES

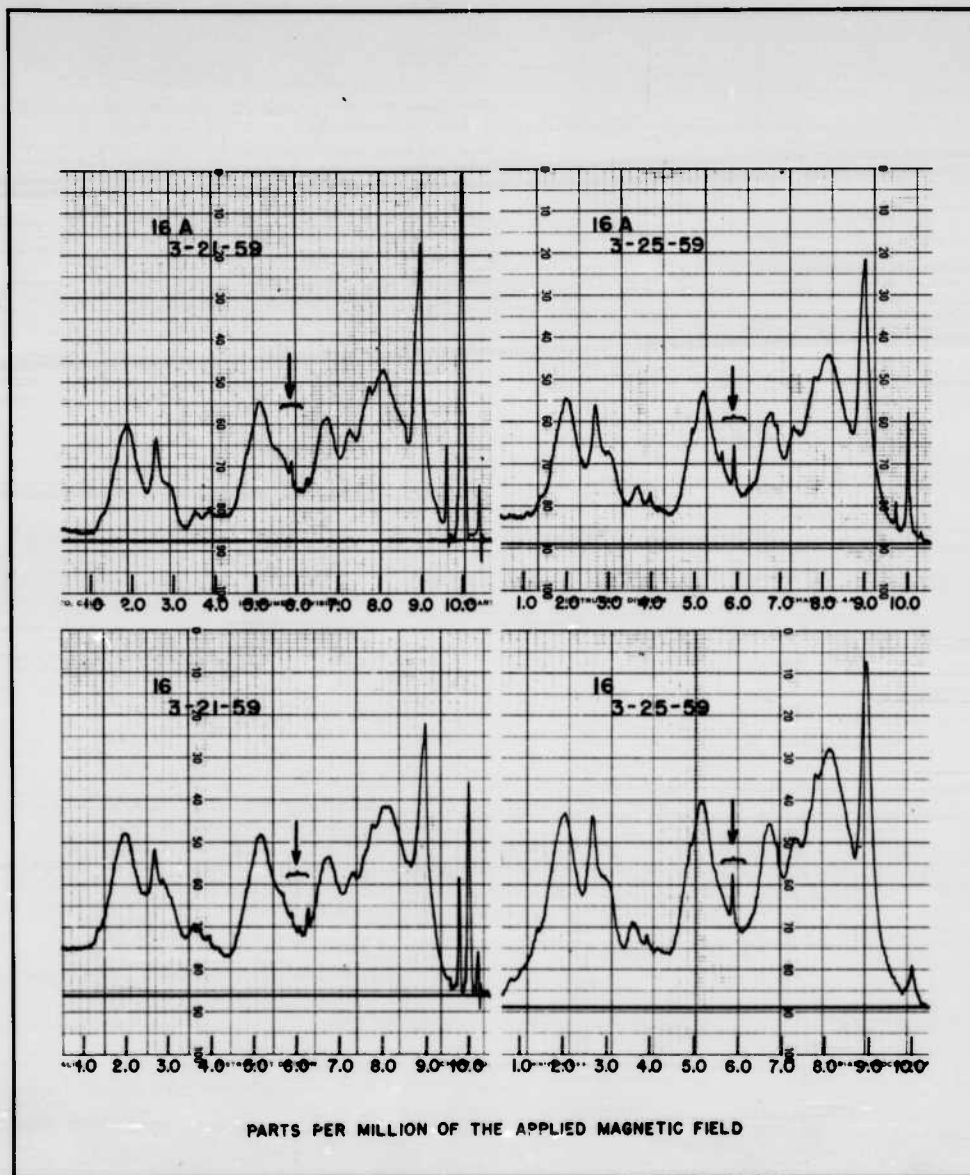


FIG. 9. NSR SPECTRA FROM NORMAL AND CATARACTOUS RABBIT EYE LENSES

changes are occurring. The region of 7.6 to 7.8 ppm coincides with the region where Bovey and Tiers have observed sharp peaks due to the SCH_3 group in methionine. Yet we have observed apparent reduction of the amplitude of this peak with time, the reaction mechanism for which is not clear. Further, we have found that sharp unexplained peaks can occur in this region in spectra from amino acids. Until this point is investigated further we cannot make positive assignment. The origin of the peaks in the region from 5.8 to 6.3 ppm is altogether unknown. Second, the samples that were observed first seem to form a group and the samples that were observed last seem to form another. Significant differences between a cataractous lens and its control were obtained throughout the first group, especially with samples where the cataract had been noted as hypermature. The only exception to this is that Samples 11 and 11A showed no difference. None of the cataractous lenses in the latter group were noted as hypermature, which may be sufficient reason for having obtained no differences between spectra from these samples and their controls. Third, since we were pumping on the unprepared lenses throughout the experiment, the lenses observed last should have contained less moisture than the first lenses. One explanation for the grouping of the samples may therefore be that the lenses in the first group contained more moisture than the lenses in the second group. The assumption is that the presence of a trace amount of moisture in the lens is a necessary condition for the enhancement of the spectral differences we noted in the samples first observed. Other work in this laboratory has shown that enhancement of the spectra from certain amino acids and proteins with the addition of moisture is possible.

RECOMMENDATIONS

1. Further EPR and NMR investigations should be continued as described in the program just reported. Conditions for obtaining more consistent and reproducible spectra should be studied.
2. Investigations should be continued at 2450 megacycles. In addition, samples which have been irradiated at some frequency below and above 2450 megacycles should be considered also.
3. To answer the question of whether the protein structure characteristics from different kinds of eyes are similar, different animals, as well as human eyes, should be compared.

A MICROWAVE MEDICAL SAFETY PROGRAM
IN AN INDUSTRIAL ELECTRONICS FACILITY

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SUMMARY

An Industrial Electronics Medical Program is reviewed with special consideration given to the microwave eye examination and the blood count. A possible case of radiation cataract is discussed. The microwave safety program is outlined with particular emphasis placed on microwave radiation detection with a survey meter and various methods of area protection.

INTRODUCTION

This presentation will concern itself primarily with the experience gained with the medical and safety aspects of microwave at the Sperry Gyroscope Company, Division of Sperry Rand Corporation. This is an electronics manufacturing plant with approximately 18,000 employees whose center of operations is located on Long Island, New York. The organization is engaged in producing large quantities of radar equipment for the Armed Services. One of the chief component products manufactured and employed in systems is the klystron tube. Common plate voltages range from 30 to 150 kilovolts but may exceed 270 kilovolts. Peak power is often in the multi-megawatt region. Average power may well be tens of kilowatts. The instruments might be considered in the same category as powerful diathermy and X-Ray machines. Most of the usual wavebands, X, S, C and L, are produced and the tendency more recently has been toward longer wavelengths and greater power for various technical reasons which I cannot discuss here.

MICROWAVE HAZARDS

There are serious problems from the safety standpoint with microwave emitters other than from the radio frequencies produced. Foremost among these is the ionizing radiation. Time does not permit detailed discussion of this but suffice to say that every attempt is made to build into the tube the necessary lead shielding. Care must be taken to eliminate leaks at seams and cooling pipe exits on the tube. Any malfunction of tubes increases the hazard. Surveys utilizing large X-Ray films to locate the rays are regularly made on new devices. The relative biological effectiveness is greater with this radiation because of transmission through the cover material of the tube, therefore the danger is increased.¹ The production of X-Ray is often greater than a therapeutic X-Ray machine.

It should also be mentioned that high voltages with the dangers of burns and electrocution are ever present, especially in the experi-

mental circuitry found in the laboratory. We have had no fatalities, but an occasional localized third degree burn is not unusual.

One of the most pressing and important facets of the microwave problem which I should like to mention first is the psychological one. The practical importance of this in an industrial plant is worthy of extensive consideration, but perhaps a brief illustrative story will give you a better picture of how the human mind may distort the true facts in this "mysterious" realm of radar.

During World War II, nineteen microwave workers became the proud fathers of girl babies. As birth after birth with the same somewhat "monotonous" result occurred wild rumors began flying about the plant that men exposed to microwave radiation could father only female children. Inasmuch as the chief tube produced was the klystron, the exposed "victim" was said to have been "klystrated." The effect on morale was severe despite efforts to present statistics from other manufacturers that it was possible to work with radar and still have male offspring. The fact that there was evidence from animal experimentation that mice did not have a similar problem with radar seemed to do little to alleviate the general anxiety. Finally, the senior engineer of the microwave division came through for the company and with flying colors produced a "bouncing" baby boy. This helped the morale immensely and efficiency began to rise again. Although this may seem somewhat humorous, many man hours of work were lost and much serious mental distress was produced. The innate desire to spread rumors and the fear of anything relatively new and unexplored can still be a major problem in this field.

Perhaps the most disturbing thing about radar to engineers with precise orderly minds is the failure of mathematical calculations and formulas in determining field strength with radar. This problem is worse with proximity to the point of emission of the microwave and is commonly called the "near field" effect. This is also more confusing at certain wavelengths because "side lobes" of radiation seem to billow out around the main beam of microwave. Of course the reflection from nearby objects and the almost liquid flowing properties of this radiation in waveguides do not lend themselves to quick analysis. The only satisfactory answer we have been able to provide in this problem is actual measurement of the field in question. This will be discussed more completely later.

Effect on Eyes

We have instituted two cardinal methods of medical control for microwave and ionizing radiation. The first is a regular eye examination by an optometrist and the second is a complete blood count.

Over a five year period from January 1954 to January 1959, 215 employees (ages 22 to 57) were examined for a total of 430 individual eyes. The examination included visual acuity, fundoscopic inspection

and slit lamp examination with particular reference to the lens. It was given at the onset of exposure and at six month or yearly intervals.

Three hundred and sixty-nine eyes or 85.8 per cent were found to have completely normal clear lenses.

A second group of fifty-seven eyes or 13.3 per cent had changes of a non-suspect nature. This category is a questionable one and requires further explanation. Such things as spokes, vacuoles, bubbles and opacities not in the posterior sub-capsular area were included.

Because exact normal standards are not available this was an inexact grouping.

The third group was a definitely suspect category and consisted of four eyes or 0.93 per cent of the total number. These were all opacities in the posterior sub-capsular region. One eye of two individuals was involved and two eyes of one technical employee were affected. These three people were examined by an ophthalmologist who was particularly interested in ionizing radiation and its effect on the eyes. It was his feeling that the opacities in the patients with unilateral involvement were not due to radiation. He also was of the opinion that the bilateral opacities in the one individual might be due to radiation (presumably ionizing in nature). Careful evaluation of the radiation badges and dosimeters reported weekly showed a maximum exposure of 83 mr. The second highest reading was 55 mr. Most of the readings were from 0 to 10 mr. The tubes were operating at from 19 to 22 Kv. on the "L" band. The man made a practice at irregular intervals to look into the microwave output at a distance of about two feet in order to see the internal aspect of the instrument. Each observation lasted only a few seconds and it was prompted by a malfunction of the tube. The period of exposure was two and one half years, from 1953 to 1956 at which time a cover was put on the end of the tube output and the construction of the tube was changed. No radiation survey was done on this tube because this part of the program was not yet in effect. We have been unable to obtain a survey on a similar tube because this tube is no longer in production. The lenses were described as follows by the ophthalmologist.

Right Eye: Exhibited a few anterior sub-capsular vacuoles and moderate posterior sub-capsular opacities which are typical of radiation. The corrected vision was 20/30 plus 1 in December 1957. There was a deterioration to 20/60 minus 2, November 1958 with an increase noted in the cataract density and size.

Left Eye: Showed anterior vacuoles and posterior sub-capsular opacities less pronounced than the other eye but similar in nature.

There was only minimal progression of the lesions in the left eye. The vision was corrected to 20/20 at all times.

There was an additional history of contact with radar during

army service, 1943 to 1945 but the maximum exposure he admitted to was reading the radar "scope." He did not dismantle or service any of the units and was not knowingly exposed to any radiation. He is now thirty-seven years of age.

The significance of this case is far from clear. In view of the minimal badge and dosimeter readings it might be assumed that the necessary roentgens required for a radiation cataract² could not have been reached with this man. It is possible of course that he did not always wear his badge or actually exposed his eyes when the radiation did not hit the badge or dosimeter. It seems less probable that the effect might be due to microwave. No microwave exposure would have occurred under these conditions in a normally operating tube. Occasionally a malfunctioning tube will produce microwave which might have been in the region of the man's eyes, but this is unlikely and there is no way to be certain now.

At the present time we have suspended all but the initial eye examination on newly exposed personnel. We are preparing to participate in a larger study in the process of development under the Armed Forces Research Program with Colonel George M. Knauf and Dr. Conrad Berens. A large number of industrially exposed workers will be studied with more adequate controls. A specially trained ophthalmologist will conduct the examination program. We hope this will provide better information on normal lenses and shed some light on the potential of microwave to produce cataracts in the human.

Effect on Blood Count

The second aspect of our medical program is concerned with complete blood counts. Our schedule called for one or two counts at the onset of exposure to ionizing or microwave radiation followed by a repeat count every six months to a year. We had approximately 100 individuals listed at any one time for this purpose (from 1954 to 1959). This was not a controlled study but we could find no abnormal results that persisted on repetition of the count or that could not be explained on the basis of a known illness or disorder. It was felt that this was in conformity with other controlled studies.^{3, 4} We discontinued all regular blood counts in January 1959 and we do not plan to reinstate this part of our medical program for microwave exposure. We would consider further complete blood counts if the level of ionizing radiation exposure is found to be significantly higher in the future than the minimal levels currently being monitored. It has been extremely unusual to find any dosimeter or badge readings over 100 mr. in a 40 hour week. This is far below the level necessary to change a blood count. We do not feel it is necessary or practical to screen for blood dyscrasias prior to exposure to low doses of ionizing radiation.

SAFETY PRECAUTIONS

For the second part of the discussion, I will consider the safety controls in regard to microwave energy.

We have a written and widely distributed Company policy out-

lining safety requirements. This is distinct and separate from our policy on ionizing radiation which is mostly directed by Federal and State Laws and A.E.C. regulations. Unfortunately, there is no law to guide us specifically in the microwave field. In view of this we have taken our guidance from the experiences of the Armed Forces and other large industrial organizations.

Ten milliwatts/cm² has been the standard maximum permissible level adopted. Without entering into a prolonged discussion as to why we chose this level, I would like to state that we have found it fairly easy to maintain and we feel secure with it for whole or partial body radiation. We do not correct or modify this level in any way because of the pulsation of the energy or the duty cycle used. Any over exposure must be reported to the Medical and Safety Department.

We advise our personnel not to rely on heat sensation as a warning signal of exposure because of its unreliability at certain wavelengths and under cool and windy climatic conditions.

When a source of microwave is used in the confines of a building we insist that the energy be dissipated in a "dummy" water load whenever possible. If it must be radiated inside a structure, we advise it being directed to an exterior wall and upward. When an antenna check must be made and a water load cannot be used the area must be completely enclosed with absorbent screening material. The Safety Department is delegated to examine and measure the adequacy of shielding.

No person is permitted to look down a travelling waveguide. This formerly was a very common procedure and many of the older engineers will remark privately that they previously did this and often noticed heat or a warm sensation in the area of the face and eyes. Needless to say, this is a very important regulation and we feel it should be enforced without exception.

Special requirements for outside radiation protection will be discussed subsequently.

Field Intensity Measurements

One of the key factors to our approach to the microwave problem is the use of a field density meter. The Sperry Microwave Electronics Company of Clearwater, Florida, has produced a commercially available model which we have found satisfactory.

The instrument is a self-contained, portable and battery powered unit. The dial is set at zero reading outside the field before using. The center of the dial represents 10 milliwatts/cm² and the left of the scale is in the "safe" range and the right is in the "dangerous" range. The readings are in decibels plus or minus center standard of field density. We have this type of detector for the "C" and "S" bands now. All the usual bands should be available in the near future, but different horns must be used for each. It should be noted that this

is not a dosimeter. It records field strength by means of a minute barium compound thermistor which has a very rapid response time. There is automatic correction for ambient temperatures. The instrument must be polarized correctly by manually turning in the field until a maximum reading is obtained. We usually walk across the field with the radiating unit fixed in a position near ground level. Neon tubes may be used to locate the beam but are not very reliable as quantitative detectors. The most powerful and narrowest beam is called the "acquisition" beam. The broader lower powered beam is the "guidance" beam. These are usually distinguishable although they are close together or superimposed.

Frequently there are side lobe projections of radiation extending out from the main beam that are peculiar to each unit. These must be mapped out for each instrument. Side lobes are very much less powerful than the main beams and are generally not a problem from the safety standpoint unless the instrument is malfunctioning.

Protective Equipment

Various protective devices are utilized in our safety program. We have obtained a metal impregnated nylon cloth suit with a similarly treated nylon mesh head cover. This is used chiefly when surveying new units in order to determine the safe distance in various directions (where the field intensity is likely to be above the approved level). This clothing is not utilized as yet for routine day to day purposes. Our engineers and safety personnel have crudely tested the efficiency of the material in screening out microwave and have found it satisfactory.

We make extensive use of absorbent materials in our indoor test areas. This is especially necessary in the antenna testing operation where the energy cannot be dissipated into a "dummy" water load. The specific material now in use is animal hair impregnated with a rubber and graphite mixture. Canvas or linsn has also been similarly treated with rubber and graphite and was formerly widely used. The amount of protection is dependent on the usual factors of power, distance to the antenna, wavelength and thickness of the absorbing material. We check the effectiveness of protection with a density meter in each case.

Many of our installations are in densely populated communities especially in the New York area. Because of this we have been forced to make broad use of screening materials and automatic cut-off devices for outside radar units.

The screening is generally wire mesh and it is constructed and placed according to rather exact mathematical calculations. The primary purpose of the mesh is reflection of the energy. A small amount of radiation is transmitted through the mesh and a small quantity is absorbed by the metal. The necessary fineness of the mesh can be calculated depending on the wavelength, the distance from the antenna and the diameter of the wire. We always test the actual screening effect with a field density meter. There is a certain degree of dif-

fraction of microwave behind the mesh. This varies with the wavelength but must be considered in shielding an area. It often requires a higher screen to be constructed than would be expected on a "line of sight" basis. The wire mesh usually is angled backward in order to reflect the microwave harmlessly upward. If this is not done the reflected energy could damage the antenna or the personnel in the area. The space between the wire mesh and the antenna is fenced with inter-lock gates to keep out all personnel. Galvanized coating is used to prevent arcing between the loosely connected wires of a mesh. A simple arrangement consisting of 3/8 inch or 1/2 inch galvanized wire mesh draped doubly over a light wood frame can be used for temporary or portable protection.

The second outdoor protective device is an automatic gear sector control. This usually consists of a cam arrangement which cuts off the power when the beam is pointing toward nearby areas where personnel may be working or there is an uncontrolled outside zone. Without exception, these devices must be practically fool-proof. Often a parallel series of three successive circuits are used, each checking the others. A "fail-safe" arrangement is also used in case all three circuits fail. This would then cut-off all power to the unit. Again it should be emphasized that a survey meter should be used freely to check the actual conditions. It is possible for instance, to have a reflection of microwave off the yoke or metal frame of the unit exceeding the 10 milliwatt level.

CONCLUSION

This then completes the review of our program. We feel it is adequate in the light of present knowledge, but looking only a short distance into the future at the larger more powerful radar units to come, we are compelled to continue to be very cautious.

respect to the microwave source failed to demonstrate any interface effect as an explanation for the posterior cortex of the lens being the typical site of microwave-induced opacities. It was also shown that the distance of the eye from the microwave source was not in itself a factor of significance in our results.

Although not directly related to our experiments on the eye, it has been shown that microwave affects differentiation but not growth in the breadmold *Neurospora crassa* and that this result apparently cannot be attributed to a thermal influence.

METHODS AND INSTRUMENTATION

During the past year, we have been concerned chiefly with three general questions which previously reported experiments had raised. These questions have to do with:

1. The cumulative effects of repeated subthreshold exposures of the rabbit eye to microwave radiation.
2. A comparison of the effects on the eye of continuous wave and of pulsed wave radiation.
3. The possibility that microwave radiation may exert a non-thermal effect on tissues in addition to the thermal effect demonstrated by various workers, including ourselves. We have conducted a variety of experiments under this heading.

Our investigative group has again consisted of Mr. David Biddle, Mrs. Claire Van Ummersen and myself. In addition, during the year we were fortunate to have with us for different periods over several months two ophthalmologists, Dr. Arthur Leith and Dr. Daniel Weiss, both of whom pursued certain aspects of this work.

The eye offers several advantages for assessing the biological effects of radiation. The following may be noted:

1. It is one of the few organs which in the living intact animal can be exposed to radiation directly rather than through intervening skin and varying amounts of adipose tissue.
2. The crystalline lens of the eye has been shown to be peculiarly susceptible to the effects of radiated energy, whether ionizing, infrared or radio-frequency, all of which cause the development of opacities (cataracts) in this normally transparent optical body.
3. These opacities can be identified promptly and their further development followed in the living animal by ophthalmoscopic or slit-lamp examination without the necessity of anesthesia. The non-irradiated left eye serves as a control.

STUDIES ON THE EFFECTS OF 2450 MEGACYCLE RADIATION ON THE EYE OF THE RABBIT

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SUMMARY.

Exposure of the rabbit eye to 2450 Mc. microwave causes development of lenticular opacities (cataracts) under experimental conditions as follows:

1. With continuous wave radiation, the higher the power density, the shorter the duration of a single exposure period which can cause an opacity. Conversely, the lower the power density, the longer the single exposure period required to cause opacities.
2. There is a cumulative effect of repeated exposures of the eye to continuous wave radiation at power densities and for times which in single exposure periods cause no apparent damage. The lowest power with which we have induced a lens opacity in this manner has been 80 mw. /cm.^2 applied for one hour daily for a total of 19 times.
3. With pulsed microwave, opacities may result from a single one hour exposure to radiation at an average power density as low as 80 mw. /cm.^2 but with peak power of 400 mw. /cm.^2
4. Ocular temperature, as measured in the vitreous body at the posterior pole of the lens, exhibits a rise of only 4 degrees C. during a one hour period of irradiation at an average power density of 80 mw. /cm.^2 . We question whether the resulting opacity may properly be termed a thermal effect.
5. Exposure to microwave radiation at powers sufficient to cause cataracts seems not to cause any particular discomfort to the animal and such exposures can be carried out without anesthesia.

Although cataracts induced by microwave radiation and those caused by ionizing radiation are similar in several respects, microwave does not act in any complementary manner to shorten the latent period for formation of X-ray induced cataracts.

Rabbit eyes were exposed to infrared radiation sufficient to induce a temperature rise in the vitreous body comparable to that caused by a cataractogenic exposure to microwave. In contrast to microwave-induced opacities, those caused by infrared radiation occurred in the anterior cortex of the lens.

Experiments in which the orientation of the eye was reversed with

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We use the rabbit as our experimental subject for several reasons. Much of the previous work on the effects of various kinds of radiation on the eye has been done on this animal. The diameter of the rabbit eye is approximately three-fourths and its volume approximately one-third that of the human eye. Body temperature of the rabbit, measured rectally, is 38.7°C . (101.6°F .), compared to the human normal rectal temperature of 37.5°C . (99.6°F .). Finally, and of some practical consideration, pure bred strains of this animal are available usually in good supply and it is easily maintained under laboratory conditions. We use in our experiments rabbits of the New Zealand White strain, aged 6 to 10 weeks.

Our microwave source was designed and made for us by the Raytheon Manufacturing Company. It is based on their Model CMD4 Microtherm, with additional circuitry to provide either continuous or pulsed wave at a frequency of 2450 Mc. and wave length of 12.3 cm. The output can be pulsed at duty cycles of 0.5 per cent to 66 per cent. Pulse width can be varied between 50 microseconds and 2000 microseconds and pulse repetition rate between 140 and 2200 per second. The antenna is the Microtherm Director "C", which is a corner reflector type. The instrument is powered from a voltage stabilized line.

A directional coupler in the coaxial cable to the antenna permits leading off a small known fraction of the power output to a Hewlett-Packard No. 430C microwave power meter and thus provides for constant monitoring of power. An S-band silicon microwave diode mounted on the back wall of the exposure chamber is connected to a Tektronix oscilloscope for checking the pulse width, shape and amplitude.

All exposures are carried out in a chamber 36 by 34 by 20 inches which is lined by microwave absorbent material (Sponge Rubber Products Company Type 1, 12 cm., absorbent). The animal is placed in this anechoic chamber in a copper-lined wooden box, with only its head exposed to the microwave field. The corneal surface of the eye is positioned exactly opposite the dipole crossover of the antenna and two inches distant from the surface of the plastic housing which covers the dipole.

Measurements of the power density of the microwave field are made calorimetrically in the anechoic chamber at the position of the rabbit eye, with the rabbit box in place. The maximal output of our microwave generator yields a power density of 400 mw./cm.^2 at the 2 inch position of the eye.

Changes in the temperature of the eye during irradiation are measured by means of hypodermic needle thermistor probes of 22 or 24 gauge connected to a balanced bridge circuit which feeds into a four channel strip-chart recorder. For body temperatures, a thermistor enclosed in vinyl plastic at the end of a one-eighth inch vinyl

plastic covered lead is inserted in the animal's rectum. Accurate temperature records can thus be obtained for the entire period of irradiation, as well as before and after.

TIME AND POWER THRESHOLDS FOR INDUCTION OF LENS OPACITIES BY CONTINUOUS WAVE RADIATION AT 2450 MC.

We had previously reported for each of a series of different power densities the minimal single exposure period which would cause an opacity to form in the lens. Although the graph expressing these time and power thresholds was based on 56 cases, we felt that more experiments should be performed for the lower power densities, those below 280 mw./cm.² We have therefore extended our series of acute exposures to 136 cases. These form the basis for Figure 1, in which the duration of single exposures is plotted against the applied power density. Black circles represent opacities; white circles indicate no effect. The chief difference between this and our previously reported data consists of a shortening of the time thresholds for powers below 200 mw./cm.² The broken line represents a projection which may or may not be valid for power densities below 120 mw./cm.²

CUMULATIVE EFFECTS OF REPEATED SUBTHRESHOLD EXPOSURE PERIODS.

We reported a year ago at the Rome meetings that at a power of 280 mw./cm.² lens opacities developed as the result of repeated exposures of relatively brief duration, any single one of which was not sufficient to cause an opacity. The minimal exposure we employed was 3 minutes. The shortest single exposure period which has proven to be cataractogenic at this power is 5 minutes.

We can now report that exposures of 3 minutes made daily for five successive days caused opacities in every one of five cases. The same results were obtained in five cases in which the eye was exposed to radiation three times for 3 minutes each time but with the exposures being made every fourth day. When the interval between the 3 minute exposure periods was increased to a week, however, the lens remained unaffected after five successive exposures in every one of the five experiments done.

These results suggest that if the cataractogenic factor of microwave radiation is one that initiates a chain of events in the lens, the visible and end result of which is an opacity, then this factor must initially require an adequate power density acting for a sufficient duration of time in order to start the chain of events. If either the power density or the duration are less than a certain threshold value, then the damage done to the lens is not irreparable and recovery can take place, provided sufficient time elapses before a subsequent similar insult. In the experiments cited above, it appears that the interval necessary for recovery after the damage done by a 3 minute exposure at 280 mw./cm.² must be greater than four days but need not be longer than one

week.

It must be emphasized that this statement applies only to the conditions described, namely, a 3 minute exposure at 280 mw. /cm.^2 . We have previously shown that if the exposure period is 4 minutes at this power density, then lens opacities may result from two exposures given a week apart or from two or three exposures separated by two-week intervals. We have not yet determined what must be the requisite recovery period following a 4 minute exposure at this power level.

We referred above to "the cataractogenic factor of microwave radiation". What this factor may be, we are not prepared to state. It has been quite generally assumed that the effect of microwave radiation on living tissues is entirely a thermal one, an effect of the heat that results from absorption of R-F energy by the tissue. It is certainly true that localized heating occurs as a result of absorption of microwave energy. Yet one is reminded that thermal effects usually occur at or above a specific temperature (e.g., melting point, boiling point, flash point) and not as the result of intermittent excursions to temperatures below critical value. It is possible to conceive of a thermal effect which may be the result of either a high temperature for a short time or a lower temperature for a longer time, but it is difficult to imagine the same effect resulting from a low temperature occurring for a short time but repeated at widely separated intervals.

At the power density of 280 mw. /cm.^2 , the shortest single exposure period which will cause a lens opacity is 5 minutes, at which time the temperature of the vitreous body at the posterior pole of the lens has reached 49.3° C. At the end of a 3 minute exposure period, however, this temperature is only 47.2° C. Inasmuch as this duration of exposure if repeated at four day intervals causes a lens opacity to form but if repeated at weekly intervals has no effect, then one can support a "thermal effect" viewpoint only by assuming that a lesser temperature increase may be cataractogenic if it occurs frequently enough. This argument collapses of its own weight, for if we continue to reduce the temperature and increase the frequency of its occurrence, we eventually arrive at the constant normal body temperature, which obviously is not a cause of cataract.

A cumulative thermal effect as the cause of microwave-induced opacities becomes even less probable in the light of further experiments we can report. Recognizing the valid criticism that 280 mw. /cm.^2 is still, from the biological viewpoint, a fairly high power density and that a vitreous temperature of 47.2° C. , even though occurring ever so briefly, is nevertheless well outside the range of a rabbit's normal or even its pathological variation, we undertook experiments in which the eye would be exposed repeatedly to radiation at relatively low power densities.

Having ascertained that at a power density of 120 mw. /cm.^2 the minimal single exposure period necessary to induce lens opacities was 35 minutes, we exposed eyes at this power level for periods of 30

minutes repeated at two day intervals. Of the four experiments which have been completed, lens opacities developed after two such exposures in one case and after three exposures in three cases. In a rabbit under sodium Nembutal anesthesia, the temperature of the vitreous body at the end of 30 minutes of irradiation at this power density is 44° C.

A point worth mentioning with regard to these experiments is that at this power level the animals remain quiet in the microwave field without anesthesia. In the four experiments, two animals were irradiated without anesthesia. In these two cases, therefore, we can be sure that the heat dissipating vascular system was not functionally affected by an anesthetic and that the ocular temperature was surely not higher than 44° C. and was probably less than that. The animals did not appear to be experiencing any discomfort whatsoever.

We are now extending this series of experiments to include daily exposures of the eye to one hour of continuous wave radiation with the power density reduced to 80 mw./cm.^2 and in some cases to 40 mw./cm.^2 . No anesthetic is used but otherwise the animals are under conditions identical to those of other experiments, except that a small lucite neck yoke prevents backward movement of the head. This may well be a superfluous precaution, for in nearly every case the animal appears content to remain in the box without moving. Judging from its behavior, if it experiences any sensation from having its head in the microwave field, the sensation is not one of pain or even of discomfort.

Of the three experiments attempted so far at this 80 mw./cm.^2 level, one animal died after four exposures and another after nine. We have not ascribed these deaths to radiation, for in both instances the exposures were being done during the lunch hour and although the power was turned off automatically at the end of the exposure period, the animals were inadvertently left in the box for a considerably longer time. Rabbits suffer no ill effects from being restrained in this manner for an hour but with much longer periods they appear to accumulate excess mucus in their respiratory tracts, causing choking.

The third animal was irradiated for an hour each day for five consecutive days each week, this schedule being maintained until a total of 19 hours of irradiation had been given. At this time, slit-lamp and ophthalmoscopic examinations were negative: the lens was clear and without sign of opacity. Examined 13 days later, however, the irradiated eye showed a well developed cataract in the posterior cortex. The non-irradiated eye was normal.

It is of interest to note that in the anesthetized animal, one hour of exposure to continuous wave radiation at this power raises the temperature of the vitreous body to 42.8° C., which is only 4.1° above body temperature. If further experiments at the 80 mw./cm.^2 power level also result in cataracts, we shall have to conclude that if they are induced as a thermal effect of microwave radiation, then it

is a thermal effect requiring neither a critical temperature nor even a marked elevation of temperature in the tissue.

EFFECTS OF PULSED MICROWAVE RADIATION.

At last year's Tri-Service Conference, we reported on a preliminary series of experiments testing the effect on the eye of pulsed microwave at 2450 Mc. With pulsed radiation, the eye can be subjected to rapidly repeated peaks of high microwave power while the average power during the exposure period remains relatively low. Inasmuch as the thermal flux is identified with the average power alone, we hoped in this manner to discover whether microwave energy may exert other than a thermal effect.

We reported on the first 16 of our initial group of 25 experiments then in progress. In these experiments, the eye was exposed to pulsed microwave radiation at an average power density of 140 mw./cm.^2 , with pulse peaks of either 280 or 560 mw./cm.^2 . The duty cycle we employed was therefore 50% or 25%, the latter being the lowest duty cycle we could obtain with the equipment then being used. In 62 per cent of the experiments, lens opacities resulted from exposure periods and associated ocular temperatures - which were significantly less than those required for induction of opacities by continuous wave radiation of identical average power. We therefore suggested that the cataractogenic effect of microwave radiation might not be primarily a thermal one and we advised giving attention to peak powers of the microwave field when assessing the possibility of hazards to personnel.

Further experiments along this line promised to be fruitful, particularly if we could employ pulsed wave having a greater disparity between peak power and average power. This would demand much lower duty cycles than we were then able to obtain. We therefore had our equipment redesigned to allow duty cycles as low as 0.5 per cent. With this equipment, we irradiated 15 animals, using average power densities ranging from 120 mw./cm.^2 down to 40 mw./cm.^2 and with accompanying peak powers of 400 mw./cm.^2 up to 800 mw./cm.^2 . Under these conditions, opacities developed in 53 per cent of the experiments.

For example, opacities occurred after 60 minute or longer exposures of the eye to pulsed microwave when the average power density was only 80 mw./cm.^2 but the peak power was 400 mw./cm.^2 . A 45 minute exposure had no effect. We consider it to be significant that at the end of a one hour exposure period at this 80 mw./cm.^2 power, the temperature within the eye has risen to 42.8°C. , which is only 4 degrees above body temperature. Also significant is the fact that this same power has no effect when applied as continuous wave radiation for a one hour period. Indeed, as we have already related in this report, daily exposure periods of this power and duration have had a cumulative effect on the eye only after 19 such periods. These results point to

the peak power as being an important factor in causing opacities to develop when the eye is exposed to pulsed microwave.

In order to compare the new pulsing circuit with the former one, with respect to biological effects, we exposed 24 rabbits under conditions similar to those which obtained in our original group of 25 experiments (50 per cent duty cycle, 140 mw./cm.² average power, 280 mw./cm.² peak power, 20 minute exposure period). Opacities developed in only 21 per cent of the animals, compared to 53 per cent in the original group. This discrepancy appears a little too great to be ascribed to the variability of biological material and we are still perplexed by this difference in response. It seems unlikely that the strain of rabbits has undergone a change and we can detect no change in either our exposure chamber or our antenna. Indeed, calorimetric measurements show that there is no difference in the applied power density in the two groups of experiments. It may be that when the redesigned equipment is used at a high duty cycle, such as 50 per cent, there is a change in the characteristics of the Magnetron tube, so that the frequency spectrum is broadened and only a portion of it has any effect on the lens. On the other hand, perhaps in our original circuit there occurred a frequency deviation which enhanced the cataractogenic effect of pulsed waves. We hope to clarify the situation by further experiments.

NON-COMPLEMENTARY EFFECTS OF MICROWAVE RADIATION AND X-RAY.

Lens opacities induced by microwave radiation and those caused by ionizing radiation are similar in several respects. Typically, both develop in the posterior subcapsular cortex, changes occur initially in the region of the posterior suture, and frequently the opacity takes the form of striate masses concentric with the equator, which later migrate axialward to form ring-shaped cataracts. One marked difference is with respect to their latent periods. Cogan and Donaldson have shown that after single doses of 1200 to 1500 r. of X-ray at 1500 kv., opacities develop in the lens after 25 to 30 days.¹ In contrast, we found that following exposure to microwave radiation, opacities appeared after latent periods of 1 to 8 days, the average time being 3 1/2 days.

It seemed of interest to test whether these two types of radiation could complement each other in their cataractogenic effects. Experiments were undertaken by Dr. Weiss. Through the cooperation of the Radiology Department of the Massachusetts General Hospital, the eyes in 22 rabbits were exposed to X-ray of 2000 kv. Both eyes received equal doses of 1500 r., in all but two cases given at the rate of 100r./min. Either 24 hours or one week later, the right eye was exposed to a single subthreshold dose of microwave radiation (280 or 352 mw./cm.² for 3 1/2 minutes). In a few cases, the microwave exposure was a few hours before the X-ray. In two cases, the 1500 r. dose was given at the rate of 140 r./min. and the animal received microwave radiation twice - one week later and 11 days later.

In all of the above experiments, opacities appeared in both eyes at the same time and were of similar degree. Under the conditions of the experiment, therefore, microwave radiation did not act in any complementary or supplementary manner to shorten the latent period for formation of X-ray induced opacities nor did it affect the extent of the damage to the lens by X-irradiation.

COMPARISON OF OPACITIES RESULTING FROM MICROWAVE AND FROM INFRARED RADIATION.

If the opacities formed in the posterior cortex of the lens are solely a thermogenic effect of microwave radiation and therefore are purely a result of hyperpyrexia, then it should be expected that any influence causing a comparable rise in ocular temperature would produce a comparable result. As Hartman states: "The physiological and pathological reactions to hyperpyrexia alone induced by whatever mechanisms, such as hot baths, heated cabinets, diathermy, heat stroke, thermal burns and microwaves, are essentially comparable."² Imig plotted temperature gradients induced by 12.25 cm. microwaves in excised beef eyes and found that the point of maximum temperature was in the vitreous body at the posterior surface of the lens and hence agreed with the site of damage.³

In our laboratory, Dr. Leith undertook to study the effect of increasing the ocular temperature by means other than microwave radiation. The beam from a 4.5^{mm} carbon arc source, passed through an infrared filter transmitting only wavelengths from 1000 to 3000 millimicra, was focused either on the anterior or on the posterior capsule of the lens in the anesthetized animal.

With focus on the anterior capsule and a 6 minute exposure, the cornea was severely burned, with subsequent ulceration, scarring and vascularization. Although the lens was thereby almost obscured, it remained possible to identify an opacity on or beneath the anterior capsule. In most of the experiments, focus was on the posterior capsule, so that the cornea was spared from burn and suffered only a transient clouding. With duration of exposure either 5 or 10 minutes, opacities resulted in every case but they were always located in the anterior cortex of the lens, despite the infrared being concentrated at the posterior capsule.

These results are in agreement with various other observations on cataracts caused by infrared radiation. The opacities occur typically in the anterior cortex and in this respect differ from those caused by either microwave or ionizing radiation.

STUDY OF POSSIBLE INTERFACE EFFECT AT LENS-VITREOUS BODY BOUNDARY.

Several suggestions have been offered to explain why opacities induced by microwave occur typically in the posterior cortex of the lens, just beneath the capsule. One suggestion was that there might be an interface effect at the lens cortex-posterior capsule boundary or at the capsule-vitreous body boundary, with resulting reflection of power to cause a concentration in the posterior cortex. If so, the temperature might well be higher in the lens cortex than in the vitreous body just behind the lens, which is where our ocular temperature measurements are usually made. We therefore performed the following experiment:

In an anesthetized animal, a suture was placed around the tendon of the inferior rectus muscle at its insertion on the eyeball. Traction on the suture rolled the eye upward to an extent such that the cornea disappeared behind the superior orbital margin and the inferior surface of the sclera was presented between the open eyelids. With this surface positioned two inches from the antenna, the eye was irradiated for 12 minutes at 280 mw./cm.^2 power density. The orientation of the lens with respect to the microwave source was now reversed; the power had to pass through sclera, vitreous body and posterior capsule before reaching the lens cortex - an approach from the rear, so to speak. If an interface effect should in fact exist, we reasoned that the changed orientation might so alter it as to affect the results.

Opacities appeared as usual in the posterior subcapsular cortex in all three surviving cases. We conclude that if there is an interface effect, it is not a critical factor with respect to the site of the opacity.

EFFECT OF DISTANCE OF EYE FROM MICROWAVE SOURCE.

It was also suggested that at 12.3 cm. wave length, the 2 inch distance at which we usually position the eye for irradiation may be an important factor, perhaps introducing the effect of a half wave length distance between the antenna and the lens and so promoting creation of standing waves. The distance from the dipole to the cornea is 6 cm. and from the cornea to the posterior lens surface is 8 mm., making a total of 6.8 cm.

To test whether this particular distance has significance, we irradiated eyes in 11 rabbits for 15 minutes at 240 mw./cm.^2 power. In six of these cases, the distance to the eye was increased from two to three inches but the power density at the position of the eye was kept constant at 240 mw./cm.^2 . In all 11 cases, the same type of opacity appeared in the posterior lens cortex. A similar series of experiments but with a power of 220 mw./cm.^2 has given further evidence that the two inch distance is not of itself significant in the induction of opacities by 12.3 cm. microwaves.

INHIBITORY EFFECT OF MICROWAVE RADIATION ON DIFFERENTIATION IN NEUROSPORA.

Bearing upon the question of possible non-thermal effects of radiation at this frequency are experiments performed in our laboratory by two senior students, Mr. John Banas and Mr. Robert Coli. They grew the breadmold *Neurospora crassa* on suitable culture media and then transferred small patches of the mycelia with conidiophores (fruiting bodies) to lusteroid test tubes. These were subjected to continuous wave radiation at 2450 Mc., with a power density of 400 mw./cm.², for periods of 5, 10 or 30 minutes. Spores from the irradiated fruiting bodies were then inoculated on agar slants. These spores germinated and grew but the cultures did not form fruiting bodies. Control cultures grown from non-irradiated spores developed fruiting bodies in the normal manner. It therefore appeared that microwave radiation had exerted an inhibitory effect on differentiation while not at the same time affecting growth. This finding parallels observations on the effect of microwave radiation on the developing chick embryo, as reported from our laboratory previously by Van Ummersen.

Air temperature in the test tube during irradiation of the *Neurospora* conidiophores was found to increase during 30 minutes to a maximum of 35.8° C. During a 10 minute exposure, air temperature reached 31.8°. To test whether the observed effect on differentiation was perhaps a thermal one, similar preparations were placed in an incubator for 10 minutes at 31.8° and the spores were then inoculated on agar slants. In all cases, these cultures not only exhibited normal growth but also produced fruiting bodies. It does not seem plausible, therefore, to ascribe failure to differentiate fruiting bodies to a thermal influence of microwave radiation.

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Power density
mw./cm.²

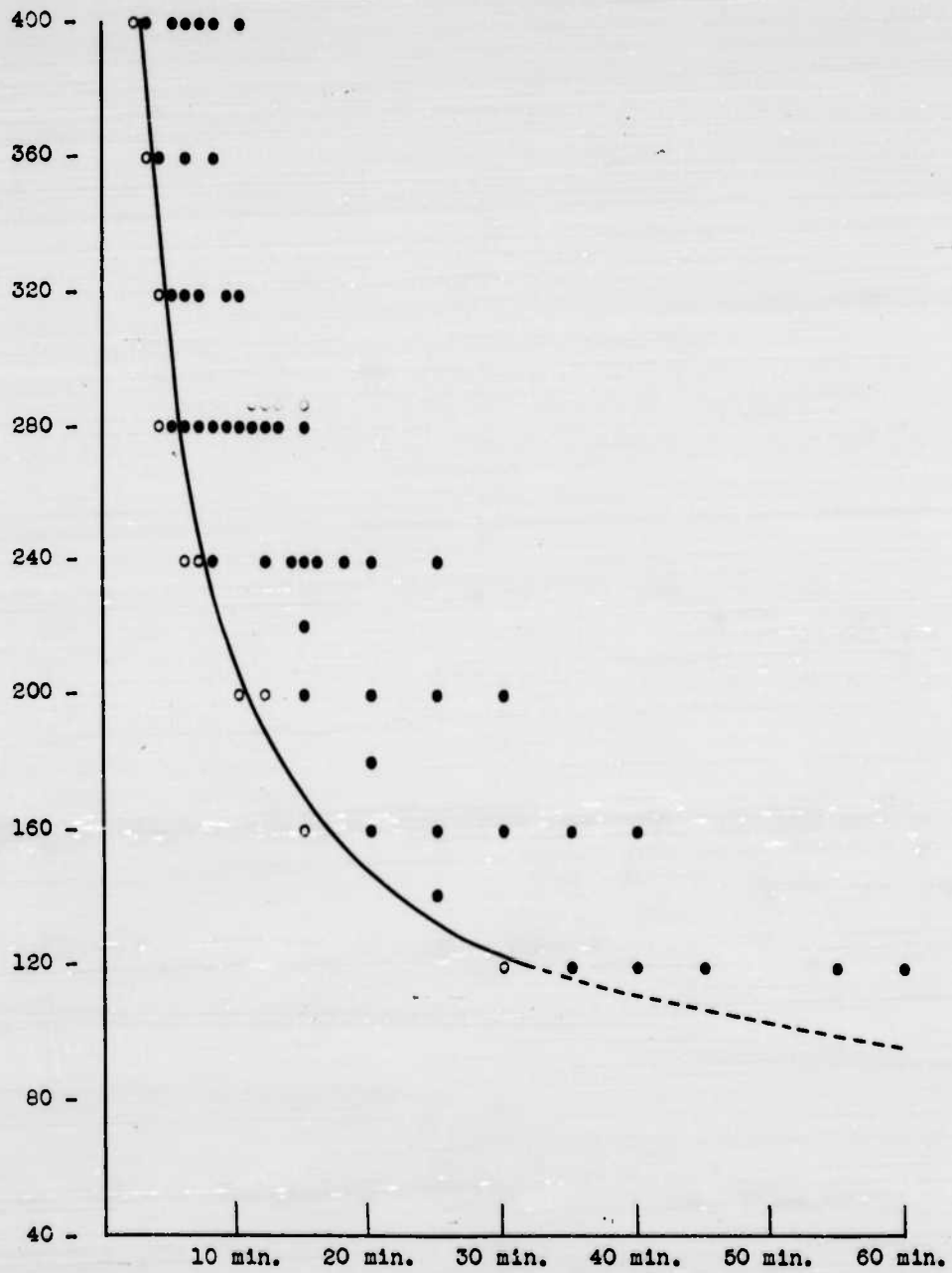


Figure 1. Time and Power Thresholds for the Production of Lenticular Opacities in Rabbits Exposed to 12.3 Cm. Microwaves.

BIOLOGICAL EFFECTS OF MICROWAVE RADIATION
WITH LIMITED BODY HEATING[#]

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Abstract

Studies of the biological effects of microwave radiation being conducted in the Biophysics Program of Tulane University are primarily concerned with effects not involving overall elevation of body temperature. Studies are centered about the determination of the causal relationships applicable to the physiological and pathological changes observed and the interpretation of data collected with animals as subjects in relation to microwave absorption by human subjects.

Results obtained in the course of these investigations indicate that most of the effects observed are explainable as heating effects of a special kind. In particular, microwaves are generally more penetrating to tissue than are infrared and visible radiation and the penetration is dependent upon the particular tissue combination being irradiated.

Effects on growth previously asserted at a low power density (10 mw/cm^2) have been discounted due to results recently obtained.

Present work includes calculations of reflection, penetration, and temperature distribution for various microwave wavelengths and the relation of temperature calculated, and inferred by measurement, to observe neural and other physiological effects. Measurements of complex dielectric constants of tissue components as a function of field strength are to be completed.

[#] This research was supported by Office of Naval Research Contract Nonr-475(03) to the Biophysics Laboratory, Tulane University.

BIOLOGICAL EFFECTS OF MICROWAVE RADIATION WITH LIMITED BODY HEATING

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1.0 INTRODUCTION

The purpose of the present paper is to introduce the microwave effects work conducted through the Biophysics Program of Tulane University, and to describe a simplified procedure for computing the distribution of absorbed energy at various microwave wavelengths. The present work is largely confined to investigations of biological effects associated with limited overall body heating. A further purpose of the work is to relate observations to possible effects on humans.

In the opening phases of the microwave effects studies in 1954, measurements of the physical parameters related to the absorption of microwaves by biological tissues including the complex dielectric constants of various tissues, power densities, and temperatures, were initiated. In addition, a technique for microwave scattering studies was developed and later employed.

Concurrently, gross pathological studies were conducted using mice with 3 cm. and 10 cm. equipment. Subsequently, an experimental design was completed for a long term histopathological survey using mice.

In these animal experiments with microwaves, mice were exposed to various frequencies, power levels, and ratios of peak to average power, and biological effects were observed. Subsequent experiments were conducted to correlate these effects using large groups of animals. A major portion of this work, consisting of various biological assays, has been described previously. At low exposure levels, results suggested a change in growth due to irradiation.

During the past year the growth change at low exposure rates has been studied using mice in an intermittent exposure environment similar to that to be expected with scanning radars. The level of exposure used is the maximum safe level presently estimated for man (10 mw/cm^2). Data were obtained from whole body and tissue wet and dry weight measurements.

As previously reported, a five minute exposure at a somewhat greater power density (45 mw/cm^2) causes growth rate changes and blood plasma

volume shifts in mice, and longer exposures (e g. ten minutes) cause death. More recently, studies have been conducted at this power density using cold-blooded animals from which heat is removed during irradiation by control of the environmental temperature to avoid gross effects related to high body temperature. With such cooling lizards can tolerate two hours or more of exposure.

In the last year, an investigation of neural effects of microwaves was initiated. Irradiation of isolated nerves and whole body irradiation comprised the initial phase of the experimental programs. A survey to indicate the relative usefulness of measuring the steady potentials of the central nervous system as an indicator of neural stimulation was conducted. Studies of mechanisms related to possible neural effects involved an investigation of the rate of change of sodium ion transport due to microwave absorption. More recent studies include power measurements in the central nervous system and localized irradiation of exposed peripheral nerve in situ.

To provide a basis for further interpretation of localized irradiation experiments, data have been collected from the work of other investigators and reflection coefficient and depth of penetration calculations have been completed using three microwave wavelengths and the near and far infrared.

Nonlinear dielectric effects have been suggested by Schwan. During the past year, the possibility of dielectric saturation of complex protein molecules by microwaves has been explored by exposing various protein complexes to high intensity fields, and assaying the results by use of paper electrophoresis techniques. The results of these experiments are inconclusive; no alteration of the protein molecule independent of heat has been demonstrated.

An analysis of this phenomenon suggests that the experiments performed offered little chance of producing the expected results, and that, in fact, a different approach to the problem should be undertaken. It is now proposed that measurements be made of the change in the complex dielectric constant of protein complexes as a function of field strength, using various frequencies. The data obtained should indicate the field strength necessary for saturation of given tissues and frequencies, and hence for denaturization since dielectric saturation must occur at the threshold of non-thermal denaturization.

2.0 GROWTH RATES FOR LOW LEVELS OF MICROWAVE EXPOSURE USING 10 CM. EQUIPMENT

2.1 General

Alterations in the growth rate of mice have been attributed to 3 cm. irradiation at 45 milliwatts per square centimeter using single 5 minute exposures. Such exposures cause gross overall changes in the body temperature of mice.

Previous studies with 10 cm. microwaves suggest that the growth rate of mice (CF-1 female) may be modified by intermittent irradiation for two minute periods at a power density of 10 milliwatts/cm².¹ Since this power density is the arbitrary maximum established for man, particular importance is attached to any effect observed. Furthermore, there is but a relatively small gross body temperature rise associated with this level of irradiation, and thus any pathological effects observed may be labeled as "non-thermal".

These earlier studies, suggesting increases in growth rate and ultimate weight, showed no definite concurrent pathological changes. Studies of tissue blood plasma volume on another group of animals irradiated with 3 cm. microwaves at a somewhat greater power density indicated shifts in body fluids¹, and, although these particular animals lost weight, the results suggested that fluid shifts and local edema might account for weight changes, whether positive or negative.

A major purpose of the growth study presented in the current report is to compare average weight changes as a function of time within a control group with such changes for an irradiated group. In addition, the reported work indicates the relationship between dry tissue weights and water content for various tissues as well as for the whole animal. The reported work consists of two experiments, the second designed on the basis of results from the first.

2.2 Method - Experiment A

The experimental animal used is the CF-1 female, as in the previous study. The CF-1 mice are obtained from Carworth Farms, and are separated into two groups of 101 mice². The mice are maintained for five days following arrival to allow for re-hydration and acclimation to environment. A separate cage is used for each of the two groups of mice.

Irradiation of the animals in one of the two cages is initiated at $t = 0$ (for interpretation of the illustrations which follow). The period of each

exposure is two minutes and, by use of an automatic timer, one exposure per hour is provided during the 21 days of the experiment. The entire populations are weighed at selected intervals, and, in particular, at $t = 0, 2, 7, 10, 14, 17,$ and 21 days.

Five irradiated and five control animals are taken from various regions of their respective cages at $t = 0, 1, 2, 3, 7, 14,$ and 21 days after the total weight of the animals in the cage is determined. Random selection is attempted. The selected animals are weighed individually, sacrificed, and dissected. The kidney, liver, spleen, lung, intestine, skin, and bone-muscle assembly are bottled and weighed separately for each animal.

Subsequent to the recording of the individual "wet weight", the tissues are dried in an oven to a constant weight. The dried tissues are placed in a de-humidified box containing "Drierite" dessicant. The box is maintained at 25 per cent relative humidity and at a temperature of 28°C . The tissues are each weighed in the box after a humidity equilibrium is reached.

The tissue wet weight and dry weight data are processed using a digital computer (IBM 650) and hand solutions are employed as a check. The computer program has been developed in order to handle the additional data from anticipated weight experiments.

In addition to the weight measurements on the groups of 5, total weight data are maintained on the remaining mice for both irradiated and control groups to observe weight trends in the population. As indicated previously, when total weight measurements are made on a day during which animals are to be sacrificed, the total weight is obtained prior to the removal of the animals.

2.3 Results - Experiment A

The mean weight for the total group or the "population" of irradiated animals may be compared with the mean weight of the control population in Figure 1. The mean weight of the irradiated population is generally higher than that of the control population throughout the 21-day period. The total number of mice in the control and irradiated cages decreases from the initial quantity of 101 mice, since 5 mice are removed from each cage on each of the days indicated (i.e., 0, 1, 2, 3, 7, 14, and 21 days), and there is normal attrition to be taken into account. Considering the decreasing population, a greater dispersion might be anticipated in the mean weight data from the latter part of the 21 day cycle. Furthermore, there is generally an increase in the range of body weights as mice mature. The curves shown in Figure 1 illustrate the growth trend for each population, neglecting the weight minimums occurring after

approximately two weeks which are attributed to disease.

On the various experimental days (i.e., 0, 1, 2, 3, 7, 14, and 21), groups of 5 mice are removed from each cage to be weighed and dissected. The mean body weights for these mice are given in Figure 2 for each of the days indicated. The mean body weight (21 grams) of the initial group of irradiated mice (at $t = 0$) is low in relation to the mean body weight (23.5 grams) of the total irradiated group. (See Figure 2). The mean body weight data from subsequent irradiated groups are more representative of the remaining irradiated animal population.

The mean body weight data from the control groups given in Figure 2 appear to have greater dispersion than the experimental group data, though the mean standard deviation is approximately ± 1.6 grams in both cases. Except for the body weight decrease in the controls during the second week shown in Figure 2, the data generally appears to deviate about rather than to follow the trends shown for the cage populations (Figure 1). This is an indication that groups larger than five mice or a greater degree of randomness in selection would be desirable.

The weight of the dried tissues in Figure 2 correlates with the points representing mean body weight after the third day of the irradiation period, indicating that the dispersion is not solely due to shifts in body fluids. Prior to the third day, the mean body weight appears to correlate with the mean weight of body water, also shown (calculated values) in Figure 2.

The normalized mean dry weight of the individual tissues indicate no differential growth trends for the various tissues are observed between control and irradiated animals. The trends in dry weight are not greatly different from the general weight trends shown in Figure 1.

In Figure 3, corresponding values for the relative quantity of water in the various tissues may be observed. In all cases, the trend is similar. The tissue water content of the initial groups of five is low. The groups which immediately follow have a relatively high tissue water content. Groups selected during the last two weeks of the experiment appear to have reached a consistent degree of hydration. Thus, the water content varies markedly during the first few days. Subsequently, no major changes in water content are seen, except possibly in the lung and the skin. The trend, then, is an initial period of fluctuation followed by stabilization at a relatively constant value.

NUMBERS OF MICE WEIGHED.

| TIME (DAYS) | 0 | 2 | 7 | 10 | 14 | 17 | 21 |
|---------------------|-----|----|----|----|----|----|----|
| NUMBER (IRRADIATED) | 101 | 92 | 82 | 71 | 66 | 61 | 61 |
| NUMBER (CONTROL) | 101 | 92 | 82 | 77 | 72 | 53 | 53 |

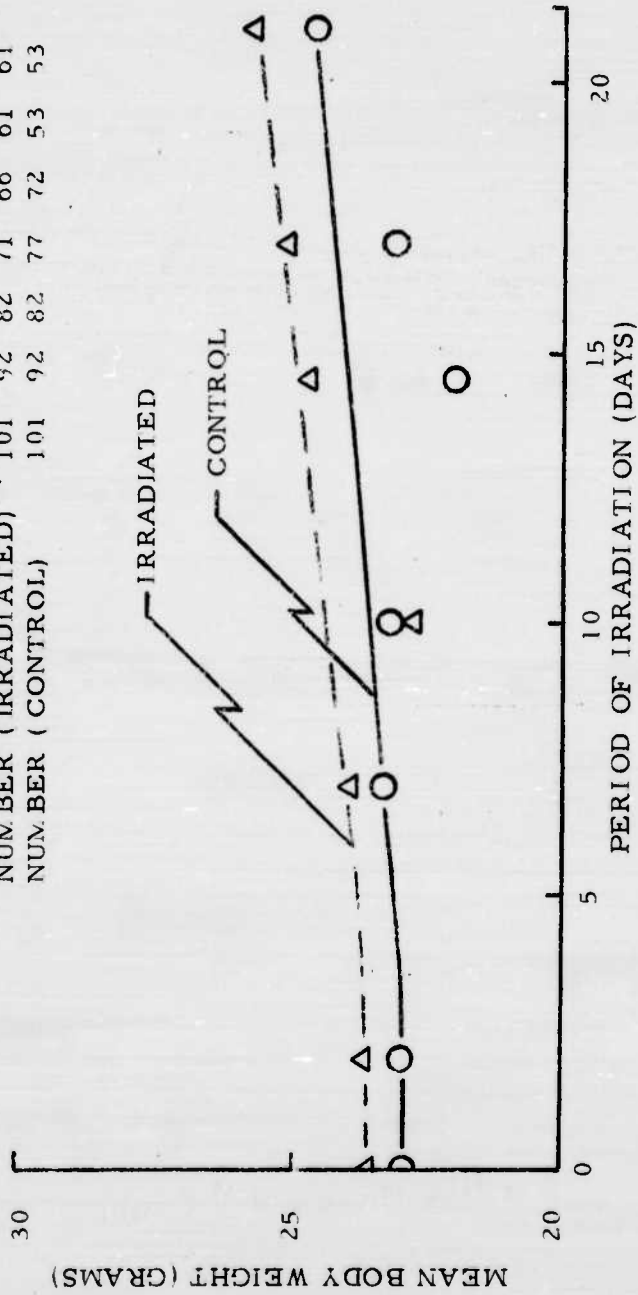


Figure 1 - Growth Trends in Mice

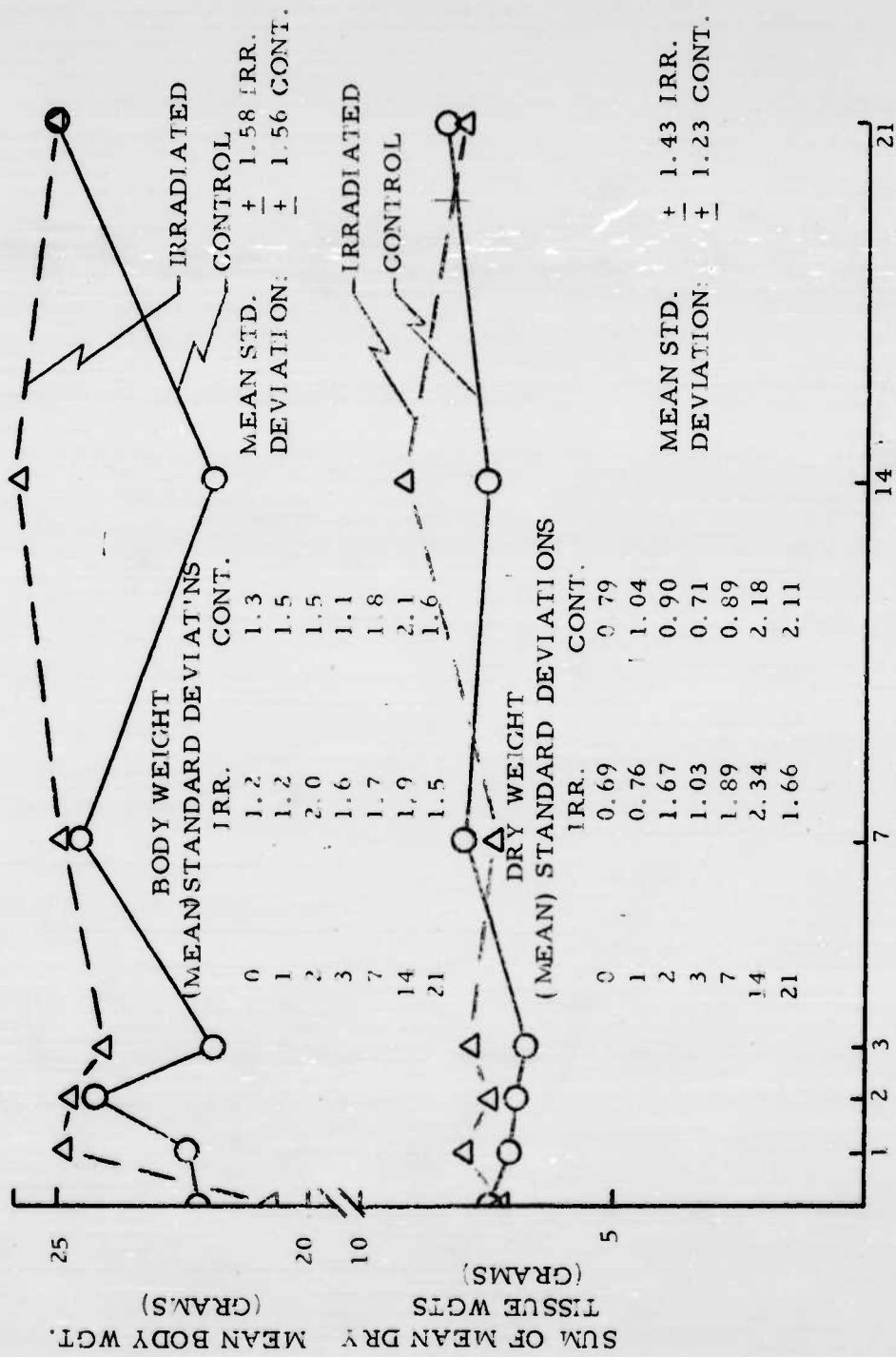


Figure 2 - Comparative Weights.

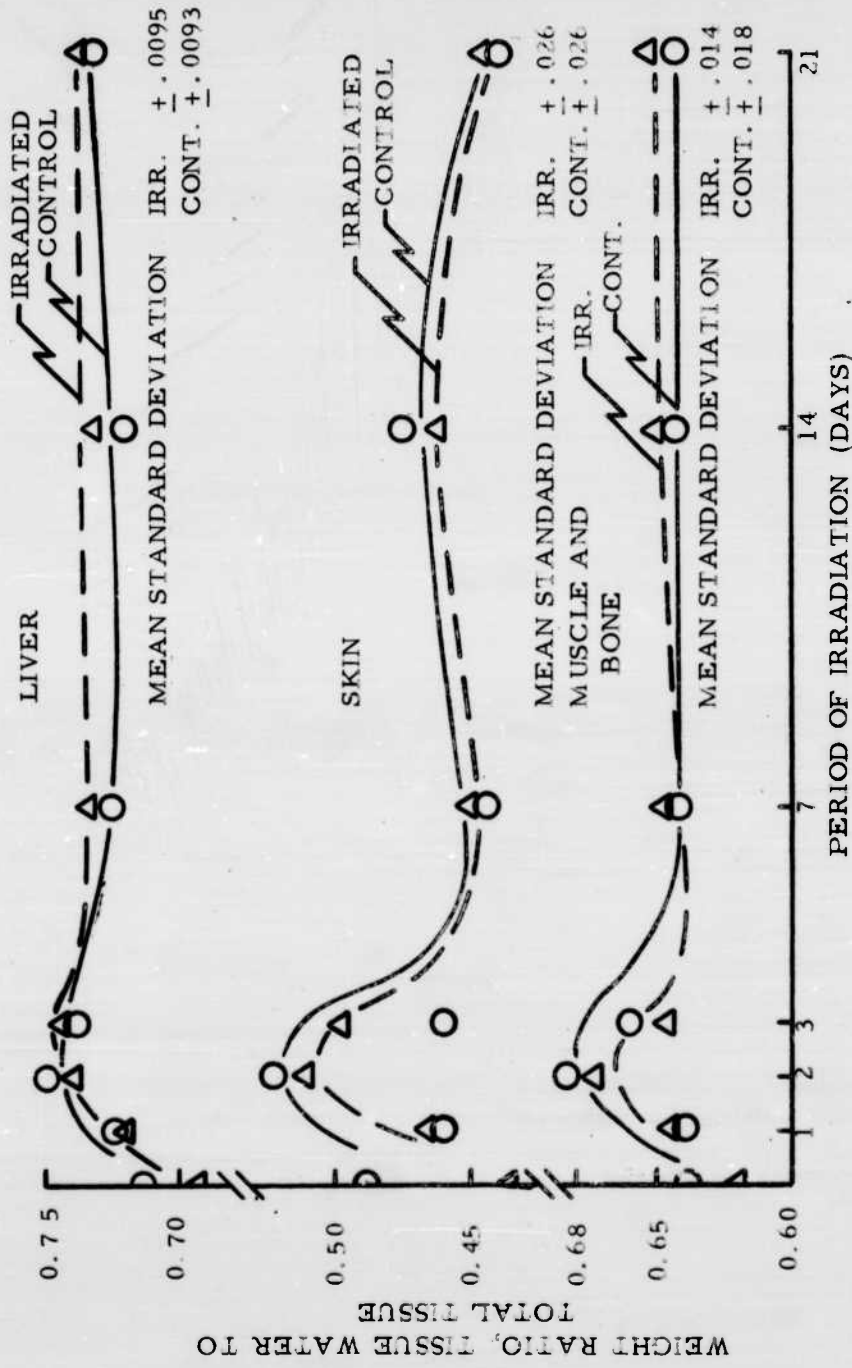


Figure 3 - Tissue Water Content.

2.4, Observations - Experiment A

Experimental groups of 5 mice selected at various intervals during the irradiation period do not adequately represent the mean body weight of the larger animal populations. A greater number of experimental samples is recommended for work of this type. Since the dispersion in weight increased with weight, smaller mice would be preferable.

The mean dry weights determined from such experimental groups do appear to represent long term growth trends in the larger populations, in spite of short term weight changes in such populations which are probably due to sickness.

Tissue water content fluctuated for a period greater than the five days allowed for acclimation, and a ten day period of acclimation following the receipt of a shipment of mice is suggested. The large fluctuations observed in the water content of skin (Figure 3) probably reflect the ease with which this hydrates and dehydrates. There is little difference between the water content of irradiated and control groups, emphasizing that weight differences observed between these groups represent differences in whole tissue mass of the mice selected, rather than systematic differences in tissue water.

2.5 Method - Experiment B

The results from the previous experimental study of the effect of 10 cm. microwaves on the growth rate of mice indicate a need for a greater number of experimental samples and the use of younger mice. These results show no systematic difference between irradiated and control animals in either dry weight or tissue water content. The present study is designed to determine the possible effect of low exposure rates upon growth rate.

Experiment B involves the following procedure. Weanling Swiss-Webster mice are obtained from Taconic Farms in New York. Following the arrival of the shipment, the mice are continued on their former diet of apples and potatoes with a new diet placed in the cage. The mice are gradually shifted to the new diet. This procedure is employed to decrease the possibility that weight changes due to diet might occur. The mice are allowed to acclimate to the cage environment for 7 days prior to irradiation, which is initiated on $t = 0$. The temperature and humidity are controlled during the experiment.

Irradiation is at a power density of 10 mw/cm^2 for a 2-minute period

each hour for 36 days using a 10 cm source. Weighings are made at $t = 0, 5, 8, 11, 15, 18, 22, 26, 29, 33,$ and 36 days after the initiation of the radiation sequence.

2.6 Results - Experiment B

The mean body weights for the various days are given in Figure 1 in section 2.0. The weight distribution for the control and experimental population on the initial day, the 18th day, and the 36th day are shown in Figures 4, 5, and 6. The curves shown are fitted in using as a basis the root mean square values computed from the original measured values which are plotted in the illustrations. The original value of each point is squared, and the squares of the two adjacent points are summed with the square of the chosen point and the square root of the mean is taken. The value obtained replaces the chosen point for purposes of curve plotting. The curves shown are fitted among the root mean square values to indicate general trends. The curves for the irradiated animals are relatively close to those for controls. Calculations indicate that no evident growth change can be attributed to irradiation using populations of the present size. The growth of the two groups may be observed in Figure 7.

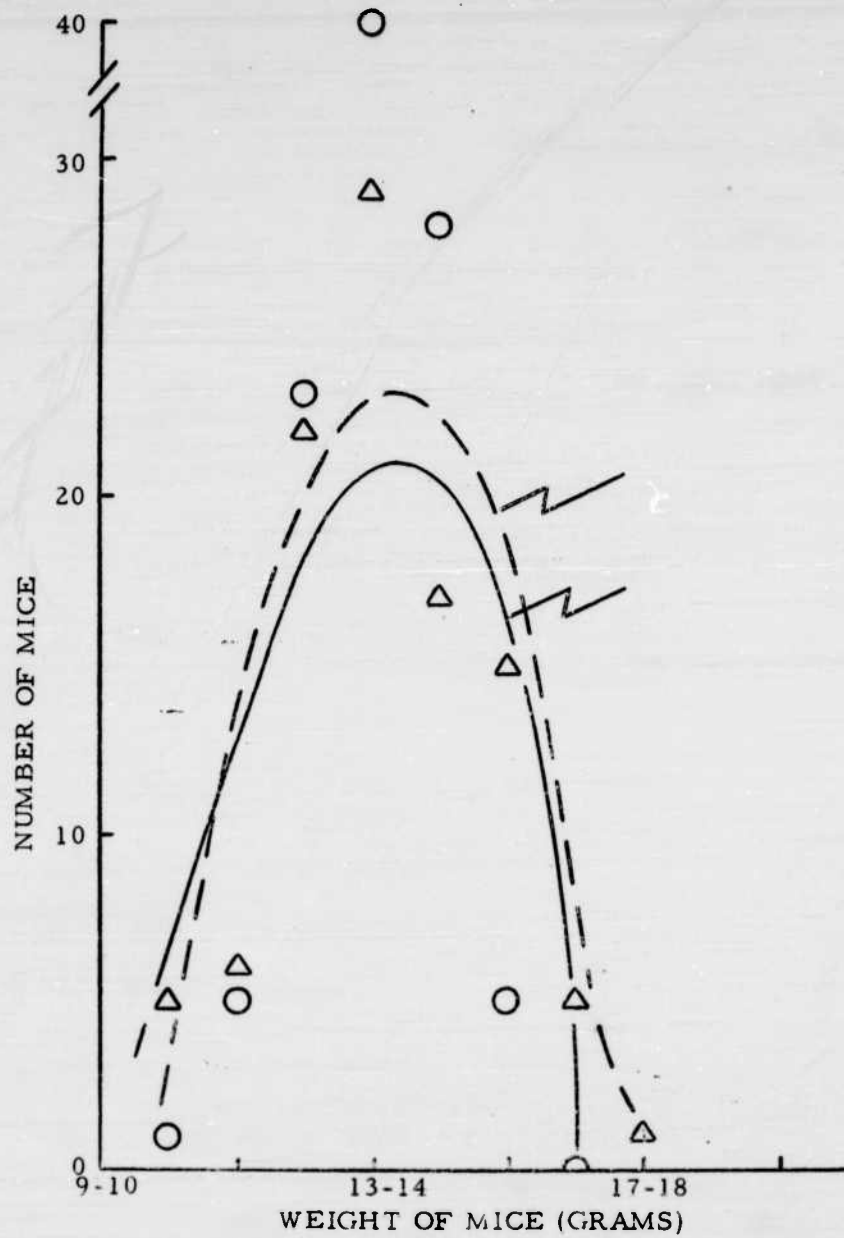


Figure 4 - Weight Distribution on Initial Day of Exposure.

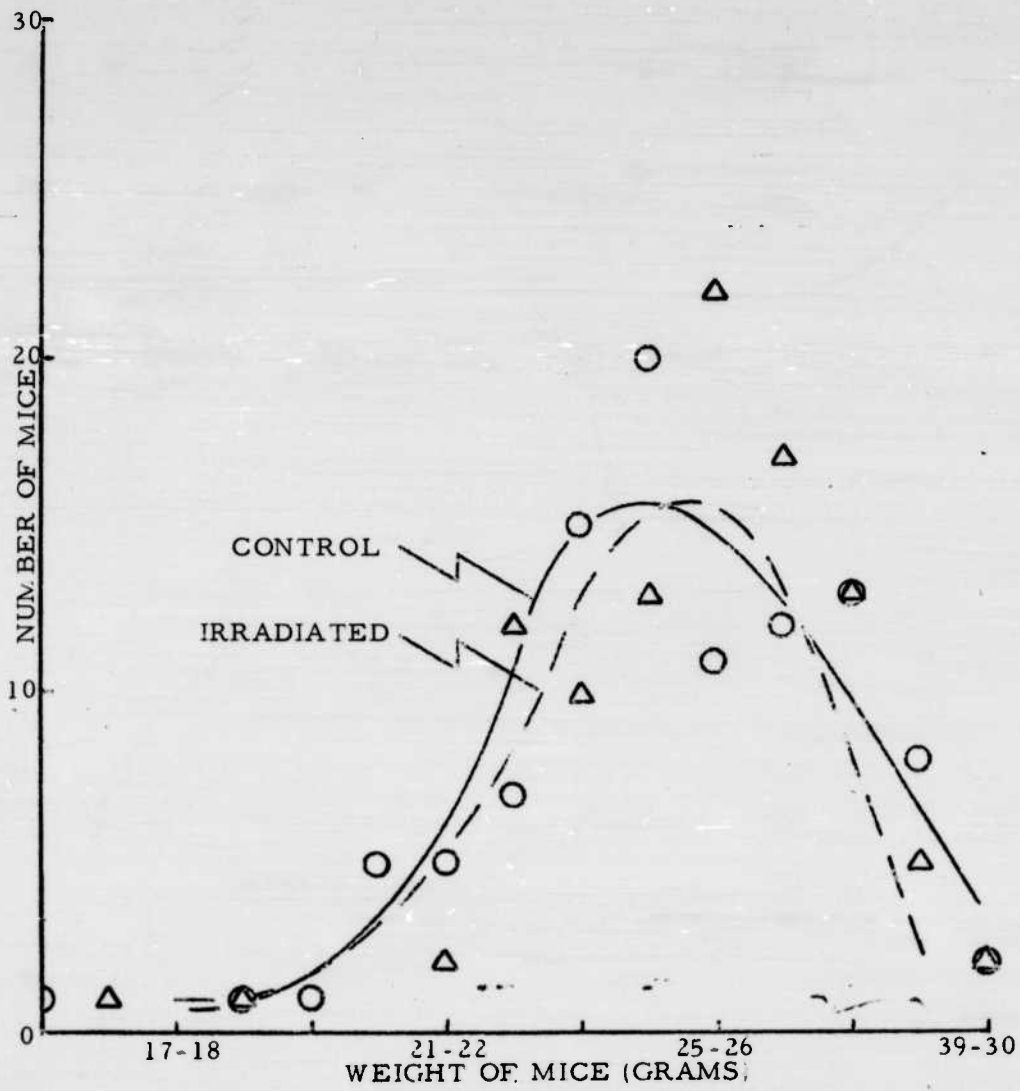


Figure 5 - Weight Distribution after 18 Days of Exposure

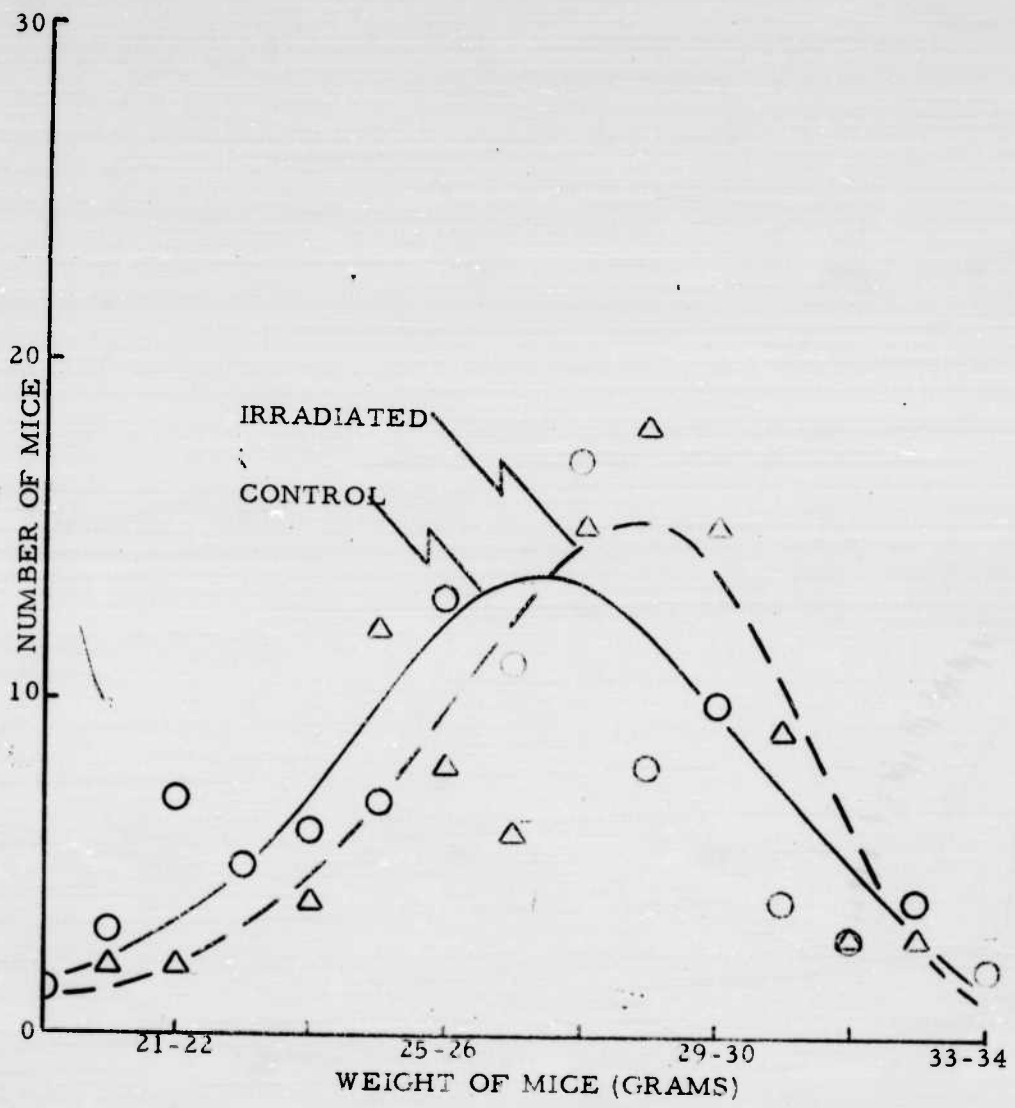


Figure 6 - Weight Distribution after 36 Days of Exposure.

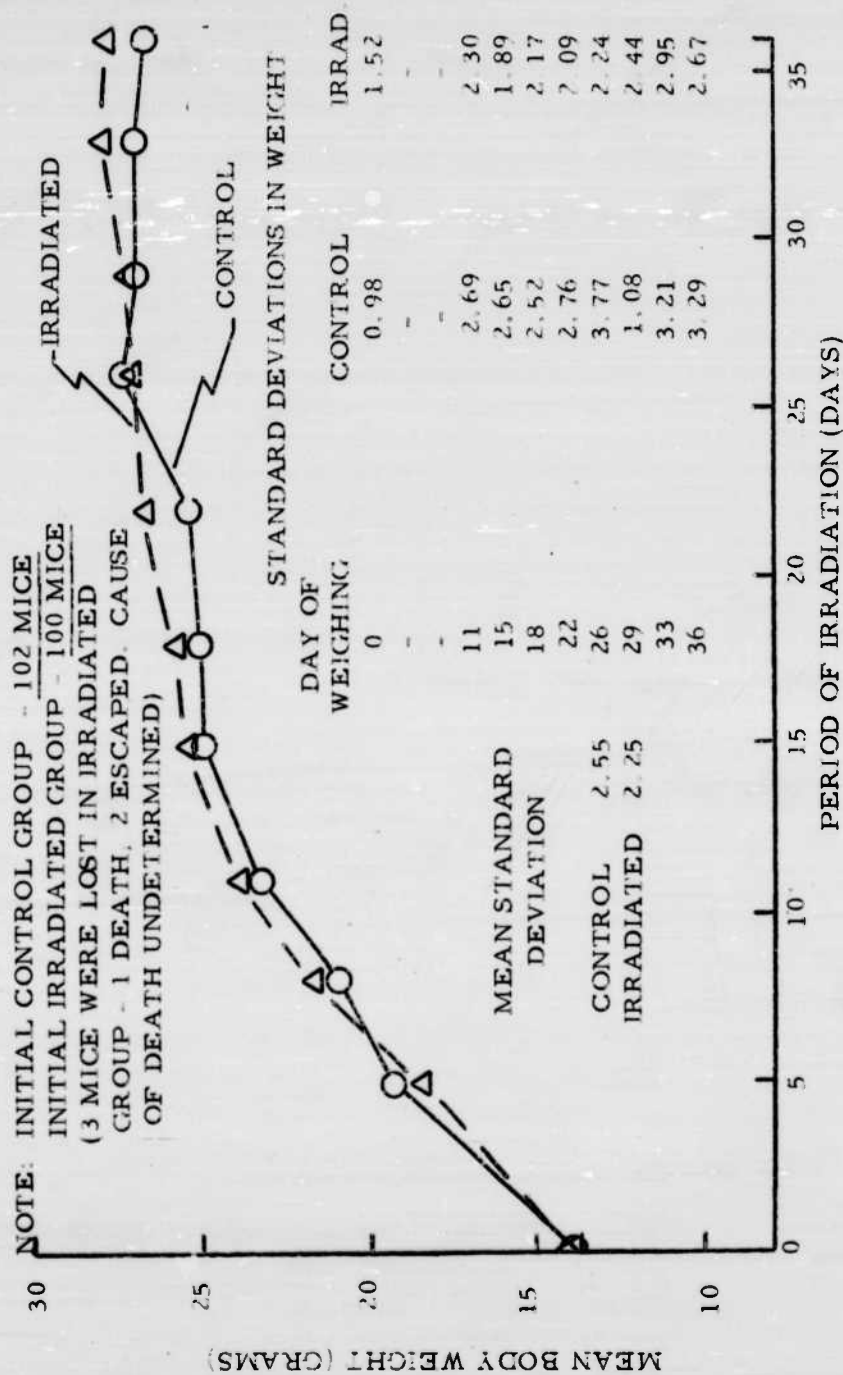


Figure 7 - Comparable Growth of Irradiated and Control Weaning Mice.

3.0 POWER DENSITY DISTRIBUTION IN TISSUES FOR 1.27 CM, 3 CM AND 10 CM MICROWAVES

In this section is listed data which can be used for the purpose of estimating the internal distribution of microwave power density in an animal subjected to a radiation field.

Tables I, II, and III give the plane wave reflection coefficients for various tissue interfaces and for wavelengths of 1.27, 3.0, and 10 centimeters. The table entry is the ratio of reflected to incident power density at the interface.

Tables IV, V, and VI give the values of penetration depth and wavelength in the tissue for various tissues and for incident free space and wavelengths of 1.27, 3, and 10 centimeters. The depth of penetration is defined as that depth at which the power density of the incident wave has been attenuated by a factor of $\frac{1}{e}$ (where $e = 2.718$). In other words, the depth of penetration is simply the reciprocal of twice the attenuation coefficient for a plane wave. Dielectric data used to calculate the values listed in tables I-VI have been obtained from Schwan,³ England⁴, and Baus⁵.

If standing waves are neglected, the tables may be used to compute the power density distribution as a function of the distance from a tissue interface. The formula to be used for this purpose is:

$$I_x = I_0 (1 - R_{1,2}) e^{-\frac{x}{D_2}}$$

- Where:
- I_x = the power density in the second medium at a distance x from the interface.
 - I_0 = the power density in the first medium incident on the interface of the two media.
 - $R_{1,2}$ = the reflection coefficient for the interface (See: Tables I, II, and III).
 - D_2 = the depth of penetration for the second medium (See: Tables IV, V, and VI).

If it is desired to calculate the power distribution within a multiple layer structure, it is simply necessary to apply the formula to each interface in turn, using the value I_x from the preceding calculations as the new I_0 .

| | WHOLE | | VITR | | | YELLOW | |
|----------------|-------|------|------|------|------|--------|--------|
| AIR | BLOOD | SKIN | FAT | HUM | LENS | BRAIN | MARROW |
| 0 | 0 53 | 0 47 | 0 10 | 0 64 | 0 54 | 0 54 | 0 32 |
| WHOLE BLOOD | 0 | 0 01 | 0 28 | 0 03 | 0 01 | 0 01 | 0 07 |
| SKIN | | 0 | 0 22 | 0 06 | 0 02 | 0 01 | 0 06 |
| FAT | | | 0 | 0 42 | 0 30 | 0 30 | 0 09 |
| VITREOUS HUMOR | | | | | 0 02 | 0 02 | 0 18 |
| LENS | | | | | 0 | 0 01 | 0 06 |
| BRAIN | | | | | | 0 | 0 08 |
| YELLOW MARROW | | | | | | | 0 |

Table 1 - 1 27 Centimeter Plane Wave Reflection Coefficients for Various Tissue Interfaces

(The Table entry is the ratio of reflected to incident power at the interface)

| | WHOLE | | | | | | | | | | YEL | MUS |
|----------------|-------|-------|-------|------|------|------|------|------|------|-------|------|-----|
| | AIR | WATER | BLOOD | SKIN | FAT | BONE | VITR | HUM | LENS | BRAIN | | |
| AIR | 0 | 0.62 | 0.57 | 0.53 | 0.13 | 0.22 | 0.60 | 0.49 | 0.48 | 0.21 | 0.53 | |
| WATER | 0 | 0 | 0.01 | 0.02 | 0.34 | 0.17 | 0.01 | 0.03 | 0.03 | 0.27 | 0.02 | |
| WHOLE BLOOD | 0 | 0 | 0 | 0.01 | 0.28 | 0.21 | 0.01 | 0.01 | 0.01 | 0.21 | 0.01 | |
| SKIN | 0 | 0 | 0 | 0 | 0.23 | 0.15 | 0.01 | 0.01 | 0.01 | 0.17 | 0.01 | |
| FAT | 0 | 0 | 0 | 0 | 0 | 0.01 | 0.33 | 0.20 | 0.15 | 0.01 | 0.24 | |
| BONE | 0 | 0 | 0 | 0 | 0 | 0 | 0.23 | 0.12 | 0.11 | 0.01 | 0.15 | |
| VITREOUS HUMOR | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.03 | 0.03 | 0.26 | 0.02 | |
| LENS | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.01 | 0.13 | 0.01 | |
| BRAIN | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.12 | 0.01 | |
| YELLOW MARROW | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.17 | |
| MUSCLE | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | |

Table II - 3 Centimeter Plane Wave Reflection Coefficients for Various Tissue Interfaces
(The Table entry is the ratio of reflected to incident power at the interface)

| | WHOLE | | | VITR | | | | |
|----------------|-------|-------|------|------|------|------|-------|------------|
| | AIR | BLOOD | SKIN | FAT | HUM | LENS | BRAIN | YEL. MAR'W |
| AIR | 0 | 0.57 | 0.55 | 0.11 | 0.63 | 0.52 | 0.52 | 0.22 |
| WHOLE BLOOD | 0 | 0 | 0.01 | 0.42 | 0.01 | 0.01 | 0.01 | 0.25 |
| SKIN | 0 | 0 | 0 | 0.19 | 0.01 | 0.01 | 0.01 | 0.37 |
| FAT | 0 | 0 | 0 | 0 | 0.28 | 0.15 | 0.12 | 0.01 |
| VITREOUS HUMOR | 0 | 0 | 0 | 0 | 0 | 0.04 | 0.05 | 0.27 |
| LENS | 0 | 0 | 0 | 0 | 0 | 0 | 0.01 | 0.14 |
| BRAIN | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0.12 |
| YELLOW MARROW | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

Table III - 10 Centimeter Plane Wave Reflection Coefficients for Various Tissue Interfaces.
 (The table entry is the ratio of reflected to incident power at the interface.)

| TISSUE | DEPTH OF PENETRATION CM | WAVELENGTH IN TISSUE CM |
|----------------|----------------------------|-------------------------------|
| WHOLE BLOOD | 0.0598 | 0.214 |
| SKIN | 0.0772 | 0.25 |
| FAT | 0.342 | 0.68 |
| VITREOUS HUMOR | 0.045 | 0.146 |
| LENS | 0.0706 | 0.200 |
| BRAIN | 0.075 | 0.200 |
| YELLOW MARROW | 0.145 | 0.368 |

Table IV - Depth of Penetration and Wavelength in Tissue for an Incident Wave Having a Free Space Wavelength of 1.27 cm. (The depth of penetration is defined as that depth at which the power of the incident wave has been attenuated to $\frac{1}{e} = .368$ of its value at the surface).

| BODY TISSUE | DEPTH OF PENETRATION CM | WAVELENGTH IN TISSUE CM |
|----------------|----------------------------|-------------------------------|
| WATER | 0.181 | 0.382 |
| WHOLE BLOOD | 0.148 | 0.449 |
| SKIN | 0.189 | 0.506 |
| FAT | 1.1 | 1.450 |
| BONE | 0.94 | 1.120 |
| VITREOUS HUMOR | 0.195 | 0.395 |
| LENS | 0.174 | 0.575 |
| BRAIN | 0.168 | 0.595 |
| YELLOW MARROW | 0.34 | 1.250 |
| MUSCLE | 0.134 | 0.616 |

Table V - Depth of Penetration and Wavelength in Tissue for an Incident Wave

Having a Free Space Wavelength of 3 Centimeters. (The depth of penetration is defined as that depth in the tissue at which the power of the incident wave has been attenuated to $\frac{1}{e} = .368$ of its value at the surface).

| TISSUE | DEPTH OF PENETRATION CM | WAVELENGTH IN TISSUE CM |
|----------------|----------------------------|-------------------------------|
| WHOLE BLOOD | 0.78 | 1.36 |
| SKIN | 0.646 | 1.49 |
| FAT | 2.45 | 3.79 |
| VITREOUS HUMOR | 0.535 | 1.18 |
| LENS | 0.500 | 1.75 |
| BRAIN | 0.476 | 1.74 |
| YELLOW MARROW | 9.924 | 3.97 |

Table VI - Depth of Penetration and Wavelength in Tissue for an Incident Wave Having a Free Space Wavelength of 10 cm. (The depth of penetration is defined as that depth at which the power of the incident wave has been attenuated to $\frac{1}{e} = .368$ of its value at the surface).

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NEUROPHYSIOLOGICAL EFFECTS OF MICROWAVE
IRRADIATION^N

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Abstract

Nerve fibers in peripheral nerves, such as the sciatic, or areas of skin rich in sensory fibers, such as the face of the cat, have been irradiated with 3 cm. microwave irradiation. A nociceptive response was obtained as the temperature of the irradiated part reached about 45° C. Since this response can be duplicated by heat from other sources and has been studied and described by C. Von Euler as a temperature effect on C and δ nerve fibers, it is concluded that the nociceptive response and its neural and hormonal components elicited by microwave irradiation are due to heating the nerve fibers to a critical temperature causing their stimulation.

^N This reasearch was supported by Rome Air Development Center, Air Research and Development Command, HDQS., Contract AF30(602)-1965 to the Biophysics Laboratory, Tulane University.

NEUROPHYSIOLOGICAL EFFECTS OF MICROWAVE IRRADIATION

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1.0 INTRODUCTION

1.1 Neural Effects of Microwave Irradiation Various reports indicate that neural effects may be one result of microwave irradiation. These effects are manifested in the behavior of the animal by such overt action as avoidance of the radar beam, confusion and staggering while within the beam, syncope, and, in some cases, convulsions^{1, 2}. Further symptoms which may be of neural origin include increased or decreased blood pressure and alteration of rate and depth of respiration^{3, 4}. These effects are often seen prior to an elevation of body temperature, and, in fact, some effects occur almost immediately on exposure to high intensity microwave irradiation². The relative immediacy of the response precludes an explanation based on hypothalamic temperature regulation mechanisms.

The reports on neural effects often indicate the possibility of direct central nervous system involvement^{1, 5, 6}. This hypothesis arises from those experiments which show marked nervous system involvement as a result of irradiating the head of an animal when contrasted with irradiation of some other region, such as the lumbar region. Apparently it is also of importance in producing the desired central nervous system effect if the animal's head is oriented in a favorable position with respect to the electromagnetic radiation⁵.

Most interesting are the reports that animals are aware of some type of painful stimulus when irradiated^{1, 2}. In fact, the stimulus may be sufficiently intense to arouse the subject from anesthesia.

Although a great many effects of microwave irradiation may be based on an overall rise in body temperature, and thus attributed to microwave heating, the neural effects, by reason of their rapid onset, seem not to fall under this general pattern. They may be classed by some along with other effects, perhaps, as non-thermal microwave effects. However, it is the purpose of this paper to demonstrate that the neural effects of microwave irradiation, particularly 3 cm. radar, may be attributed to the heat produced by microwave irradiation.

1.2 Nociceptive Reflex Pathways of the Peripheral Nervous System

and the Spinal Cord Grutzner and Heidenhain in 1878⁷ had observed during work on vasomotor reflexes that nociceptive reflexes⁴ with marked increases in blood pressure were elicited by heating a peripheral nerve.

C. Von Euler⁸ recognized their work and that of others, and utilized the phenomenon as an aid in determining the proportion and kind of fibers contained in a peripheral nerve. Prior work by Ranson⁹ showed that pain and temperature sensations elicited at temperatures over 40° C. are principally carried in the unmyelinated fibers making up the afferent group of a peripheral nerve. He showed that nociceptive reflexes were most easily elicited from skin areas which were richly supplied with unmyelinated fibers and that in nerve regeneration nociceptive sensation returns at the same time the unmyelinated fibers have grown out. It is generally accepted now that the unmyelinated fibers are specifically located at the point of entrance into the spinal cord where they pass to the tract of Lissauer and the substantia gelatinosa Rolandi. At the point of entrance into the spinal cord the unmyelinated fibers become differentiated from the myelinated fibers and lie in the lateral section of the root bundles, while the myelinated fibers enter in the medial section. Ranson and Billingsley¹⁰ severed the lateral section of the spinal root, thereby severing the unmyelinated fibers, preventing any nociceptive reflexes to strong Faradic stimulation of the distal end of this root. These nociceptive reflexes were, however, still present if only the myelinated fibers were severed by a medial incision in the root and the distal end stimulated.

Ranson¹¹ found that the unmyelinated fibers ascend ventrolaterally to the tract of Lissauer and the substantia gelatinosa Rolandi. Ranson and Hess¹² found that the tract of Lissauer and the substantia gelatinosa Rolandi form an organ for the reception and intersegmental conduction of afferent pain impulses. Some impulses from this region, passing over the spino-thalamic tract, would reach the cortex and find expression as conscious pain, while other impulses ascending and descending within it, would produce pain reflexes, that is, nociceptive reflexes. In their studies on the vasomotor reflex arcs, Ranson and Billingsley¹⁰ also showed that both pressor and depressor reflexes can be transmitted by the unmyelinated fibers but that their intraspinal tracts are differentiated.

⁴ A nociceptive reflex may be defined as a reflex which comprises movements for protection or defense, or for the withdrawal of the part from the noxious agent. They are prepotent, other less urgent reflexes being for the time inhibited, and they are imperative. A nociceptive reflex may be accompanied by alterations in the action of the autonomic nervous system. Perhaps the autonomic response may be included with the nociceptive reflex, inasmuch as the fright or flight responses involve involuntary activation of visceral musculature, glandular substance, vascular tissue, and respiratory action which are related to protection and defense.

The nociceptive impulses are conducted by unmyelinated C fibers¹³, and by the fibers of the O group¹⁴, and, in addition, by the thinly myelinated nerve fibers^{15, 16, 17, 18}.

Finally, according to Sjoquist¹⁹, pain impulses in the head region are probably conducted by the thin myelinated fibers in the trigeminal nerve of man.

1.3 The Response to Thermal Stimulation of Mammalian Nerves

Von Euler's experiments⁸ on thermal stimulation of mammalian nerves were undertaken in order to develop a means of determining the ratio and amounts of myelinated and unmyelinated nerve fibers making up a peripheral nerve fiber. He was able to correlate the nociceptive response obtained with the kind of nerve studied and with the actual anatomical count of the number and ratio of the different types of fibers which make up the nerve.

In his experiments, Von Euler used decerebrated cats in order to study the spinal reflexes which are involved in the nociceptive response. The decerebrated animal is ideal in this respect for two reasons:

1. The animal may be anesthetized with ether, decerebrated, and allowed to come out of anesthesia. Decerebration prevents the need for a central anesthesia and allows spinal and medullary reflexes to operate unexpressed.

2. The decerebrated animal exhibits strong extensor tonus which causes the animal to assume a stiff, limbs outstretched, position. The back may be arched dorsally. This tonus can be overcome, however, by a strong stimulus which in the conscious animal would appear to cause behavior indicative of a painful stimulus. This stimulus in a decerebrate cat is prepotent over extensor tone and causes marked flexion in the ipsilateral limb and hyperextension of the contralateral limb.

Von Euler exposed but did not cut the nerve under investigation and placed over a short section of the exposed nerve a thermode which consisted of a "small lacquered metal container with a cross section like a shallow U forming a deep narrow groove for the nerve." A thermode 1 cm. long was preferred. The temperature of the thermode was regulated and either increased or decreased by means of circulating water, the temperature of which was measured by means of a thermocouple.

The rate and depth of respiration, blood pressure, and pulse pressure were measured and recorded simultaneously with the temperature of the thermode. As the thermode was warmed up to between 42 and 45° C.

a strong nociceptive reflex was produced. Note that the temperature is about 110° to 113° F., and local application of heat at this temperature would not in general be thought critical to any physiological function. In particular, this local warmth would hardly be expected to produce the following nociceptive response which is described by Von Euler⁸.

"The animal responds as to severe pain. Flexor contraction occurs in the ipsilateral leg and extensor contraction in the opposite one. The body bends ventrally, and the head is pulled back towards the trunk. The breathing is greatly affected, but varies from animal to animal. As a rule, the amplitude is decreased even down to apnea; the frequency either increases or slows down. The blood pressure measured in the carotid artery rises steeply. In most cases, the increase is between 60 and 70 mm Hg..."

2.0 EXPERIMENTAL

It seems possible that there may occasionally result during microwave irradiation of man and experimental animals situations in which sensory nerves lying close to the skin may rapidly heat as a result of irradiation. The local heat may be retained locally if the nerve is embedded in and overlain by relatively microwave-transparent fat which would act as a thermal insulator. The relative avascularity of neural and fatty tissue would inhibit local cooling by the blood. It is possible that a local temperature of 112° F. may be reached at and within the nerve while the surface of the skin overlaying the nerve may be heated to a lesser degree. In addition, the mean temperature of the systemic blood may not have time to reach the level necessary to trigger the hypothalamic temperature compensating response. If such a situation should exist the subject may experience a nociceptive reflex, including an elevation of blood pressure and perhaps a sensation of pain.

In order to illustrate the possibility of such a nociceptive reaction occurring with microwave irradiation, and to explore the possibility of side effects resulting from microwave-heat induced nociceptive reflexes, the experiments reported in this section were performed.

2.1 In Situ Irradiation of Exposed Nerve Trunks. Since the 3 cm. microwave source available to us at this laboratory is not very powerful, the nerves to be irradiated and studied were exposed by reflecting overlaying skin while leaving the nerve sheath intact. In addition, exposure of the nerve allowed thermistor estimation of its temperature to be made. The thermistor probe was placed under the nerve, proper shielding of all areas except the exposed nerve and area immediately adjacent assured that the microwave irradiation was absorbed locally. The temperature of the region

surrounding the nerve under study and the body temperature were not elevated. The 3 cm. microwave radiation used in these experiments was pulsed at 1000 pps. The object to be irradiated was placed about 4 cm from the horn in the near field and received a power density of about 0.2 watts/cm².

2.2 The Effect of 3 cm. Microwave Irradiation on the Exposed Sciatic and Radial Nerve of the Decerebrate Cat

2.2.1 Preparation of the Animal. The animals were anesthetized with ether and decerebrated, the brain being removed as far back as the tentorium. The sciatic nerve was located and exposed but kept moist with normal saline. The carotid artery was cannulated for blood pressure measurement. The cats were allowed to recover from ether anesthesia, but as a result of the decerebration remained unconscious.

The animals generally developed good decerebrate rigidity with strong extensor tone and stiff limb extension. It was necessary to apply considerable force against the feet of the cats while supporting the shoulder or back in order to flex the limbs.

Measurements of respiration, pulse rate, and blood pressure were obtained, before, during, and after irradiation.

2.2.2 Microwave Irradiation of the Exposed Sciatic and Radial Nerve of the Decerebrate Cat. Irradiation of the exposed sciatic or radial nerve of the decerebrate cat caused the temperature of the irradiated nerve to increase gradually above body temperature. The animal remained quiet as the temperature increased to about 45° C., when there generally occurred a sudden, marked, nociceptive reflex from the previously quiet animal. Generally, the animal flexed the ipsilateral limb and further extended the contralateral limb. Movements of the forelimbs, head, and back often occurred. Immediately following the first sign of movement, there occurred a steep rise in blood pressure 40 to 50 mm/Hg above base line. The maximum value would be reached in about 5 seconds. Even in cases where no movement of the limbs occurred, the blood pressure rose steeply. The respiratory effects generally followed a pattern of apnea followed by increased rate and depth of respiration. In some cases, however, alteration of respiration did not occur.

If the radar was turned off and the temperature allowed to drop, the animal re-extended his leg and the blood pressure dropped rapidly. If the radar was kept on, but the temperature maintained by pulsing the power, the blood pressure dropped slowly. If the temperature was gradually increased, the blood pressure could be maintained at an elevated

level (Table I and Figure 1). The same response could be obtained from the radial nerve (Table I).

After perhaps ten repetitions of this treatment, some animals were slower in returning to a normal blood pressure, pulse rate, and respiration pattern. The blood pressure became depressed, and the pulse rate quickened and weakened. The respiration became periodic. Generally, further irradiation to the point of elicitation of the nociceptive response often resulted in the death of the animal.

2.2.3 Movie of the Nociceptive Response of a Decerebrated Cat.

2.2.4 The Effect of Infrared Heating on the Exposed Sciatic Nerve of the Decerebrated Cat. Von Euler clearly demonstrated the effect of warming exposed sciatic nerves (and other sensory nerves) on numerous cats and on rabbits, and his results appear identical with ours. Therefore, it was considered unnecessary to run a series of experiments in which the nerves were heated by a means other than microwave heating; however, infrared was tried in a few cases (Figure 2), and a nociceptive response identical to that induced by radar was obtained at about 40° C. The lower temperature reading may be accounted for by the location of the thermistor under the nerve. The probe did not indicate, therefore, the temperature at the upper surface of the infrared irradiated nerve which surface would be at a higher temperature since absorption of infrared is more superficial than the absorption of 3 cm. microwave irradiation. Mechanical stimulation of the nerve by crushing it after infrared irradiation showed the nerve to be still responsive. Indeed the nociceptive effect obtained from crushing the nerve with a hemostat is no more intense than the effect obtained by thermo-stimulation.

2.2.5 The Effect of Cooling the Exposed Sciatic Nerve of the Decerebrated Cat, during Microwave Irradiation. As a control (Figure 1) the nerve was irradiated, and a nociceptive response was obtained at about 45° C. After the animal recovered, the nerve was again irradiated at the same power density but prevented from heating above 40° C. by blowing cool air over it. The irradiation was continued for longer periods than those previously required to elicit the nociceptive response. No nociceptive response was obtained from the irradiated cooled sciatic nerve while it was being cooled. When cooling was stopped, however, a nociceptive response occurred.

2.3 Microwave Irradiation of the Zygomatic Branch of the Facial Nerve and the Trigeminal Nerve in the Decerebrate Cat

2.3.1 Theory. Nerves rich in sensory fibers of the C and S

TABLE I

| Preparation | Irradiated Site | °C At Which Response Occurred | Movement: Crossed Ext Ipsi Flex., Other | Blood Pressure mm/Hg | | Apnea | Resp Rate | Remarks |
|-----------------|-----------------|-------------------------------|---|----------------------|--------------------|-------|-----------|--|
| | | | | Initial | Max. (5-10 sec) | | | |
| Decerebrate cat | Sciatic Nerve | 43 | Yes | 100 | 150 | | | Resp de-creased after irradiation |
| " | " | 40-48 | Yes | 75 | 100 | In- | In- | B P maintained about 100 mm/Hg by continuous irradiation |
| " | " | 43 | Yes | 80 | 68 | In- | In- | Drop in B P, crease defecation, micturition, vomiting |
| " | " | 45-48 | Yes | 70 | 100-110 | De- | De- | B P va-crease ried but maintained by gradually increasing temperature |
| " | " | 45-48 | | 90 | 150 on In- 110 off | In- | In- | 5 min run. Radar on and off Temp. drops below 45° on off. B P. rose and fell with each temp. rise above 45°-5 times. |

| Decerebrate cat | Radial Nerve | 45 | Yes | Face movement | 92 | 130 | Inc rate, depth | Temp. reading may be in error |
|-----------------|--------------|-----------------|----------|---------------|-----------|-----|-----------------|--|
| " | " | 35 | | | 80 | 145 | | |
| " | " | 55 | | | No change | No | | |
| " | " | 43 | Withdraw | | 115 | 140 | change | B P dropped to 95 (Comparison of abdomen with fore paw) |
| " | " | 43 | | Movement | 90 | 135 | | |
| " | " | 55 ⁺ | | | 100 | 95 | | No change (Comparison of shielded skin flaps with forehead skin) |
| " | " | 45 | | Head movement | 100 | 130 | | |
| " | " | 40 | | | 110 | 110 | | No effect while cooling nerve when irradiated. |
| " | " | 43 | Yes | | 110 | 150 | Apnea | |
| " | " | 47 | | | 110 | 130 | | |

| | | | | | | | |
|------------------|---------------|-------|--------------|----|-----|-----------|--|
| Chloralose | Sciatic Nerve | 45 | - | 60 | 118 | De-crease | No XeX movement but cats fail to show XeX occasionally. |
| | " " | 47 | | 60 | 145 | | |
| Nembutal | Rear paw | 43 | Withdrew paw | 90 | 143 | De-crease | Cat animated, began trembling, partly awakened |
| Decerebrate cat | Sciatic Nerve | 43 | No | 70 | 110 | Incr | Dibenamine |
| Injected 2 cc | Dibenamine | 49+ | No | 73 | 73 | | appears to have blocked B. P. rise. XeX not present in control. |
| Decerebrate cat | Sciatic Nerve | 40-42 | Yes | 70 | 110 | | The effect of infrared (10.4) on blood pressure and reflexes is equal to that elicited by microwave irradiation or crushing the nerve with a hemostat. |
| Plus Infrared 10 | " " | - | Yes | 70 | 110 | | |
| Crush nerve | " " | - | | | | | |

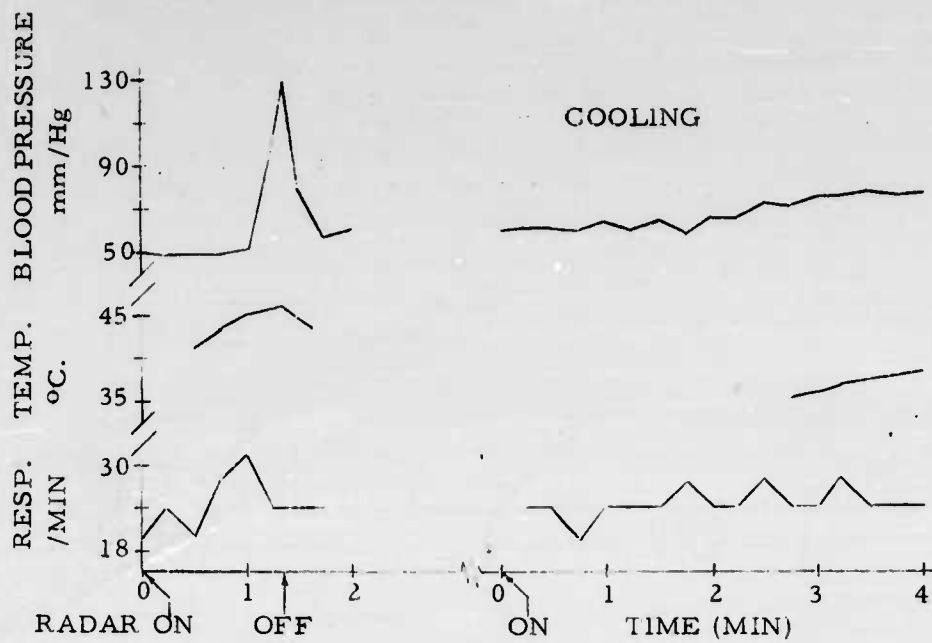


Figure 1 - Decerebrate Cat, Sciatic.

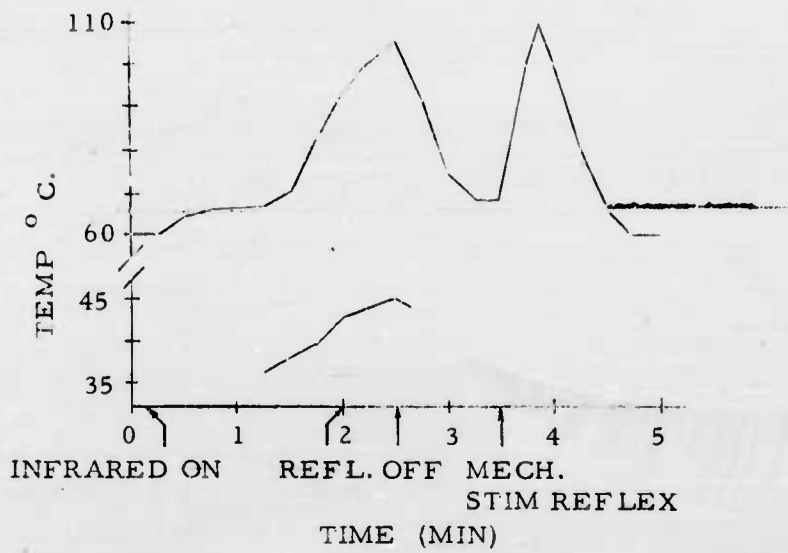


Figure 2 - Decerebrate Cat, Sciatic.

class were shown by Von Euler to be highly responsive to thermostimulation. Among the sensory nerves which are rich in sensory fibers are the cranial nerves, particularly the trigeminal and the facial. Although the trigeminal nerve emerges from the side of the pons and the facial nerve from the lower border of the pons, these nerves belong to the peripheral nervous system rather than the central nervous system. The peripheral branches of these nerves leave the skull through foramina and pass along the bony structures of the head just below the skin. These nerves are generally protected by insulating tissue where they lie most nearly exposed to the elements. Nerves in such exposed positions may become easy targets for microwave heating, especially if the head is oriented to allow the nerve to receive maximum microwave energy.

2.3.2 Results. In order to test this assumption, the zygomatic branch of the facial nerve of a decerebrated cat was exposed and irradiated with 3 cm microwave irradiation. As the temperature of the nerve reached about 45° C there was a weak nociceptive response; there was no movement, and the blood pressure rose moderately. The branches of the trigeminal nerve were exposed, and at about 45° C there was a strong nociceptive response, head movements were observed, and the blood pressure rose sharply.

2.4 Microwave Irradiation of Skin Areas Richly Supplied with Sensory Fibers Contrasted with Skin Areas Poorly Supplied with Sensory Fibers.

2.4.1 Theory The nerve trunks, sciatic, radial, trigeminal, facial, which have been investigated are bundles composed of individual nerve fibers which are distributed and dispersed peripherally under the surface of the skin at various depths and within the subcutaneous tissue. Microwave irradiation of an area of skin richly supplied with these fibers would perhaps penetrate readily to a depth within the skin where the nerve fibers lie. Thus, thermal stimulation of large numbers of sensory axons distributed within an irradiated area should be as effective in eliciting the nociceptive response as direct irradiation of a nerve trunk composed of bundles of these fibers.

2.4.2 Irradiation of Feet and Forelegs of the Decerebrated Cat - Sensitive Region. The feet and the leg region just above the feet of a cat are areas richly supplied with sensory nerve fibers. A small area about 1/2 inch square was irradiated at 45° C as indicated by a thermistor placed under the skin. A strong nociceptive reflex occurred, and the decerebrate animal withdrew his foot. The effect could be repeated with both forelimb and hind limb (Table I).

A triangular flap of skin including the subcutaneous fatty tissue was dissected from underlying tissue but not detached from the foreleg (or hind leg) skin at the proximal base of the triangle. The exposed limb muscle and tissue were wrapped in gauze and moistened with normal saline. Aluminum foil was wrapped around the limb at this site and covered with moistened gauze. The thermistor probe was placed on the gauze, and the flap of skin placed over the gauze and probe. Except for this skin flap, the rest of the leg and the animal's body were shielded from microwave irradiation. The radar was turned on, and at about 43° C. a sharp rise in blood pressure occurred (Table I).

2.4.3 Irradiation of the Ventral Surface of the Decerebrated Cat - A Region Poorly Supplied with Pain Fibers. A thermistor was placed under the abdominal skin of a decerebrated cat, and an area about 1 inch square was irradiated. No change in blood pressure or respiration rate, nor any movement was observed. The temperature was allowed to reach 55° C., when a slight response occurred. This response was then compared with irradiation of forepaw skin, and the blood pressure was seen to rise as the subcutaneous temperature reached about 40° C. (Table I).

A flap of ventral abdominal skin 1 in. square was dissected free from underlying tissue except for one side (near the midline) and prepared in the manner described for the leg skin flap (2.4.2). The radar was turned on, and the temperature rise was allowed to continue until the skin had a cooked appearance (+60° C.). No change whatsoever occurred in blood pressure or respiration, nor was there any sign of movement (Table I). As an intermediate region, the skin above the ribs of a decerebrate cat was irradiated. Not until the temperature rise exceeded 45° C. did a significant blood pressure rise occur. The pressor reaction resulting from stimulation of this region was less than that obtained from the extremities.

2.4.4 Microwave Irradiation of the Cranial Skin of the Decerebrated Cat. A 1/2 inch square section of skin over the frontal region of the skull of a decerebrated cat was exposed to 3 gm. microwave irradiation; as the thermistor under the skin indicated 43° C., a sudden, strong, nociceptive reflex with head movements and rise in blood pressure was produced (Table I). As a comparison, the skin over the back of the cat, which is a region poor in sensory fibers, was irradiated without effect.

2.5 The Effect of a Sympatholytic Drug - Dibenzamine. That the nociceptive response produced by microwave irradiation involves the sympathetic nervous system seems probable, and in order to test this possibility a sympatholytic drug was used in order to block the response of the sympathetic nervous system to the nociceptive stimulus.

An exposed sciatic nerve of a cat anesthetized with nembutal was exposed to 3 cm. microwave irradiation, and an increase in blood pressure elicited. Two ml. dibenamine was then infused into the femoral vein and 20 minutes allowed for the dose to take effect. No blood pressure changes of significance were observed on subsequent irradiation (Table I).

2.6. Local 3 cm. Microwave Radiation of Anesthetized Cats. Von Euler in his work on selective responses to thermal stimulation of mammalian nerves⁹ indicates that the blood pressure response (pressor) remain in cats which have been anesthetized with chloralose. It was desired herefore to investigate the effect of this anesthetic on an intact cat.

The sciatic nerve in a cat anesthetized with chloralose was exposed and irradiated with 3 cm. microwaves. At about 43° C. a sharp increase in blood pressure was observed (Table I).

The right rear paw of a cat anesthetized with nembutal was irradiated. A strong nociceptive response occurred at about 42° C. with micturation, alteration in respiration, and subsequent trembling (Table I).

3.0 DISCUSSION

The experiments reported herein show that local heating by microwaves of sensory nerve trunks elicits a nociceptive response identical to that reported by Von Euler for thermode temperature stimulation. In our experiments we have heated the sciatic or radial nerve by microwave irradiation, and we have compared our results with the temperature response reported by Von Euler. The average temperature at which Von Euler obtained his response, about 45° C., corresponds with the average temperature at which we obtained responses. In our experience, as in Von Euler's, there was some variation in the temperature at which the response occurred. He found that the response occurred at a definite temperature within a range between 42° and 47° C. In addition to biological variation inherent above, some error on our account may have occurred because we placed the hypodermic thermistor under the nerve, and, therefore, did not register its internal temperature. Often the movement of the cat, as a part of its nociceptive reflex, would displace the thermistor and the subsequent temperature readings would be lost.

For reasons stated in the introduction of this paper, the nociceptive response of sensory nerve trunks to heat is not a response of the heat receptors of the skin. Indeed, there are no heat receptors in the nerve trunk at the local site of irradiation. Therefore, we conclude that it is probably the same unmyelinated and thin myelinated fibers which were

shown to be reactive to heat by Von Euler, that are causing the nociceptive response to microwave radiation induced heat reported herein. Whether the myelinated fibers can be stimulated by radar induced heat in any way awaits further study.

Sensory nerve fibers which lie rather superficially in certain regions may perhaps be easily heated by microwave irradiation. Fatty tissue in which these nerve may be embedded would prevent loss of the heat generated at the nerve because of its thermal insulating properties and its poor vascularization. These nerves may therefore respond to local microwave heating by a rise in neural temperature to the critical level necessary to produce the nociceptive response while nearby skin and muscle tissue remains adequately cooled. The head and feet of an animal, and the head and hands of man, are possible regions where local neural microwave heating may occur. This would be particularly true if the subject were oriented with respect to the radar beam so as to allow sensory nerve trunks to absorb a maximum of microwave radiation.

A reasonable extension of the response to microwave heating by large sensory nerve bundles mentioned above suggested that areas of skin richly supplied by sensory fibers of the C and δ type which make up the sensory nerve bundle would produce a nociceptive response when irradiated with 3 cm microwaves. The microwave heating may reach a sufficient depth within the skin (dependent on the radar frequency) to penetrate to the tela subcutanea where the cutaneous nerve fibers branch out under the dermis prior to sending perpendicular branches through the dermis and to the epidermis. In contrast with microwaves, infrared radiation is primarily absorbed at the surface of the skin and produces its maximum heating at a depth which corresponds to the level of the heat sensitive thermal receptors. As a result of infrared heating, vascular changes in the skin take place which protect the deeper lying structures from infrared damage and prevent the neural plexus from reaching critical temperatures. Surface evaporation and cold environments aid in protecting against infrared radiation. Microwave heating is not a usual environmental challenge as is infrared heating, and the evolution of organisms has not provided an adequate warning and protecting mechanism for local heating of a penetrating nature.

4.0 SUMMARY

A nociceptive reflex can be elicited from a decerebrated or anesthetized cat by microwave irradiation. The reflex includes a sharp rise in blood pressure and pulse rate, changes in the rate and depth of respiration, and movements of the limbs and body which suggest an attempt to withdraw from the stimulus. These responses occur when an exposed

sciatic, radial, facial, or trigeminal nerve reaches a temperature between 42° and 47° C. as indicated by a thermistor probe. The nociceptive response and the temperature at which it is elicited is identical with the response from a jacketed and water heated nerve trunk as described by Von Euler⁸.

The response does not occur if the nerve is cooled while being irradiated, and it can be elicited by infrared heating. In addition, the nociceptive response may be obtained by irradiating with microwaves skin rich in sensory nerve endings but not from skin poor in sensory fibers.

The neural effects of 3 cm. microwave irradiation may be attributed to temperature stimulation of thinly myelinated and unmyelinated sensory nerve fibers of the C and A types, either gathered in nerve bundles or distributed beneath areas of skin richly supplied with sensory nerves of these kinds.

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Acknowledgement:

The interest, help and guidance of Drs. L.M.N. Bach and Herbert B. Kelly, Jr., of the Department of Physiology of the Tulane University School of Medicine, and the technical assistance of Mrs. Archie Berger, Messrs. Norman D. Stockwell, Robert Jahncke, and James R. Larose are greatly appreciated.

Comments on Papers Delivered at Tri-Service Conference on
Biological Effects of Microwave Radiation

by

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1) A Preliminary Investigation of the Application of Magnetic Resonance Absorption Spectroscopy to the Study of the Effects of Microwaves on Biological Materials.

Drs. Pish, Storey, Truby and Rollwitz have demonstrated that magnetic resonance absorption spectroscopy is potentially a very powerful analytic tool for diagnosis; that changes in living tissue manifest themselves as changes in the fine structure of the absorption spectrum. Unfortunately the spectra associated with specific changes are not predictable. Even careful analysis of single compounds for their spectra would not permit the prediction of the spectrum of this compound in the presence of others. As a result, for living tissue, the complexity of the composition requires the tabulation of the spectra associated with each basic condition of the tissue before the change in the spectrum can be used for diagnosis. The fine structures in the spectra are modified by the interactions between ions and the existence of non-linear mechanisms in the nuclear magnetic resonance. The measurement procedure is critical if reproducible results are to be achieved. The results are affected by the uniformity of the magnetic field over the sample as well as by the constancy of the amplitude and frequency of the applied radio frequency signal.

2) Techniques for Relative Absorption Cross Section

Drs. Schwan and Salati have presented a very ingenious technique for obtaining an insight into the mechanism of absorption of microwave energy by living tissue. The technique they have investigated can provide a basis for analysis of the more complex configuration of the human head, but is not to be taken as an equivalent electromagnetic model. The thermal distribution associated with their liquid filled sphere will provide a qualitative evaluation but not an absolute quantitative representation. The ability to vary the dielectric and resistive characteristics of the liquid will permit the simulation of individual constituents of the human head and provide a basis for obtaining the expected limits of absorption coefficient. This would provide a valuable first step essential to the treatment of the geometric effects on the absorption coefficient. The effect of geometry must not be overlooked. The amount of energy absorbed by a material of given dielectric constant and resistivity can be greatly increased if the material is formed into a pyramid pointed toward the source of radiation. The various

geometries encountered in the human form will enhance or decrease the energy absorbed, depending on the orientation and the frequency of the radio signal.

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Comments on Papers Delivered at Tri-Service Conference on
Biological Effects of Microwave Radiation

by

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As an ophthalmologist, I was particularly interested in the lack of description of refraction and diffraction in Dr. Silver's presentation on the Physical Aspects of Microwave Radiation. It may well be that these two phenomena are not as important in the microwave band as they are in the visible spectrum. However, microwave propagation does follow optic system patterns and certain unexplained observations may be better understood if so described.

Diffraction, for example, is the optical phenomenon whereby microwave energy can be condensed as it passes through steel wool. It also explains the danger of wearing wire mesh on safety goggles. It may be responsible for the production of side lobes. A system of diffraction grids may prove to be the means of measuring the quality of a microwave field as well as its intensity.

Refraction probably does not occur in the eye. I do not believe that dimensional resonance occurs in the eye, either, because the eyeball is intimately surrounded by other soft tissues which form a similar media. The first discontinuity of media occurs at the bony surface of the orbit. The orbit is shaped like an imperfect concave mirror and it can be assumed that a beam of microwave radiation striking this surface would condense the reflected portion of the beam to form a caustic near the focal point of such a system. This location for the caustic would coincide anatomically with the vitreous humor and it may be a more important factor than the faulty vitreous circulation in producing the temperature gradient within the eye. The seeming discrepancy between Dr. Carpenter's group at Tufts and Dr. Addington's group at Buffalo may be resolved on this basis as they used different wavelengths.

Refraction and the importance of focussing a caustic really came to mind when Dr. Bach presented his fascinating work with monkeys. The animal's bony skull is shaped like an imperfect convex lens and therefore it would focus a beam of transmitted microwave into a caustic instead of a focal point. The distance of the caustic from the incident plane of the skull would vary with the wavelength and thus seemingly small variations in megacycle operation would be significant in locating the distance of the caustic from the surface of the skull. In addition to this, the critical nature of head tilt so that it was necessary to align the roof of the fourth ventricle and the aqueduct of Sylvius in the axis of the caustic was most significant to me. In so doing, all of the neuro-ophthalmological findings

could be explained by a single lesion less than only millimeters in diameter in this region. Nystagmus occurs only in lesions of the nuclei for extra-ocular motility as well as adjacent to the aqueduct of Sylvius. I would experiment performed with an appropriate catheter samples of cerebro-spinal fluid could be drawn and a temperature curve plotted against time.

Before discussing the eye findings, I will give an explanation for the findings exhibited by some contraction or traction of the subcuticular tissues. When the subcuticular circulation is embarrassed, its thermal effect would be enhanced at such a point that were demonstrated. Certainly it would

Dr. Carpenter's work represents a major comparative study of the conditions of exposure of the rabbit eye. There are a few points of variance that I should

The data presented does not necessarily indicate an effect, for the reported conditions of exposure in establishing a minimal thermal exposure to produce the changes detected were of a macroscopic and not

Acute injury of the lens leads first to opacification, providing no lens protein denaturation has taken place. Banding, striations and opacification are ev

Thus protein denaturation is the primary effect and it may begin on a microscopic or submicroscopic level. Fibres may last for many days and if the extent of denaturation has occurred, no permanent reversal is possible. Thermal injury intervenes at a time when the effect may be a summation of effect.

This is in contrast to ionizing radiation. In a normally healthy lens, damage occurs first in the form of developing into lens fibres. The development of these fibres and they migrate subcapsularly to the posterior pole affecting the substance of the lens itself. It may take several days. If the dose of ionizing radiation is sufficient to produce an acute reaction in the lens, however, it affects all of the tissues of the eye.