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BIOLOGIC EFFECTS OF MICROWAVE EXPOSURE

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University of Rochester
Department of Radiation Biology and Biophysics

TECHNICAL REPORT NO. RADC-TR-67-461
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Rome Air Development Center
Air Force Systems Command
Griffiss Air Force Base, New York

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Foreword

This report describes biologic effects of electromagnetic radiation of wavelengths between three meters, (100 Mc)* and one millimeter (300,000 Mc). Results of microwave studies at 200 Mc, 1240 Mc, and 2800 Mc conducted by personnel of the University of Rochester Atomic Energy Project, between 1958 and 1965 are presented.

Evaluation of dosimetry and instrumentation techniques were not an objective of this study; therefore these items will be discussed only where pertinent. Information on physical factors and dosimetry can be obtained from several sources (4,5,66,94,150-156,161,182).

Our observations are amalgamated with information of other investigators to provide a basis for assessing the present "state of the art" insofar as radar hazards are concerned.

The investigations were performed by Joe W. Howland, Sol M. Michaelson, and R.A.E. Thomson with the assistance at various times of Myra Berman, Theodore Elliott, Karl Emilson, Walter Krasavage, Kevin Mahoney and William Quinlan, Jr. Some of the hematologic determinations were performed by Susan Gilt and Kathleen Scheer.

The contributions to this investigation by post-doctoral studies of M. D. Harris, Lt Col, USAF (VC); L. T. Odland, Lt Col, USAF (MC); H. S. Seth, Wg Cmdr, Indian Air Force (Med), and M. L. Tamami, M. D., are acknowledged with appreciation.

The assistance of Mr. Joe Parmentier in maintaining and operating the equipment, and the advice of Prof. Herbert Mermagen and Dr. Joe H. Vogelman are acknowledged with appreciation.

The cooperation of Dr. Alan Keller of the U.S. Army Medical Research Laboratory at Fort Knox, Kentucky, and Dr. C. Osborne of the University of Buffalo, in making their facilities available for some of these studies is greatly appreciated.

Dr. Henry Blair, George M. Knauf, Col, USAF (MC), Robert Zellner, Maj, USAF (MC) (deceased), Mr. Herbert Brownstein and Mr. William Doherty provided the administrative support which was invaluable in these studies.

The critical comments of Dr. Henry Blair, Dr. Joe H. Vogelman, and Lawrence T. Odland, Lt Col, USAF (MC) who reviewed the rough draft of this report are acknowledged with appreciation.

The investigations described in this document were performed at the University of Rochester, Rochester, N.Y., and Rome Air Development Center, Griffiss Air Force Base, Rome, N.Y., under RADC contract AF30 (602)-2921 U.S.

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*In this report, Mc or Mc/sec are used to designate microwave frequency in preference to MHz (the international standard) for the sake of uniformity.

Air Force "dated 1 March 1958" and contract W-7401-eng-49 U.S. Atomic Energy Commission.

This certifies that the investigations under the above contract were conducted according to the "Guiding Principles in the Care and Use of Animals" promulgated by the American Physiological Society.

The RADC Project Engineer is Mr. William J. Doherty, EMEDI.

This final report has been reviewed and is approved.

Approved: WILLIAM J. DOHERTY
Project Engineer
Information Techniques Section

Approved: WILLIAM P. BETHKE
Chief, Engineering Division

FOR THE COMMANDER:
IRVING J. GABELMAN
Chief, Advanced Studies Group

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EVALUATION

Through the efforts of a Tri-Service Ad Hoc Committee, established by the Air Force in 1957, a preliminary tolerance limit was derived and implemented to safeguard personnel from potential hazards associated with exposure to microwave power fields. This limit, expressed as a maximum permissible level of 10 mw/cm^2, was based principally on animal research and theoretical predictions of energy effects on the human body. While it was recognized that the low power density figure would impose an operational burden on military commands employing high power microwave devices, the limitations of available bioeffects data dictated a conservative estimate. Since the need for more substantial biomedical evidence of microwave hazards was apparent, it follows that an essential objective of this program was to acquire, through laboratory experimentation, a basis for validating protective criteria to insure a safe radiation environment for personnel at the least possible cost to military operations.

During the period of study, a considerable number of findings, many of clinical significance, have been compiled. Much has been gained and much remains to be learned. The wealth of data contained in this report comprises pertinent facts gathered from many sources, domestic and foreign, relating to the biological consequences of microwave radiation experiences. These data may be valuable to many investigators for a variety of uses, but the singular most important contribution of the report is to uphold the maximum permissible level of 10 mw/cm^2 as advocated by the Tri-Service Committee. Sufficient evidence is presented to obviate any proposal to increase the tolerance level until more is known about the chronic effects of long-term exposures to low level radiation densities.

It should be understood that the present maximum permissible level of 10 mw/cm^2 is based on average power measurements and defines a safe radiation ambient for unrestricted personnel operations. The possibility of alleviating present restrictions on the use of high power microwave equipment by permitting time limited exposure to power densities exceeding 10 mw/cm^2 remains to be explored. A formidable amount of research is attendant to this approach since variables such as wavelength, energy/time relationships, power distribution characteristics, exposure/recovery cycles, mixed field conditions, external environmental factors and physiological states of the experimental subjects must be critically examined through complex, multivariate procedures. These problems, coupled with the lack of standardized microwave dosimetric techniques, pose an impressive obstacle to the modification of the currently accepted tolerance limits.

This report concludes all work planned by RADC in the area of microwave radiation hazards. Determination of needs for additional study now resides with the Aerospace Medical Division of the United States Air Force.

William J. Doherty

WILLIAM J. DOHERTY
Project Engineer

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1. INTRODUCTION

The development in recent years of radar equipment with peak power in the megawatt range, and communications equipment with effective radiating power of several megawatts, has necessitated evaluation of the biologic hazards of radiofrequency (rf) radiation. In 1956 the Department of Defense, through the U. S. Air Force and the Tri-Service Ad Hoc Committee, undertook a comprehensive analysis of the biological aspects and associated hazards of exposure to microwave radiation. Information current at that time indicated that exposure to microwaves could cause injury in experimental animals, and that no injurious effects had been observed in radar workers. The injury appeared to be of thermal origin and related to the absorption of the microwave energy. The eyes and testes were especially susceptible to injury. Damage to these organs and other biologic effects were associated with a significant rise in body or tissue temperature. It was evident that marked increase in body temperature was the primary hazard whenever the whole or selected areas of the body were exposed to rf. Attempts to establish "safe exposure" levels required consideration of such variables, as frequency, duration of exposure, and field strength. Sufficient reliable data were not available to establish "safe exposure" levels. Based partly on experimental evidence, theoretical considerations and intuitive reasoning, a safe exposure level of 0.01 Watt/cm^2 was established, which represented <u>average</u> power level and <u>not</u> peak power. The maximum power density recommended applies to continuous wave (CW) and pulsed microwave exposure.

The U. S. Air Force, on 1 March 1958 contracted with the Medical Division of the University of Rochester Atomic Energy Project to:

(1) detect and define the injurious effects of acute and chronic microwave exposure.

(2) establish, if possible, a tolerance or safe exposure level for radar workers.

(3) establish, if possible, a biological effect or index in terms of timed exposure at specific power densities.

Initial studies were undertaken to recognize the general biologic responses caused by irradiation from a 2800 Mc pulsed radar unit. This provided base-line data for comparison with studies at other frequencies. Animal response following 1240 Mc pulsed exposure was later investigated. The scope of the investigation was extended in 1961 to include the interaction of microwave and ionizing radiation in mammals.

2. MATERIALS AND METHODS

The studies were primarily conducted at the Verona Test Site facility of Rome Air Development Center, Griffiss Air Force Base. Some investigations were performed at the University of Rochester, the University of Buffalo, the Environmental Temperature Laboratory at Griffiss Air Force Base, and the U.S. Army Medical Research Laboratory at Fort Knox, Kentucky.

Healthy dogs, rabbits, rats, and mice were used. Male and female dogs of mixed breed that weighed between 9 and 18 kg and ranged from 1 to 5 years of age were used. The animals were quarantined for at least 30 days prior to study. During this period, they were immunized against canine distemper and hepatitis. Appropriate anthelmintic therapy was instituted if fecal examination revealed ova or parasites. The dogs were housed singly in roomy cages which permitted them to turn freely. Water was available at all times and a commercial kennel ration was provided once daily. Dogs previously exposed to ionizing radiation (IR) were included for comparative purposes.

At the Verona Test Site two microwave power sources, an AN/FPS-6 operated at 2800 Mc/sec (2750-2880 Mc/sec) and an AN/FPS-8 operated at 1240 Mc/sec (1240-1285 Mc/sec) were used. The AN/FPS-6 was operated at 360 pulses/sec, with a 2 microsecond pulse width, and the AN/FPS-8 at 360 pulses/sec, with a 3 microsecond pulse width. The microwave exposure chamber measured 7 x 7 x 15 ft. and was lined with ECCO-SORB (R) CH-475, that absorbed approximately 98 percent of the incident energy.

Power measurements made with a Ramcor (R) Model 1200 densiometer indicated a relatively uniform microwave field pattern across the animal's body during exposure, with the energy at the periphery differing by less than 20 percent from that at the center.

A double compartment Plexiglas (R) cage 46 inches long, 12 inches wide and 24 inches high, was constructed to confine two dogs separately while they were subjected to 2800 Mc microwaves (Fig. 1). Because of the limits of the 1240 Mc microwave field pattern, a single compartment exposure cage 20 inches long, 20 inches wide and 23 inches in height was used. The animal was free to turn to any position. Adequate ventilation was provided in the exposure cages.

An electronic thermometer (Telethermometer (R)) with appropriate thermistor probes, was used to determine rectal, subcutaneous or skin surface temperature. In studies at 2800 Mc the rectal temperature of dogs was usually monitored continuously from immediately before, during, and on occasion after microwave exposure. This was accomplished by shielding a tubular thermistor probe with Plexiglas and fixing it in the rectum. Because the microwave energy interfered with the operation of the thermistor probe during 200 Mc and 1240 Mc exposures, temperature determinations were made immediately before and after exposure, or during the study period by temporarily suspending the microwave exposure to permit such measurements.

Hematologic tests were performed on blood obtained from the jugular vein.

Dogs that survived several months or years following an LD (50/60) to LD (80/60) dose of ionizing radiation were concurrently exposed to microwaves with normal dogs in the double compartment cage previously described or alone in the single compartment cage. Another group of dogs was exposed to microwaves, four days after Co^{60} irradiation. For comparative purposes additional animals were maintained at ambient temperature of 103.5 to 120° F for periods up to six hours.

DOGS IN DOUBLE COMPARTMENT PLEXIGLAS CAGE IN ANECHOIC CHAMBER

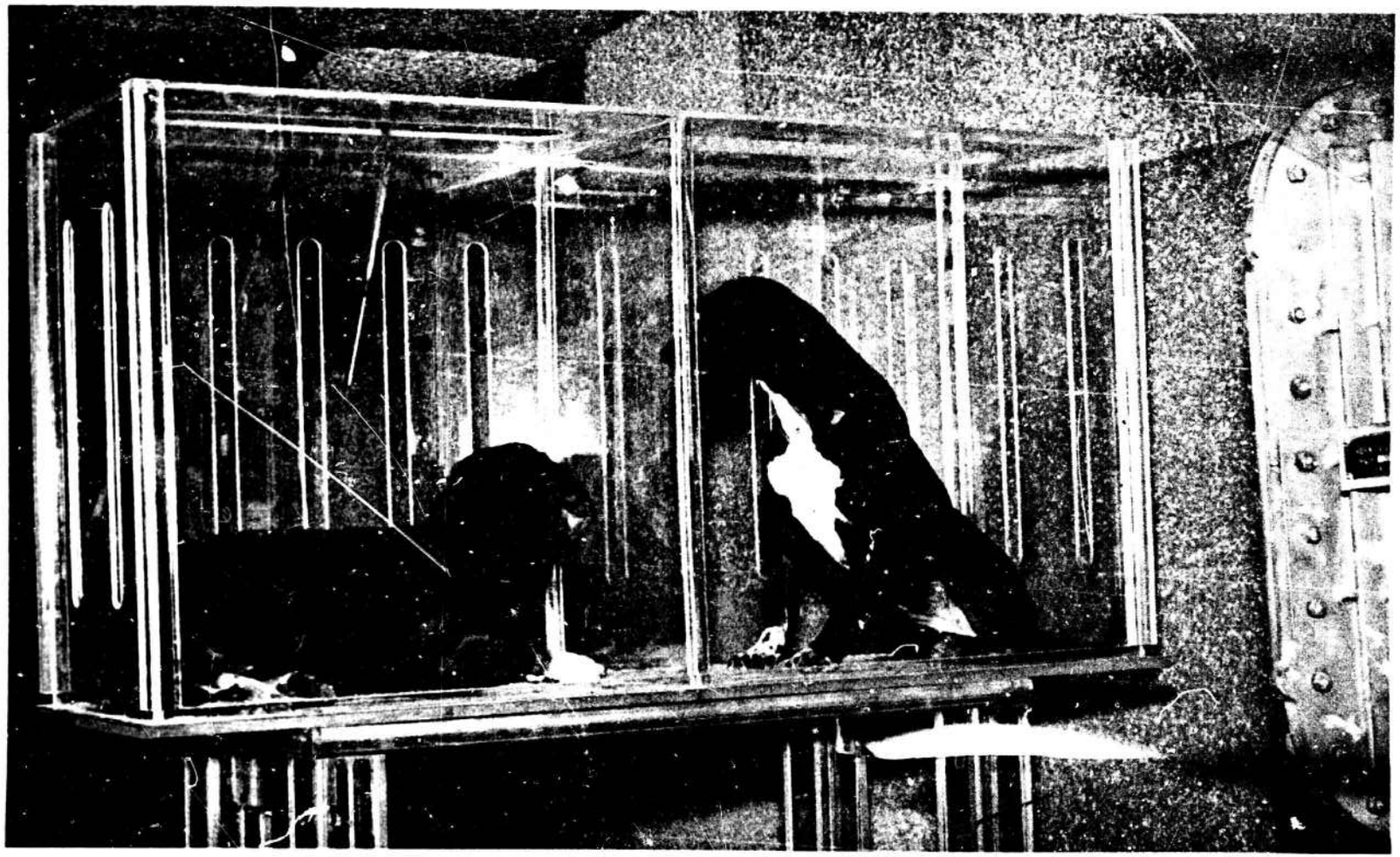


Fig. 1

Rodents were individually confined in a multi-compartment Plexiglas cage that faced the horn during exposure. Each compartment was 6 inches long, 4-1/2 inches wide, 4-1/2 inches high with holes for ventilation. The animals were within a field 30 inches wide and 22-1/2 inches high.

In one study, pairs of dogs were exposed simultaneously to 165 mW/cm^2. During each exposure the dog that developed a rectal temperature of 106°F earliest, was at that time, provided with water ad libitum. The other was not permitted access to water. Approximately 200 ml aliquots of water at 50° F were delivered through Tygon (R) tubing into a Plexiglas cup positioned in a corner of the cage. At all times, approximately 100 ml of water was left in the cup. To improve palatability, water was siphoned off and replaced as it became warm. At arbitrarily chosen times during and at the termination of the exposure, each animal was weighed to within 10 g, and blood obtained for hematocrit determination.

Antibody half-life and disappearance rate of passively transferred antibodies were determined in rabbits that were exposed to 2880 Mc at 100 mW/cm^2 until a critical rectal temperature was reached. Antigen in the form of sheep red blood cells (RBC) was given iv to rabbits two days or four hours before or two days after microwave irradiation. Animals that received sheep RBC but were not subjected to microwaves, served as controls.

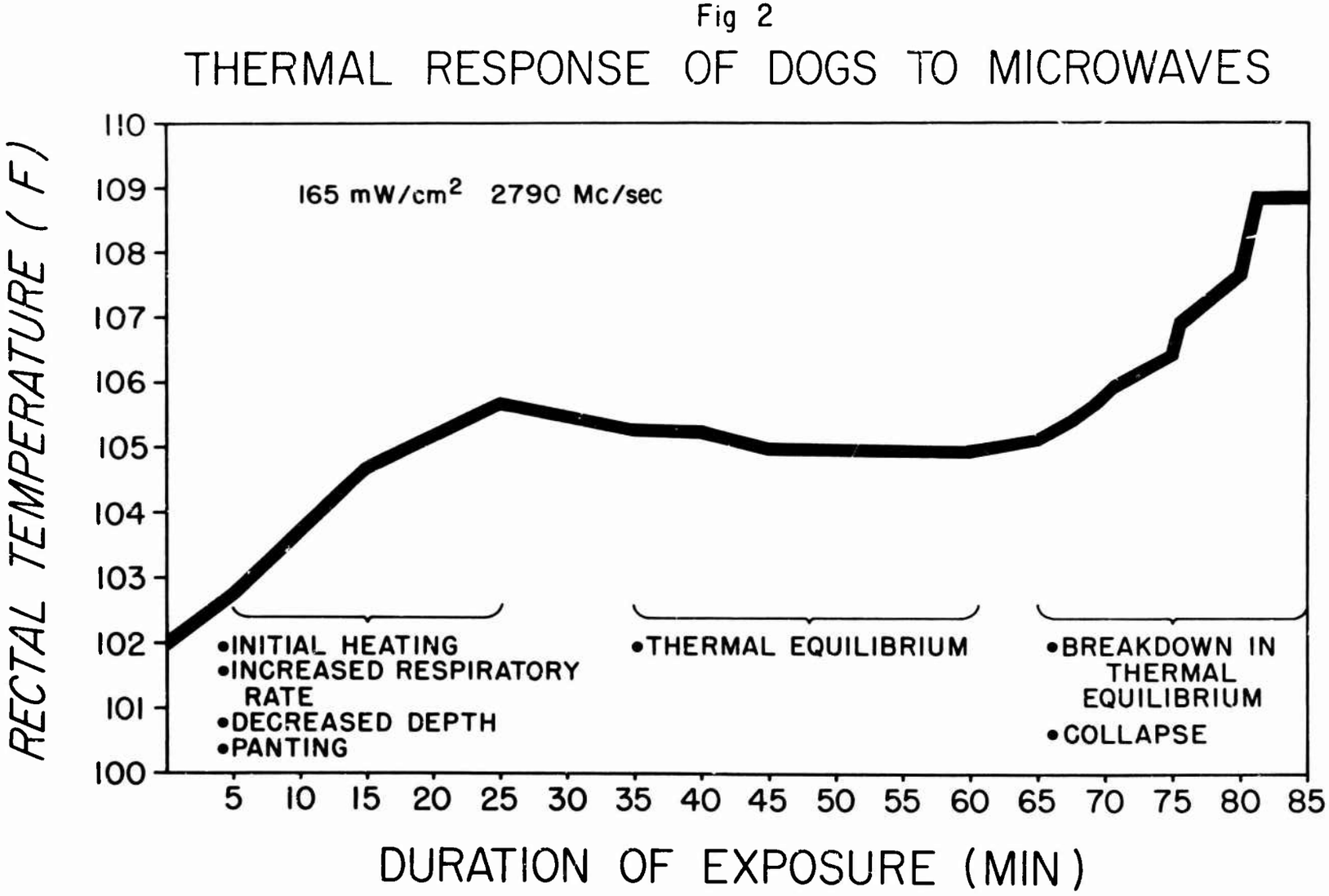
Studies at the University of Buffalo utilized a 200 Mc continuous wave (CW) unit with a helical antenna. At the University of Rochester, a bench-model 2800 Mc (CW) generator was used.

3. RESULTS

A. Thermal Regulation

During exposure to 2800 Mc, 165 mW/cm^2 (Fig. 2), dogs exhibited an initial period of heating during which the rectal temperature increased two to three degrees F within 40 to 60 minutes after start of the exposure. Panting usually began as soon as the exposure started, and became more marked as the exposure continued. Respiration rate increased and the animals became restless. This was followed by a period of thermal equilibrium for about an hour, which was characterized by a cyclic variation in rectal temperature between 105 and 106° F. During the period of thermal equilibrium, the animals appeared comfortable with increasing and decreasing rate of panting that was rhythmical in pattern. Following the equilibrium phase, at the onset of breakdown in thermal regulation there was a striking increase in rectal temperature with clinical signs of hyperpyrexia such as increased salivation, impaired locomotion, and excitability. Acute distress and collapse resulted when the rectal temperature exceeded 107°F. Upon removal from the cage within 15 minutes after cessation of the exposure the animal could stand, but displayed prominent weakness of the hind quarters with staggering and incoordination. Except for animals that exhibited the most marked responses after exposure, there was generally an increased desire for water. Temperature recovery was exponential and within an hour post-exposure values approximated the pre-exposure level. When death occurred, it was usually within 30 minutes

Fig 2


THERMAL RESPONSE OF DOGS TO MICROWAVES
RECTAL TEMPERATURE (F)
110
109
108
107
106
105
104
103
102
101
100
165 mW/cm2 2790 Mc/sec
•INITIAL HEATING
•INCREASED RESPIRATORY RATE
•DECREASED DEPTH
•PANTING
•THERMAL EQUILIBRIUM
•BREAKDOWN IN THERMAL EQUILIBRIUM
•COLLAPSE
5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85
DURATION OF EXPOSURE (MIN)

after termination of the exposure; rigor mortis developed rapidly.

Exposure to 100 mW/cm^2 for periods up to six hours, (an arbitrarily selected time based on physical and technical support factors), did not result in a critical rise in rectal temperature. The initial period of heating resulted in a one to two degree rise in rectal temperature. The animal thereafter remained in thermal equilibrium.

Rectal temperature in dogs exposed to 1240 Mc, was comparable to dogs exposed to 2800 Mc, at similar microwave field intensities. The response varied little from the normal state until a critical rectal temperature level was approached, when a moderate increase in panting was noted. The animals, however, remained comfortable or slightly agitated; salivation was normal or slightly increased. Because of inactivity during exposure, impairment of locomotion could not be ascertained until the animal was removed from the exposure cage.

At 200 Mc, 165 mW/cm^2, the dog equilibrated later than at 2800 Mc and remained in equilibrium for a longer period (five hours average), before thermal breakdown was evident (Fig. 3). Frequent interruptions necessary during the exposure to facilitate temperature measurements may have influenced the duration of the equilibrium period. Small and large dogs responded similarly.

Rectal temperature of dogs in a 103.5-106^o F, 20 percent humidity environment, was stable during a six hour period. With exposure to 100 mW/cm^2, 2800 Mc at this increased ambient temperature the rectal temperature increased to a critical level within one hour (Fig. 4). Exposure to 165 mW/cm^2 at increased ambient temperature resulted in the development of a critical rectal temperature within a half hour (Fig. 5).

Dogs displayed a longer equilibrium period at 200 Mc than at 2800 Mc and lack of equilibration at 120^o F, 50 percent humidity (Fig. 6). The response of different species to various exposures is shown in Figures 7, 8, and 9. The rabbit is more sensitive to exposures at 2800 Mc than to exposures in a hot environment, or at 200 Mc (Fig. 7). The rat could better tolerate 200 Mc exposure than 2800 Mc or 120^o F ambient temperature (Fig. 8).

Body size did not seem to be a factor in the thermal response at 2800 Mc 165 mW/cm^2. Both rat and rabbit responded similarly. A critical rectal temperature was usually reached in ten minutes in the rabbit and 20 minutes in the rat with no equilibration. Fox terriers, equal in body weight to rabbits, (about 4 kilograms), responded in the same way as dogs that weighed between 8 and 20 kilograms, (Fig. 9).

In studies at 2800 Mc in which skin surface temperature was measured, the temperature difference between the side facing the horn, and the rectal temperature, became smaller during the first 30 minutes of exposure, then stabilized while the rectal temperature continued to increase (Fig. 10).

The "Thermal Circulation Index" of three dogs was calculated using the

Fig 3

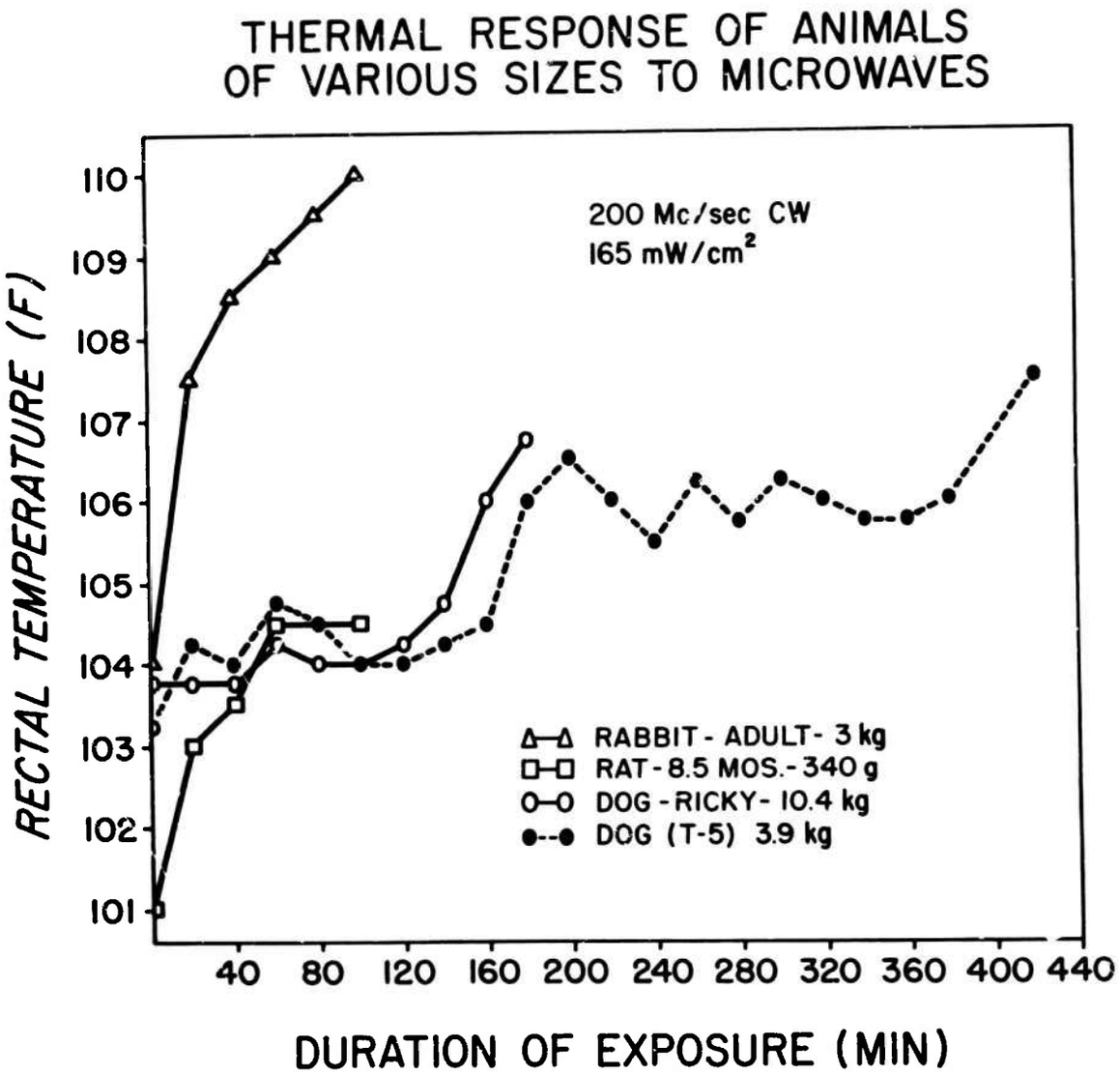

THERMAL RESPONSE OF ANIMALS
OF VARIOUS SIZES TO MICROWAVES
200 Mc/sec CW
165 mW/cm²
RECTAL TEMPERATURE (F)
110
109
108
107
106
105
104
103
102
101
RABBIT - ADULT - 3 kg
RAT - 8.5 MOS. - 340 g
DOG - RICKY - 10.4 kg
DOG (T-5) 3.9 kg
40 80 120 160 200 240 280 320 360 400 440
DURATION OF EXPOSURE (MIN)

Fig 4

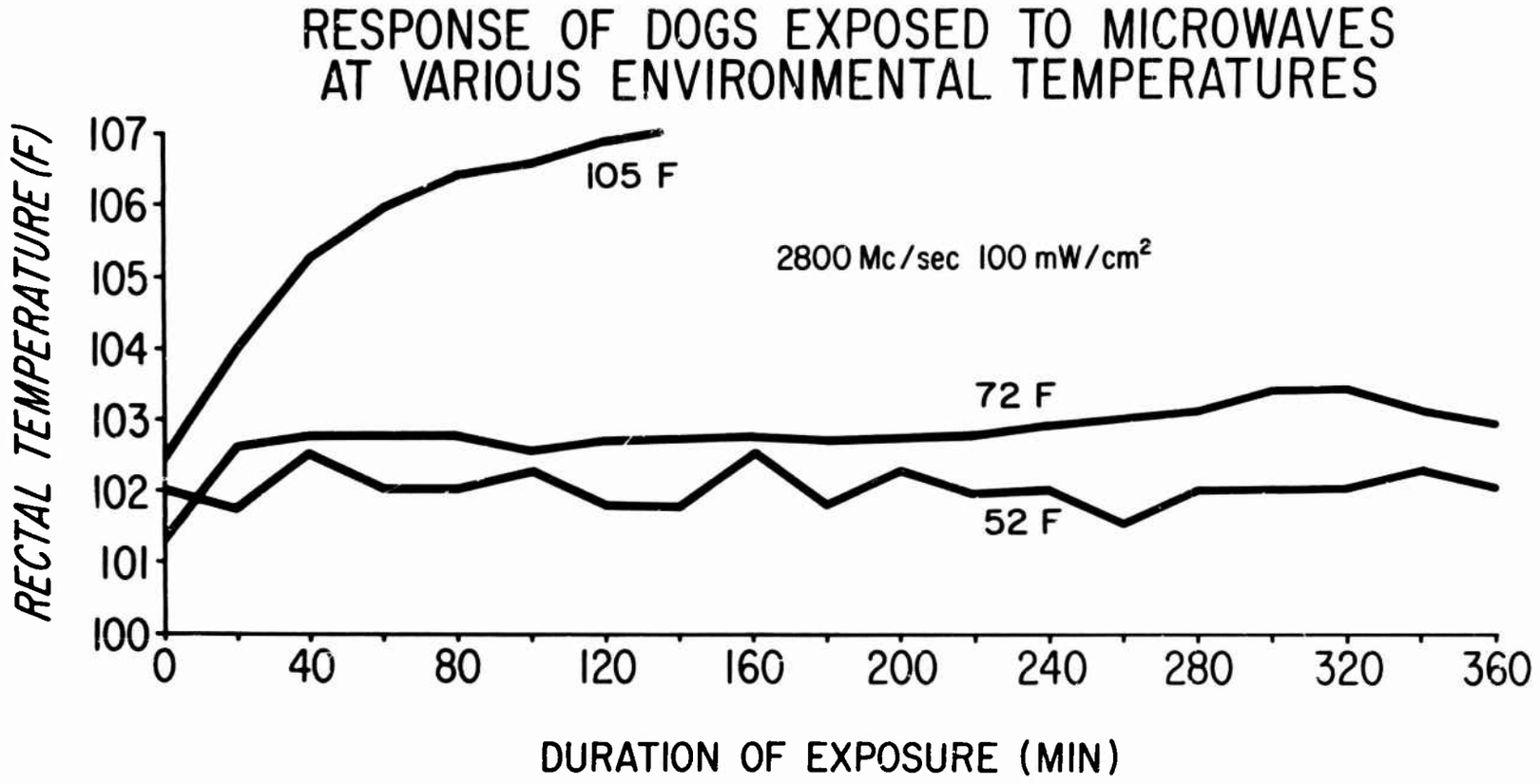

RESPONSE OF DOGS EXPOSED TO MICROWAVES
AT VARIOUS ENVIRONMENTAL TEMPERATURES
RECTAL TEMPERATURE (F)
107
106
105
104
103
102
101
100
105 F
2800 Mc/sec 100 mW/cm²
72 F
52 F
0
40
80
120
160
200
240
280
320
360
DURATION OF EXPOSURE (MIN)

Fig 5

RESPONSE OF DOGS EXPOSED TO MICROWAVES
AT VARIOUS ENVIRONMENTAL TEMPERATURES

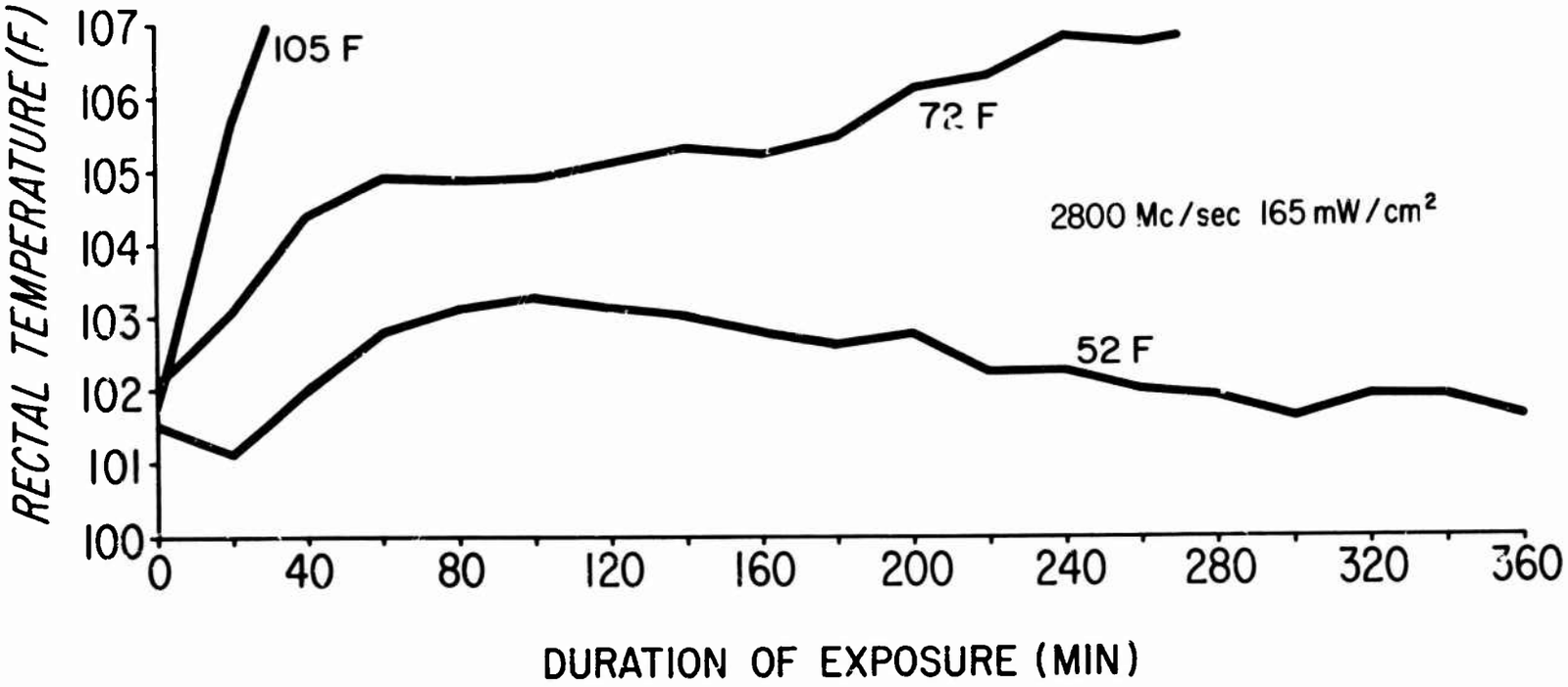


Fig 6

THERMAL RESPONSE OF DOGS EXPOSED TO MICROWAVES OR INCREASED ENVIRONMENTAL TEMPERATURE

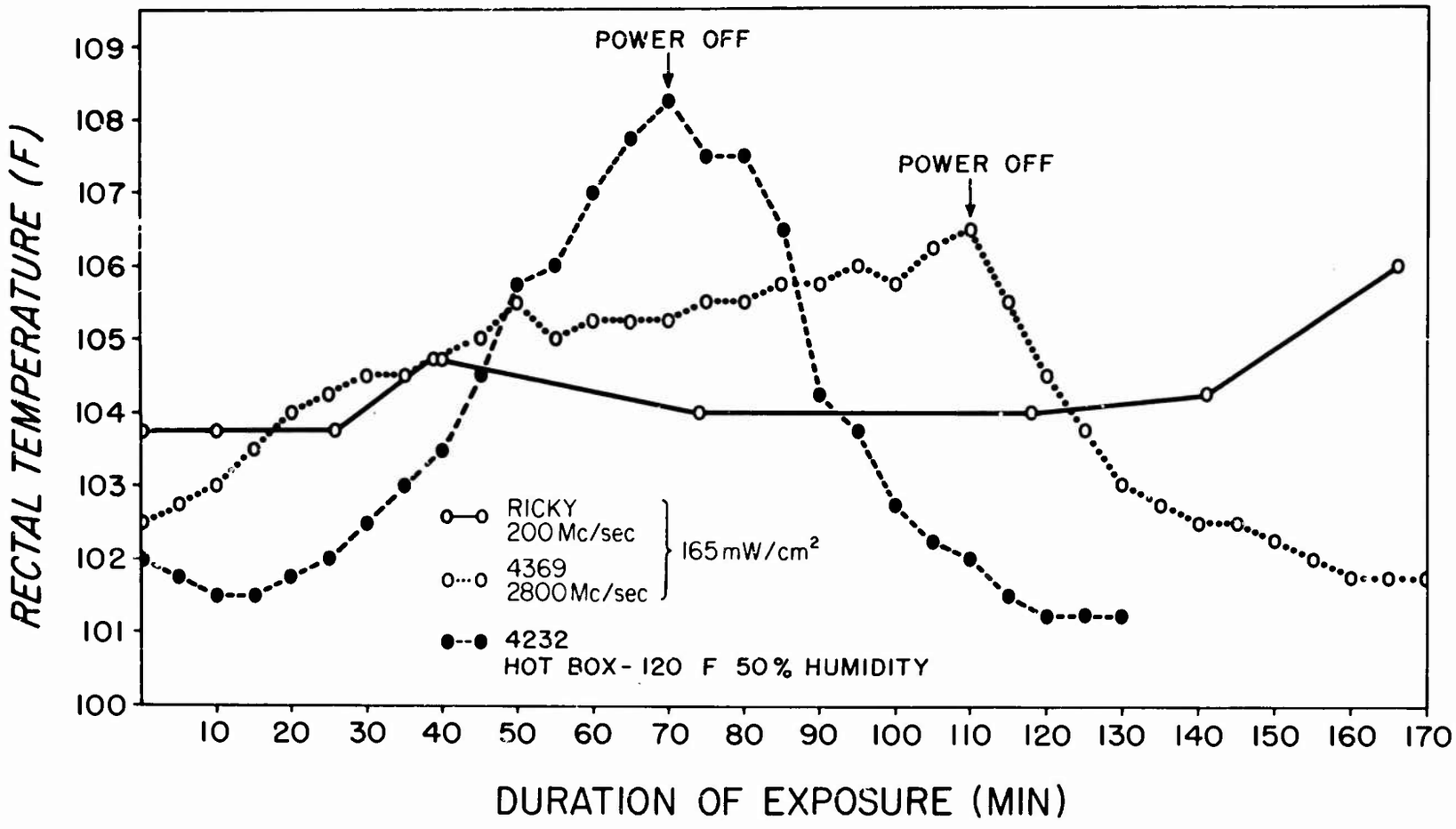


Fig 7

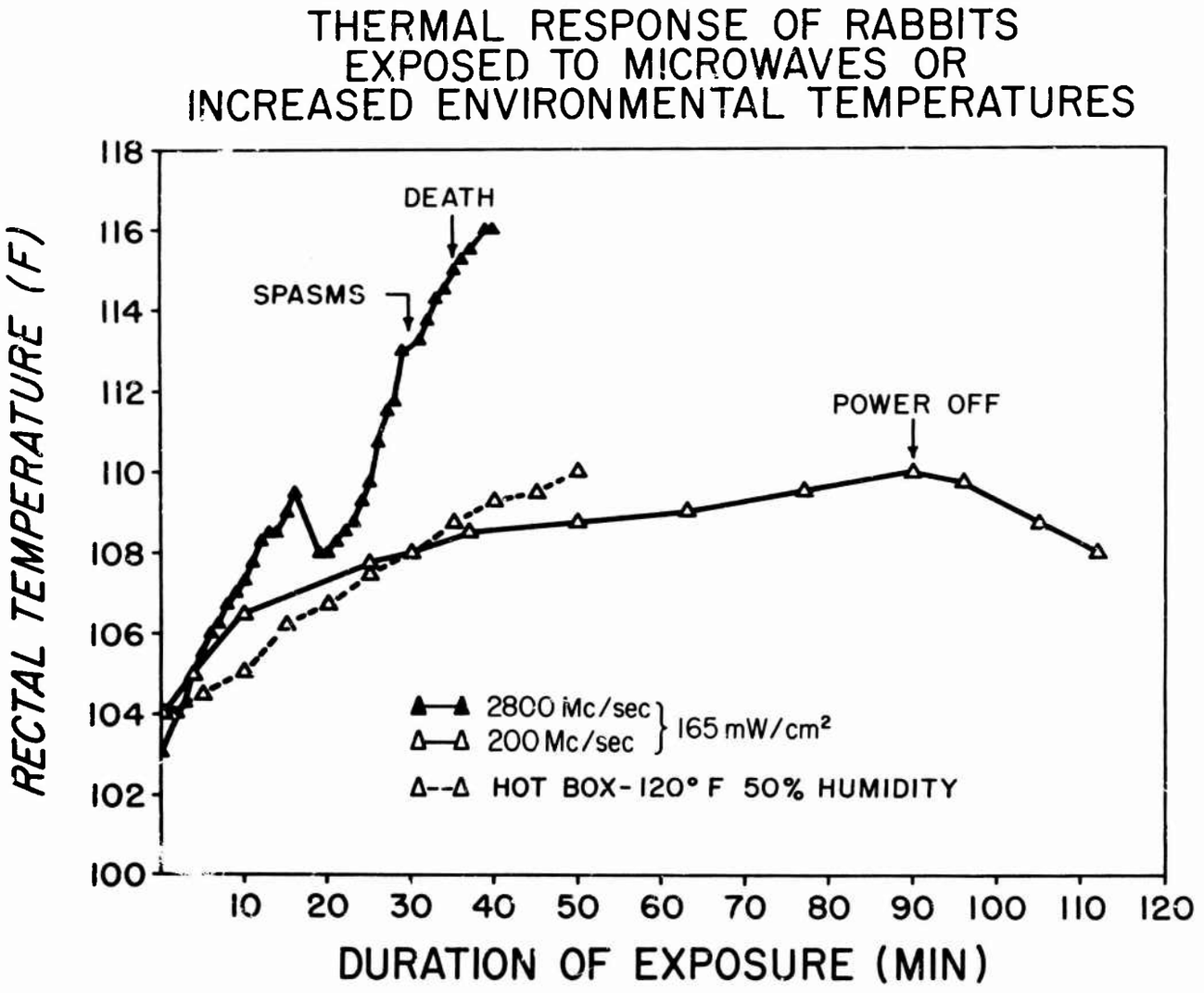

THERMAL RESPONSE OF RABBITS
EXPOSED TO MICROWAVES OR
INCREASED ENVIRONMENTAL TEMPERATURES
RECTAL TEMPERATURE (F)
118
116
114
112
110
108
106
104
102
100
DEATH
SPASMS
POWER OFF
2800 Mc/sec
200 Mc/sec
165 mW/cm²
HOT BOX - 120° F 50% HUMIDITY
10 20 30 40 50 60 70 80 90 100 110 120
DURATION OF EXPOSURE (MIN)

Fig 8

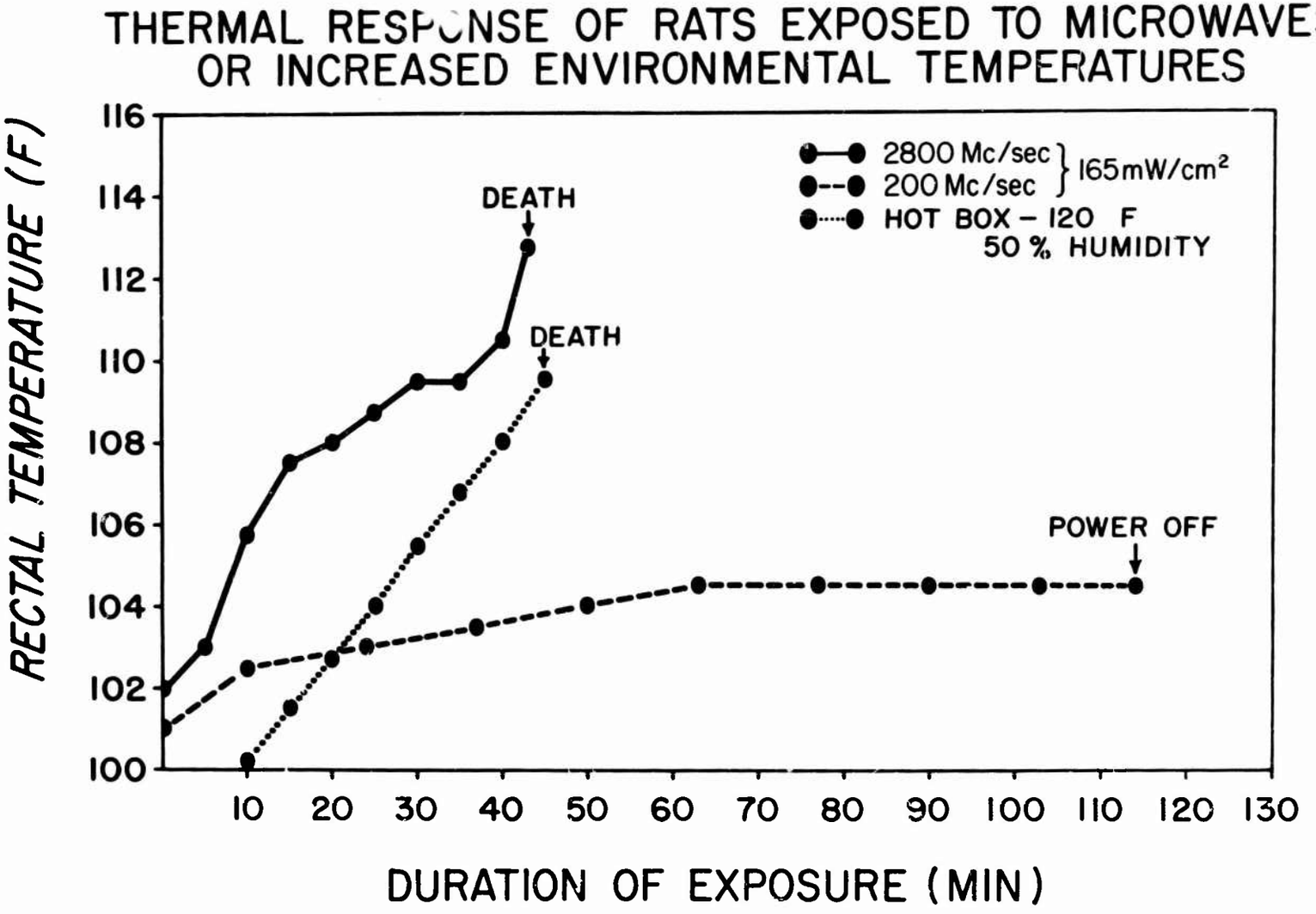

THERMAL RESPONSE OF RATS EXPOSED TO MICROWAVES
OR INCREASED ENVIRONMENTAL TEMPERATURES
RECTAL TEMPERATURE (F)
116
114
112
110
108
106
104
102
100
2800 Mc/sec
200 Mc/sec
165mW/cm²
HOT BOX – 120 F
50 % HUMIDITY
DEATH
DEATH
POWER OFF
10 20 30 40 50 60 70 80 90 100 110 120 130
DURATION OF EXPOSURE (MIN)

Fig 9

THERMAL RESPONSE OF VARIOUS SPECIES OF ANIMALS EXPOSED TO MICROWAVES

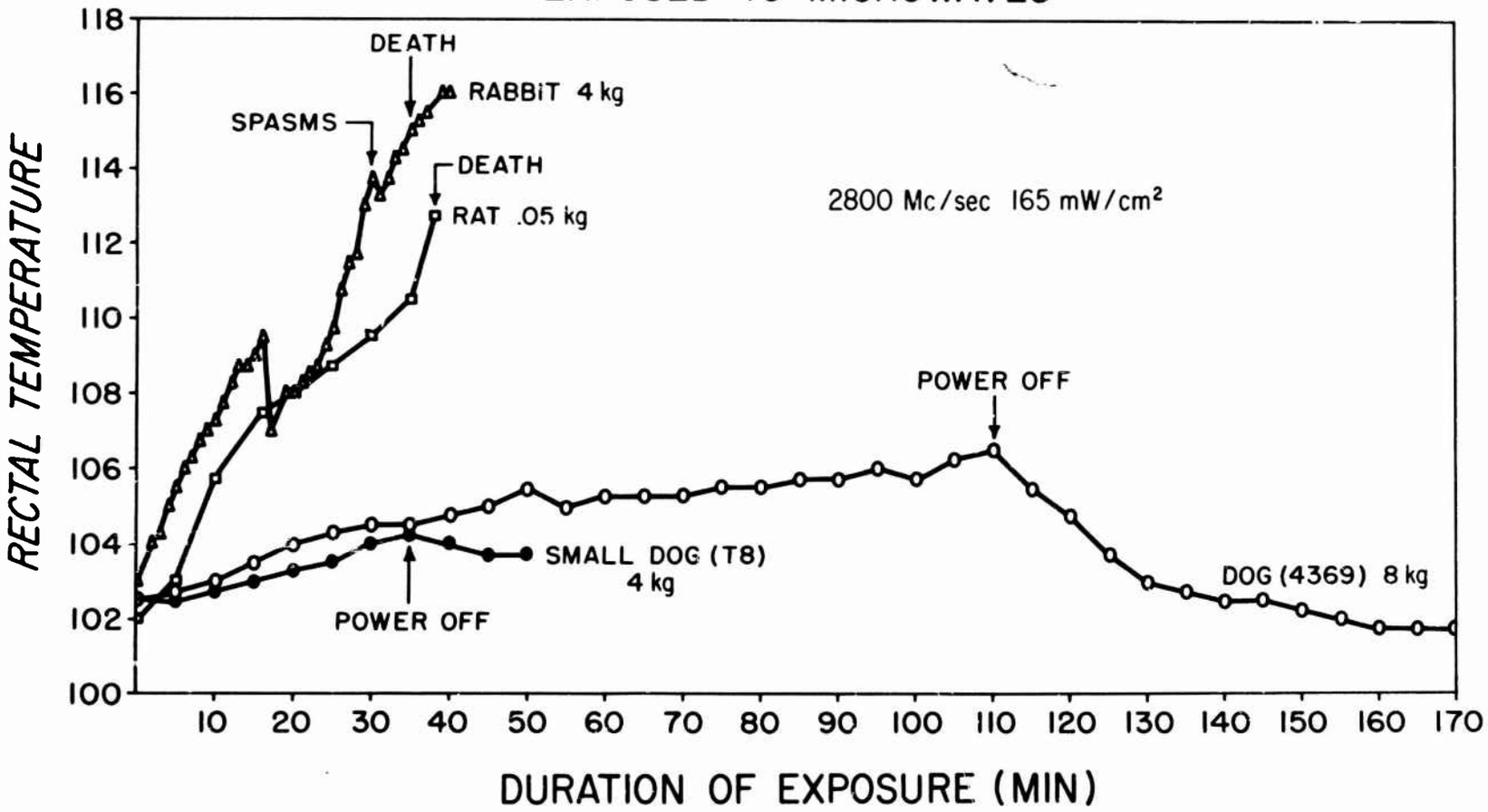
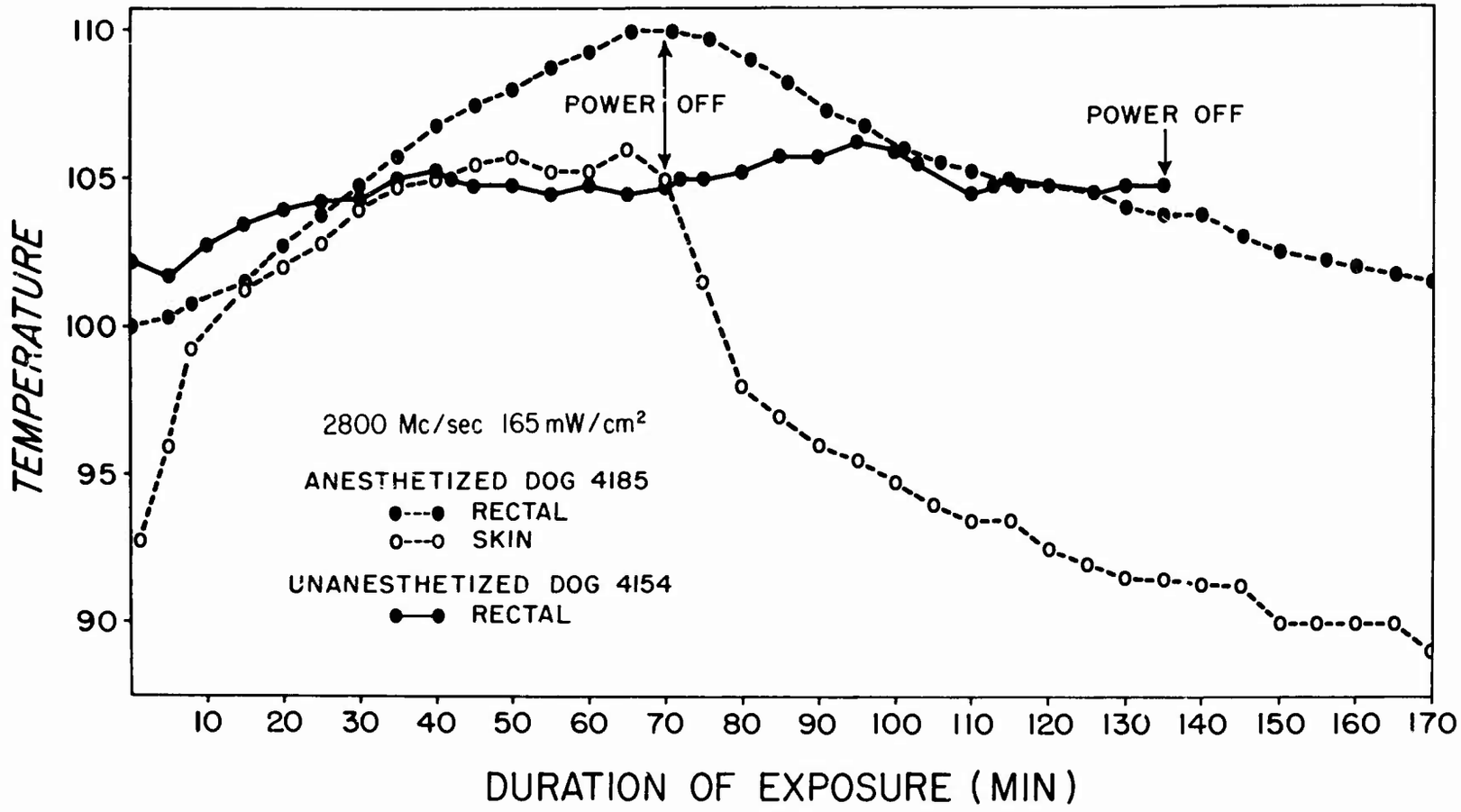


Fig 10

RELATIONSHIP BETWEEN RECTAL AND SKIN TEMPERATURE
DURING EXPOSURE TO MICROWAVES



formula $\frac{Ts-Te}{Ti-Ts} = \frac{\text{Skin Temp - Ambient Temp}}{\text{Rectal temp - Skin Temp}} = \frac{\text{Physical Gradient}}{\text{Physiological Gradient}}$ (160). The difference in thermal circulation index reflects the depth of microwave energy absorption of 200 Mc and 2800 Mc (Fig. 11).

B. Pathologic Effects

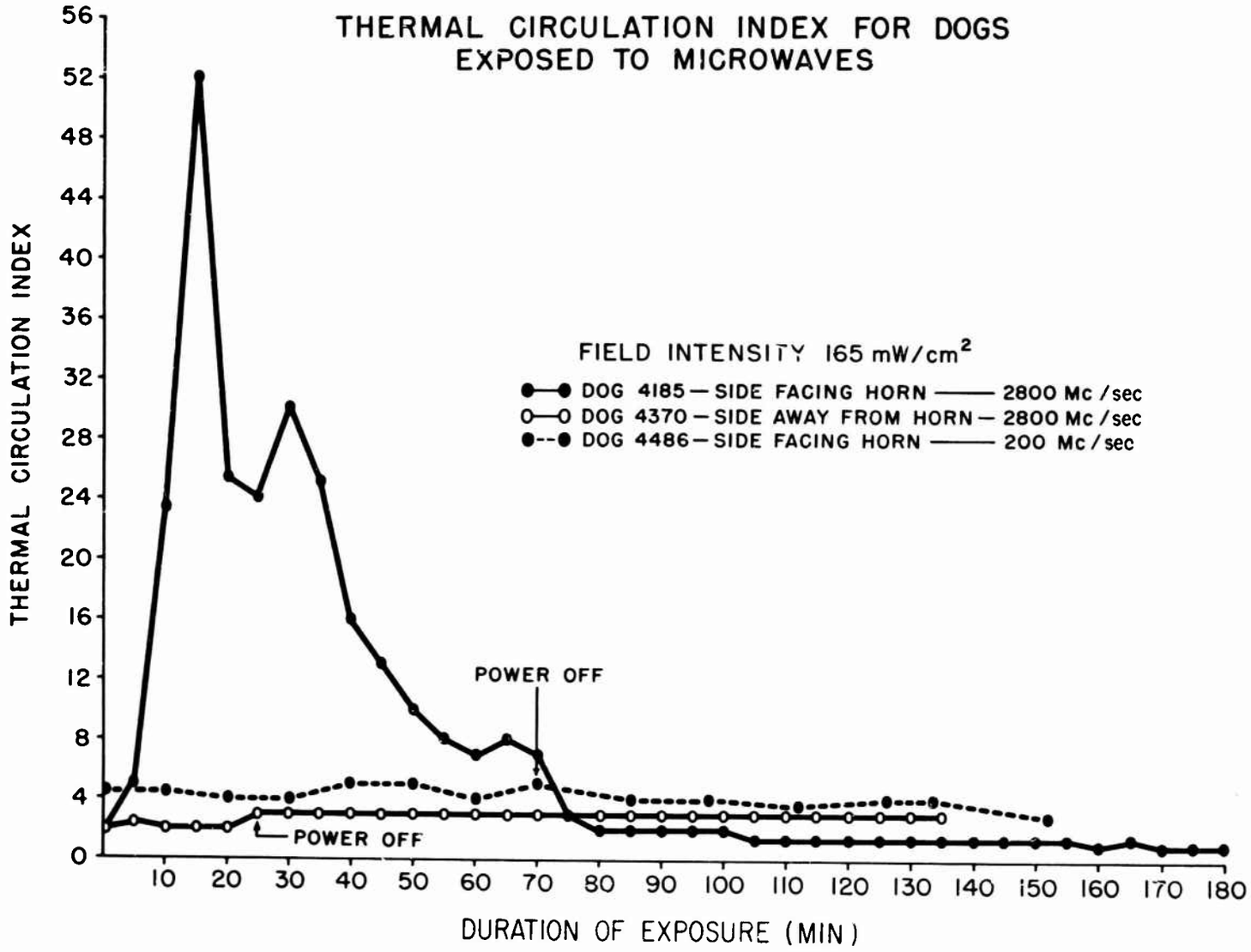
Superficial and deep burns occasionally developed after exposure to 2800 Mc at 165 mW/cm^2. Although the burns occurred in various portions of the body, the lateral aspect of the rib cage seemed most susceptible to this injury (Fig. 12). A period of up to six days in normal dogs, and ten in dogs previously exposed to X-irradiation (IR) elapsed following exposure before the burn was evident. A deep clean wound, identical in appearance with a third degree burn, resulted. The central portion of the burn appeared devitalized with development of a process that suggested dry gangrene, prior to sloughing. Healing was slower in the IR survivor dogs and was accompanied by considerable suppuration. In the others, suppuration was negligible. No scarring or keloid development was noted. A dog with burns that healed in three weeks was re-exposed six weeks after the initial exposure. Burns recurred in the original site as well as in a new one. This response may indicate differential sensitivity of various parts of the body.

Dogs under pentobarbital anaesthesia, when positioned in ventral recumbency with legs extended and side facing the aperture of the microwave generator horn, developed burns in the skin-fold between the trunk and the limbs of the exposed side, which was moderately taut due to the position of the animal. Anaesthetized dogs were susceptible to burns from exposures as short as 30 minutes. Since non-anesthetized animals had freedom of movement during exposure, change in position permitted one side to cool, while the other heated. Under such conditions devitalization of a vascularly poor area is minimized before the period of thermal breakdown is reached. Factors of importance in the production of these burns include rate and/or duration of heating, vascularization of the exposed area, anaesthesia, movement, and specific sensitivity of the animal.

Histologic sections from areas with burns were examined by Dr. Charles Yuile, of the University of Rochester. His findings were: "Changes noted at the earliest times post-exposure (1/2 hour, 1-1/2 hours, and 1 day) are mild and consist of edema and congestion which is, if anything, more marked in the deeper tissues (fat and muscle) than in the dermis. At 5 days, the normal dog showed changes as above, but somewhat more marked, while the X-radiation survivor showed normal skin and underlying tissue. At 7 days, the normal dog showed moderate, focal necrosis in the dermis, vesiculation of the epidermis and marked inflammatory and degenerative changes in the fat and muscle. The survivor of X-radiation at 7 days showed extensive, deep tissue destruction with ulceration and eschar formation. At 14 days, a normal dog also showed a deep ulcerating lesion, while an X-ray survivor was normal. At 15 days, both normal dog and X-ray survivor had deep ulcerative lesions.

It is noteworthy that between 5 and 15 days, degenerative muscle changes tended to be most severe at a considerable distance lateral to the areas of

Fig 11


THERMAL CIRCULATION INDEX FOR DOGS
EXPOSED TO MICROWAVES
THERMAL CIRCULATION INDEX
56
52
48
44
40
36
32
28
24
20
16
12
8
4
0
FIELD INTENSITY 165 mW/cm²
DOG 4185 — SIDE FACING HORN —— 2800 Mc/sec
DOG 4370 — SIDE AWAY FROM HORN — 2800 Mc/sec
DOG 4486 — SIDE FACING HORN —— 200 Mc/sec
POWER OFF
POWER OFF
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
DURATION OF EXPOSURE (MIN)

SKIN BURN IN DOG PRODUCED BY MICROWAVES
(2800 Mc/sec, 100 mW/cm^2, 6 hrs)

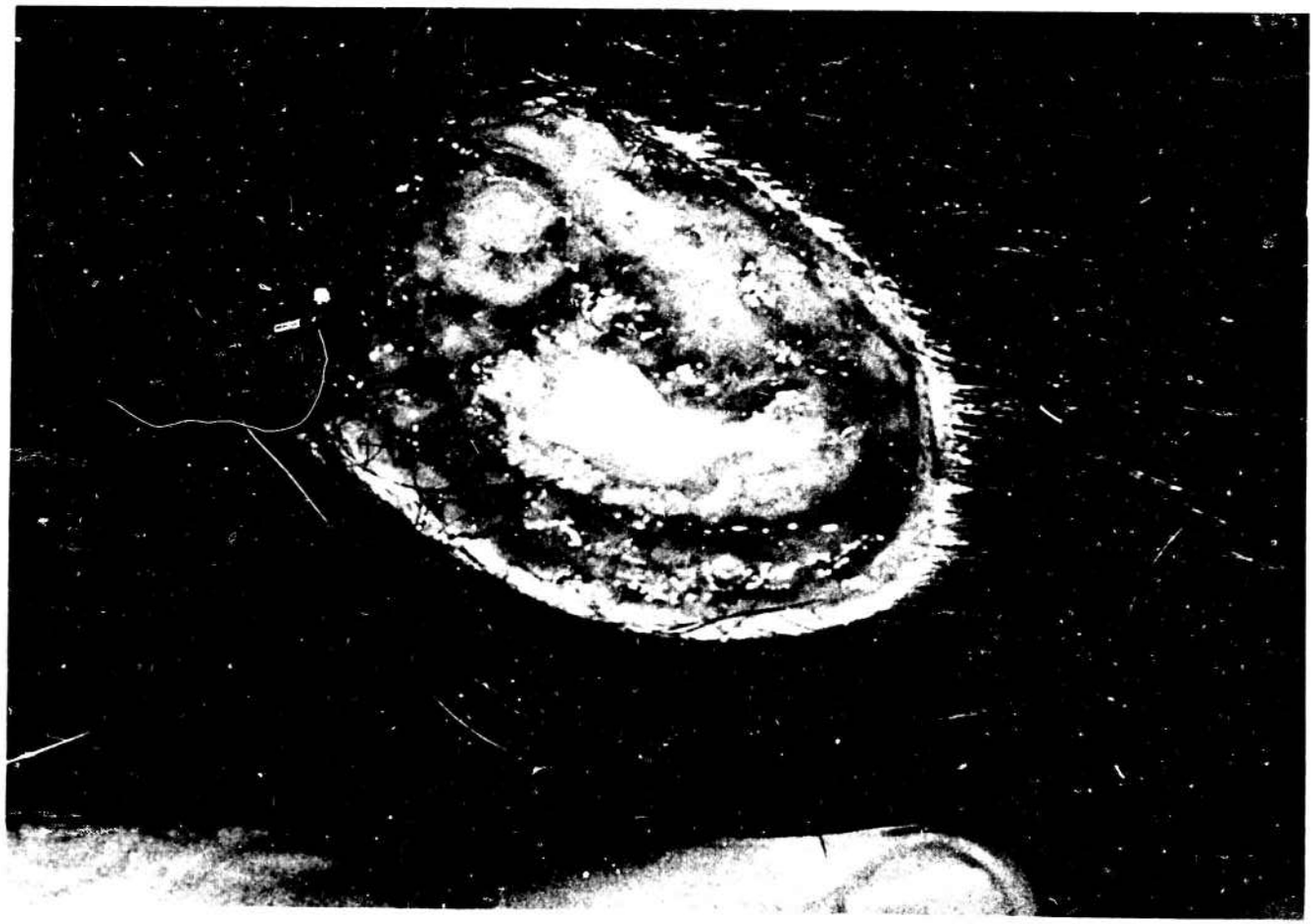


Fig. 12

more superficial damage. This is illustrated in Figure 13 and 14."

Dogs were euthanized immediately after exposure and at 1, 7, 28, and 84 days after two to three hours exposure to 2800 Mc, at 165 mW/cm^2. Necropsy revealed generalized congestion of most organs, particularly the liver, spleen, kidneys, and lungs. The testes, brain, pancreas, and heart were slightly to moderately congested. The gastrointestinal tract was generally cyanotic and the mucosa of the small intestine had scattered areas of inflammation and hemorrhage. Grossly, the blood seemed darker red than normal with little tendency to clot. Tissue sections from three of these dogs were examined by Dr. Frank W. Hartman (Office of the Surgeon General, USAF). Report on one dog that died immediately after exposure was: "Sections of all organs and tissues shown in the four microscopic slides are congested. There is nothing else definitely pathological with the possible exception of the degeneration of the Purkinje cells in the cerbellum and a few of the cells in the basal nuclei. The changes that are seen (in C7305-1) are comparable in every way to changes seen in animals and man exposed to hyperthermia ranging from 106°F to 108°F of four to five hours duration. There is nothing to differentiate these tissue changes following exposure to microwaves with hyperthermia from the changes seen in hyperthermia and hypoxia observed in animals and man following fever therapy." Sections from dogs 4 weeks and 12 weeks after exposure revealed no definite pathologic changes.

C. Hematology

Whole-body exposure of normal dogs to microwaves resulted in leukocyte changes related to microwave frequency, field intensity, and duration of exposure. There was a marked decrease in lymphocytes and eosinophils after six hours, 2800 Mc, 100 mW/cm^2 exposure (Fig. 15). The neutrophils remained slightly increased at twenty-four hours, while eosinophil and lymphocyte values returned to normal levels. Following two hours of exposure at 165 mW/cm^2, there was a slight leukopenia and decrease in neutrophils. When the exposure was of three hours duration, leukocytosis was evident immediately after exposure. The leukocytosis was more marked at twenty-four hours, and reflected the neutrophil response. There was a moderate decline in lymphocytes immediately following two to three hours of exposure, with recovery to the pre-exposure level at twenty-four hours. Following two hours of exposure, there was a slight decrease in eosinophils which was unchanged at twenty-four hours. Eosinophil change was negligible at the termination of three hours of exposure, and moderately decreased at twenty-four hours.

After exposure to 1285 Mc, 100 mW/cm^2, for six hours, there was an increase in leukocytes and neutrophils. At twenty-four hours the neutrophil level was still noticeably increased from the pre-exposure level. Lymphocyte and eosinophil values were moderately depressed. At twenty-four hours, they slightly exceeded their initial value.

Six hours of exposure to 200 Mc, 165 mW/cm^2 resulted in a marked increase in neutrophils and a mild decrease in lymphocytes. The leukocyte count was

SECTION OF SKIN (X35) FROM SITE OF MICROWAVE BURN
(2800 Mc/sec, 165 mW/cm^2, 6 hrs)

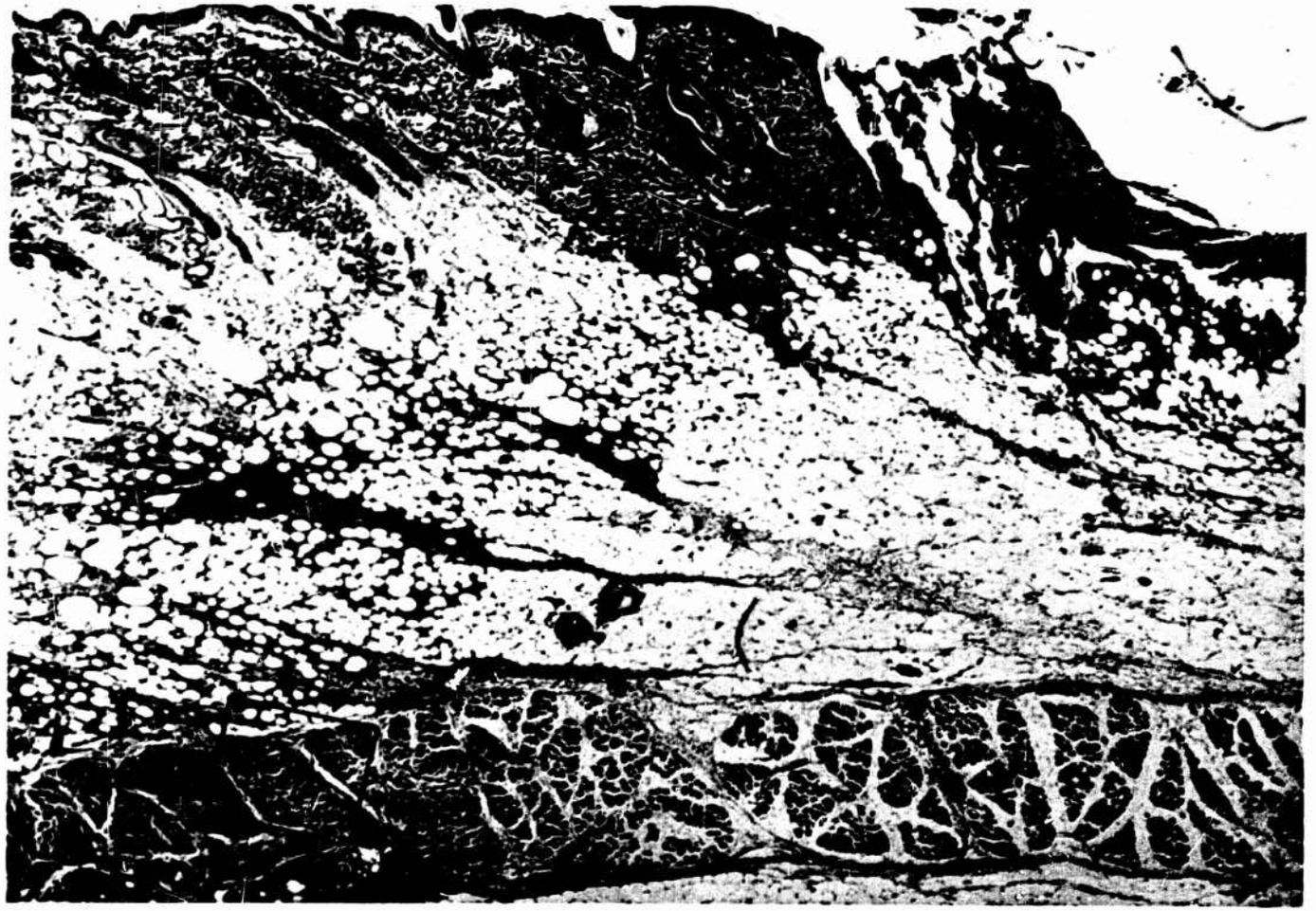


Fig. 13

BURN AREA "A" (X270) FROM SITE OF MICROWAVE BURN (Fig. 13)

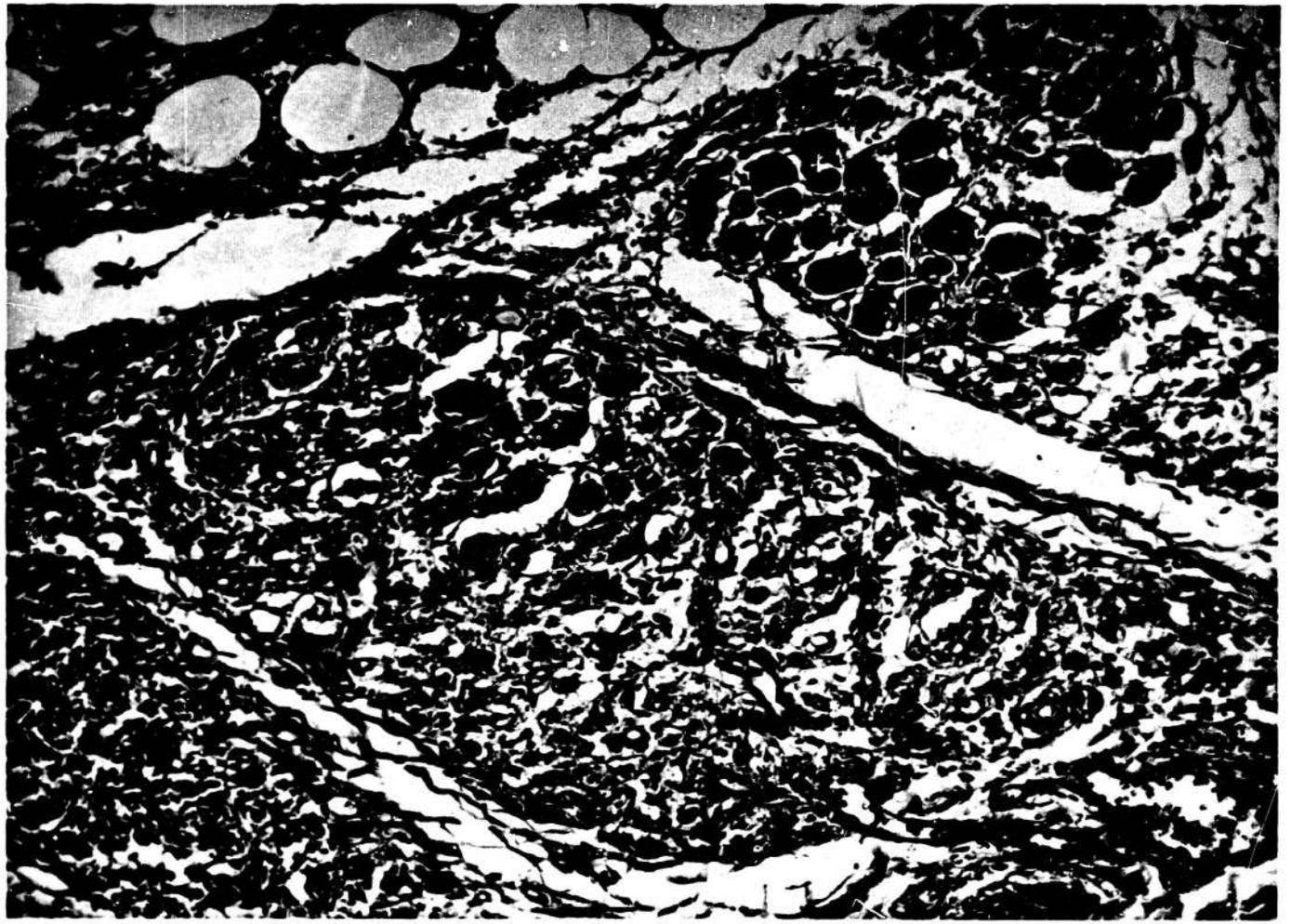
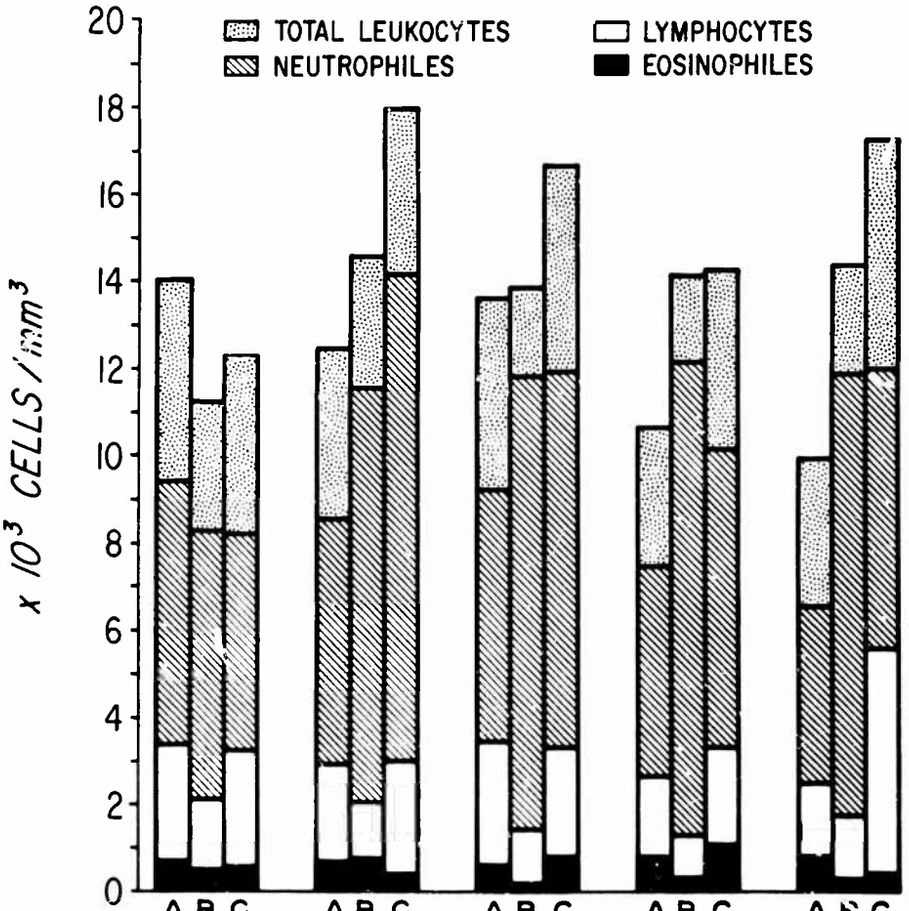


Fig. 14

Fig 15

EFFECT OF MICROWAVES ON WHITE CELL COUNT OF DOGS



FREQUENCY (Mc/sec)	2800	2800	2800	1285	200
FLUX DENSITY (mW/cm²)	165	165	100	100	165
HOURS EXPOSED	2	3	6	6	6

A=INITIAL VALUE B=IMMEDIATELY POST EXPOSURE C=24 HOURS

further increased, and the lymphocytes markedly increased the following day. Eosinophils were moderately decreased.

Comparison of leukocyte changes over a sixty day period after a six hour exposure at 100 mW/cm^2, 2800 Mc or 1285 Mc revealed that 1285 Mc had a slightly greater and more prolonged effect on leukocyte response (Tables IA, IB, Fig. 16). Recovery of leukocytes and neutrophils to the pre-exposure level occurred one to two weeks after 1285 Mc and within one week after 2800 Mc exposure. A 25 to 40 percent lymphocyte increase from the pre-exposure level was noted from one day to two years after 1285 Mc exposure (Tables IA, IB, IIA, IIB). The reticulocyte count was moderately diminished during this period. Lymphocytopenia from 2800 Mc was followed by recovery to 95 percent of the initial value in 24 hours and a gradual decrease to 54 percent of the initial value by sixty days.

Daily exposures to 1285 Mc resulted in changes related to the microwave field strength (Figs. 17-20). The changes were ill-defined at 20 mW/cm^2, better defined at 50 mW/cm^2 and very definite at 100 mW/cm^2. During the first week of exposure neutrophil increase was very evident. Lymphocytes were decreased following 50 mW/cm^2 and 100 mW/cm^2. A slight lymphocytosis following repeated daily exposure at 20 mW/cm^2, persisted up to four months after the last exposure. Hemoconcentration resulted from 100 mW/cm^2 and hemodilution from 20 mW/cm^2 and 50 mW/cm^2. A more marked decrease in hematocrit was seen after each 20 mW/cm^2 and 50 mW/cm^2 exposure. Decreased hematocrit and reticulocytosis also occurred in dogs subjected to daily 2800 Mc exposure at 100 mW/cm^2. There was a progressive decline in pre-exposure hematocrit values during 50 mW/cm^2 and 100 mW/cm^2 exposures. Correlated with the decline in hematocrit was a decrease in red cell count, and an increase in reticulocytes. Following the final exposure, there was a transitory reticulocytosis that subsided when the hematocrit level returned to or exceeded the pre-exposure level.

Increased fragility of erythrocytes was noted twelve months after a single 1285 Mc exposure, at 100 mW/cm^2 for 6 hours (Table III). At twenty-four months after exposure erythrocyte fragility values were comparable to those of normal dogs. There were no changes from that of unexposed animals in erythrocyte fragility immediately following 2800 Mc at 100 mW/cm^2 or 165 mW/cm^2. Clotting and clot retraction time were within the normal range. There was an increase in blood viscosity and hematocrit.

A hematologic effect from microwave exposure was indicated by measurement of red cell survival time and selected phases of iron metabolism. Cr^{51} studies revealed an increased disappearance rate of Cr^{51} tagged erythrocytes after 2800 Mc, 50 mW/cm^2, and 100 mW/cm^2 followed by a gradual return toward the normal rate. The plasma Fe^{59} clearance rate after exposure at 50 mW/cm^2 for 9 hours and 100 mW/cm^2 for 7 hours was not stimulated to the same degree as in dogs subjected to sham irradiation. Hypoferremia was marked and the total iron plasma turnover rate was depressed in comparison with sham exposed animals. There was a significant increase in the total plasma iron binding capacity at 45 days following nine hours of exposure at 50 mW/cm^2; non-significant changes resulted from exposure at 100 mW/cm^2 for seven hours. Dogs exposed to 100 mW/cm^2 for 7 hours had a significant increase in erythrocyte

TABLE IA

Leukocyte Changes After Microwave Exposure

(2800 Mc/sec, 100 mW/cm^2, 6 hours)

Sample	Total Leukocytes	Neutrophils	Lymphocytes	Eosinophils
Initial	13.5 ± 0.8*	9.2 ± 0.9	3.4 ± 0.5	0.6 ± 0.2
Terminal	13.8 ± 1.4	12.0 ± 1.6	1.4 ± 0.3	0.2 ± 0.1
24 hrs.	16.6 ± 2.5	12.1 ± 1.8	3.2 ± 0.7	0.8 ± 0.3
7 days	11.6 ± 0.9	7.5 ± 0.6	3.2 ± 0.5	0.8 ± 0.2
14 days	9.5 ± 1.0	5.8 ± 0.4	2.9 ± 0.6	0.5 ± 0.1
28 days	9.1 ± 0.7	6.1 ± 0.4	2.4 ± 0.4	0.6 ± 0.1
60 days	10.0 ± 0.7	7.8 ± 0.9	2.0 ± 0.4	0.8 ± 0.2

*cells x $10^3/mm^3$, Mean ± Standard Error of the Mean, n=9

TABLE IB

Leukocyte Changes After Microwave Exposure

(1280 Mc/sec, 100 mW/cm^2, 6 hours)

Sample	Total Leukocytes	Neutrophils	Lymphocytes	Eosinophils
Initial	10.6 ± 1.2*	7.4 ± 0.8	2.7 ± 0.2	0.8 ± 0.2
Terminal	14.1 ± 1.4	12.1 ± 1.2	1.3 ± 0.2	0.3 ± 0.1
24 hrs.	14.2 ± 1.6	10.1 ± 1.0	3.3 ± 0.5	1.1 ± 0.3
7 days	11.7 ± 1.4	7.4 ± 0.9	3.8 ± 0.3	0.9 ± 0.2
14 days	11.0 ± 1.3	7.1 ± 0.7	3.6 ± 0.4	1.1 ± 0.3
28 days	9.1 ± 0.9	5.6 ± 0.5	3.3 ± 0.4	0.8 ± 0.1
60 days	10.3 ± 0.5	5.6 ± 0.3	3.3 ± 0.3	0.9 ± 0.2

*cells x $10^3/mm^3$, Mean ± Standard Error of the Mean, n=11

Fig 16

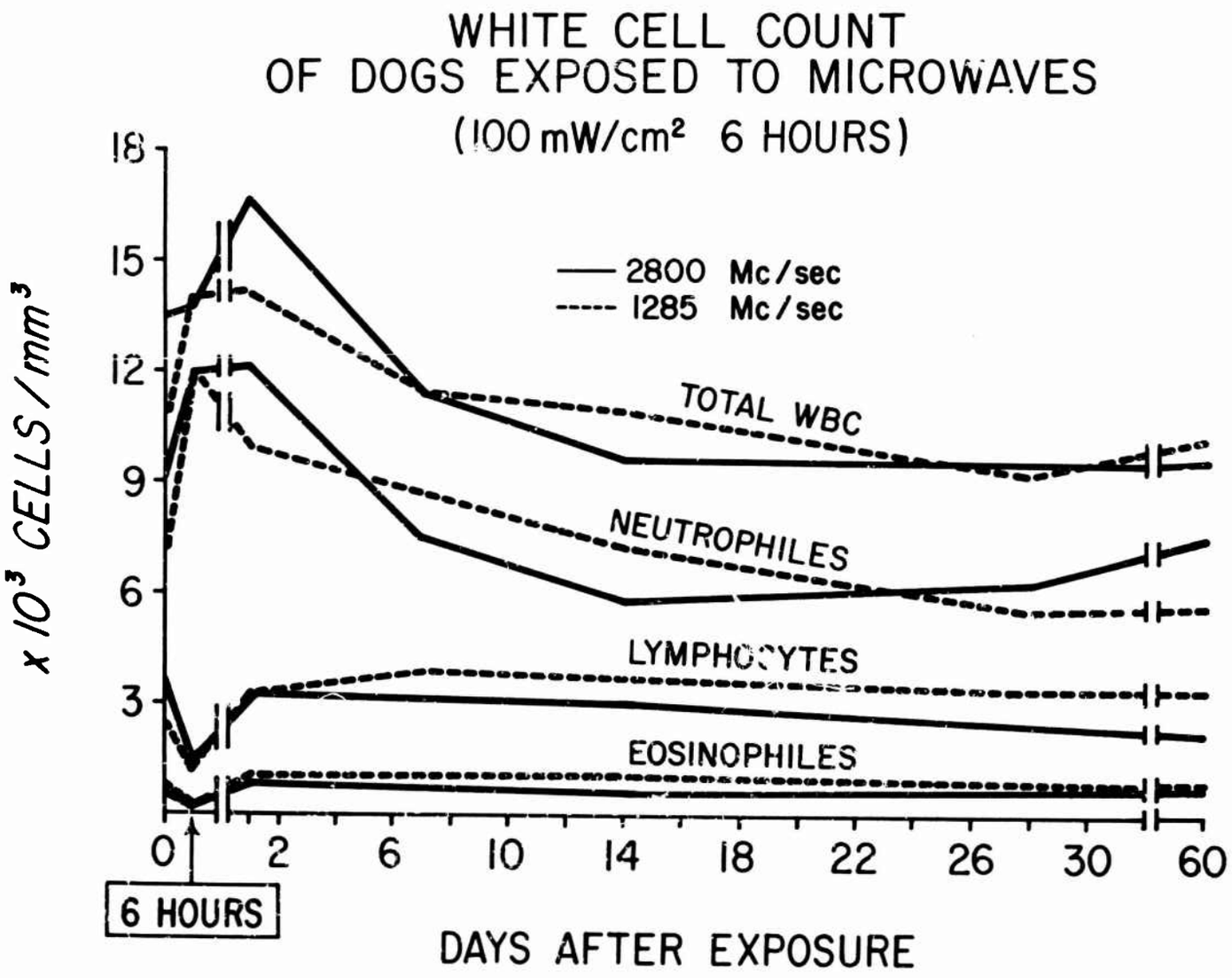

WHITE CELL COUNT
OF DOGS EXPOSED TO MICROWAVES
(100 mW/cm² 6 HOURS)
x 10³ CELLS/mm³
18
15
12
9
6
3
2800 Mc/sec
1285 Mc/sec
TOTAL WBC
NEUTROPHILES
LYMPHOCYTES
EOSINOPHILES
0
2
6
10
14
18
22
26
30
60
6 HOURS
DAYS AFTER EXPOSURE

TABLE II A

Hematologic Response in Dogs Following 1285 Mc/sec Exposure

100 mW/cm^2 for 6 hours

x 10^3 Cells/mm^3

No. of Dogs	Determination (months post)	Total Leukocytes	Neutrophils	Lymphocytes	Eosinophils	Hematocrit	Reticulocytes (%)
11	0	10.6 ± 1.2*	7.4 ± 0.8	2.7 ± 0.2	0.8 ± 0.2	51.0 ± 1.5	0.8 ± 0.2
10	1	9.1 ± 1.0	5.6 ± 0.5	3.3 ± 0.3	0.8 ± 0.1	50.5 ± 1.0	0.4 ± 0.1
9	2	10.3 ± 0.5	5.6 ± 0.3	3.3 ± 0.3	0.9 ± 0.2	49.7 ± 1.0	0.3 ± 0.1
9	3	9.5 ± 0.5	5.0 ± 0.3	3.1 ± 0.3	0.9 ± 0.3	53.2 ± 1.4	0.3 ± 0.1
9	4	10.1 ± 0.6	5.3 ± 0.4	3.5 ± 0.4	0.7 ± 0.1	51.4 ± 1.6	0.4 ± 0.3
9	5	10.8 ± 0.9	6.0 ± 0.6	3.3 ± 0.2	0.9 ± 0.2	53.9 ± 1.9	0.6 ± 0.2
9	6	10.7 ± 0.8	6.2 ± 0.5	3.1 ± 0.3	1.0 ± 0.2	53.1 ± 1.1	0.6 ± 0.1
9	7	10.9 ± 0.8	5.9 ± 0.4	3.6 ± 0.3	0.9 ± 0.2	50.9 ± 1.6	0.2 ± 0.1
9	8	11.0 ± 0.7	5.9 ± 0.4	3.8 ± 0.3	0.8 ± 0.2	51.9 ± 0.9	0.3 ± 0.1
9	9	10.6 ± 1.0	5.7 ± 0.5	3.4 ± 0.4	1.2 ± 0.3	52.9 ± 1.8	0.2 ± 0.0
9	10	8.9 ± 0.3	4.9 ± 0.3	3.3 ± 0.3	0.5 ± 0.1	52.8 ± 1.9	0.2 ± 0.1
9	11	10.9 ± 0.9	6.4 ± 0.9	3.4 ± 0.4	0.9 ± 0.2	53.3 ± 1.4	0.2 ± 0.0
9	12	11.2 ± 0.6	6.9 ± 0.5	3.1 ± 0.3	0.7 ± 0.1	54.2 ± 1.4	0.3 ± 0.1

* Mean ± Standard Error of the Mean

TABLE II B

Hematologic Response in Dogs Following 1285 Mc/sec Exposure

100 mW/cm^2 for 6 hours

$\times 10^3$ Cells/mm^3

No. of Dogs	Determination (months post)	Total Leukocytes	Neutrophils	Lymphocytes	Eosinophils	Hematocrit	Reticulocytes (%)
9	19	9.4 ± 0.8*	5.4 ± 0.5	2.7 ± 0.2	0.7 ± 0.2	53.1 ± 1.6	0.3 ± 0.1
9	20	10.6 ± 1.1	5.7 ± 0.5	3.4 ± 0.3	1.0 ± 0.4	50.4 ± 1.6	0.4 ± 0.1
9	21	8.9 ± 0.8	5.1 ± 0.6	2.8 ± 0.3	0.7 ± 0.2	52.6 ± 1.5	0.3 ± 0.1
9	22	10.2 ± 0.8	6.2 ± 0.6	2.9 ± 0.3	0.7 ± 0.2	52.8 ± 1.7	0.6 ± 0.2
8	23	9.1 ± 1.0	5.3 ± 0.8	2.8 ± 0.4	0.6 ± 0.1	50.6 ± 2.0	0.4 ± 0.1
8	24	10.0 ± 1.3	5.8 ± 0.9	3.2 ± 0.4	0.6 ± 0.1	52.4 ± 1.9	0.5 ± 0.1

* Mean ± Standard Error of the Mean

Fig 17

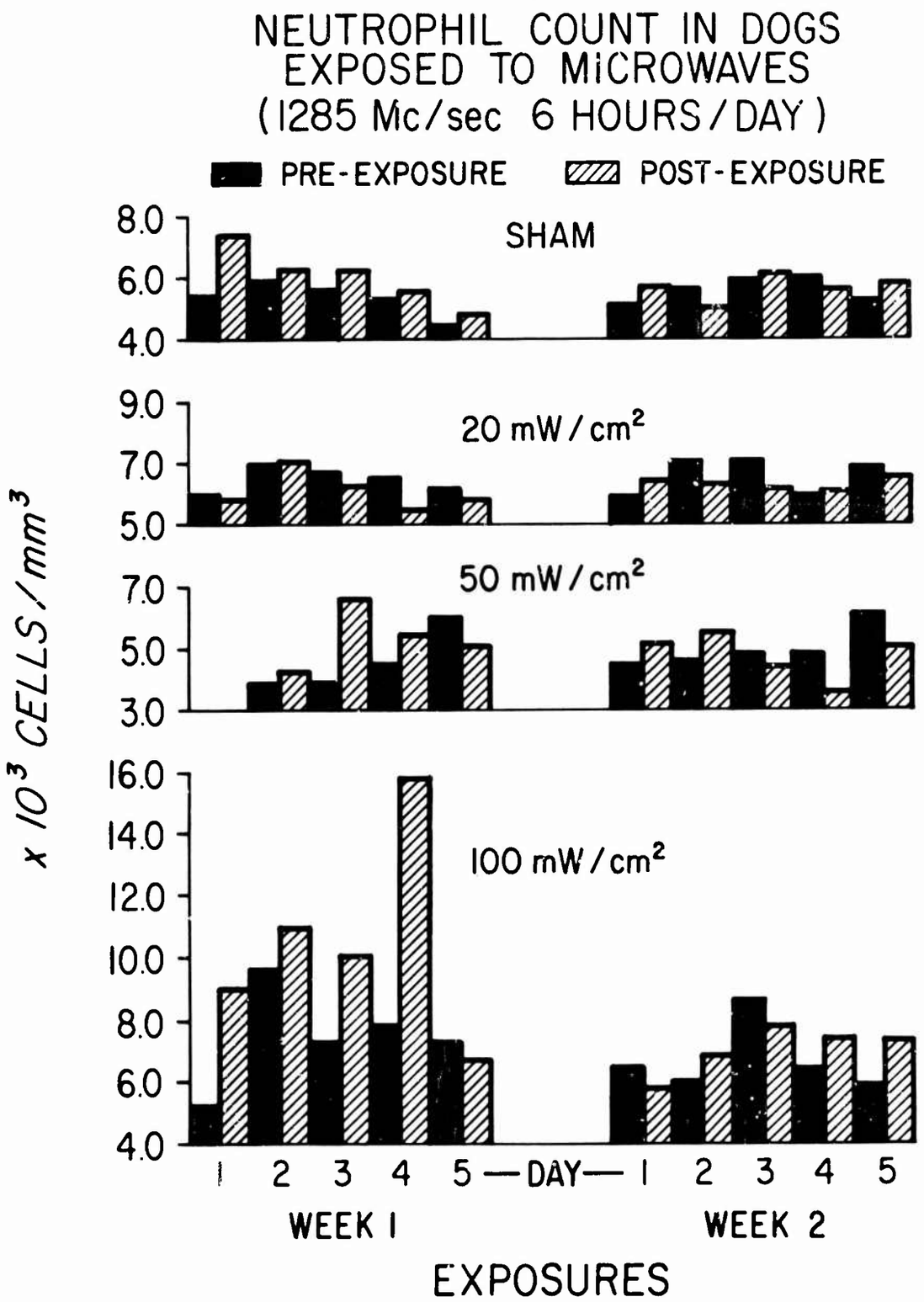

NEUTROPHIL COUNT IN DOGS
EXPOSED TO MICROWAVES
(1285 Mc/sec 6 HOURS/DAY)
PRE-EXPOSURE
POST-EXPOSURE
SHAM
20 mW/cm²
50 mW/cm²
100 mW/cm²
x 10³ CELLS/mm³
8.0
6.0
4.0
9.0
7.0
5.0
7.0
5.0
3.0
16.0
14.0
12.0
10.0
8.0
6.0
4.0
1 2 3 4 5 —DAY— 1 2 3 4 5
WEEK 1
WEEK 2
EXPOSURES

Fig 18

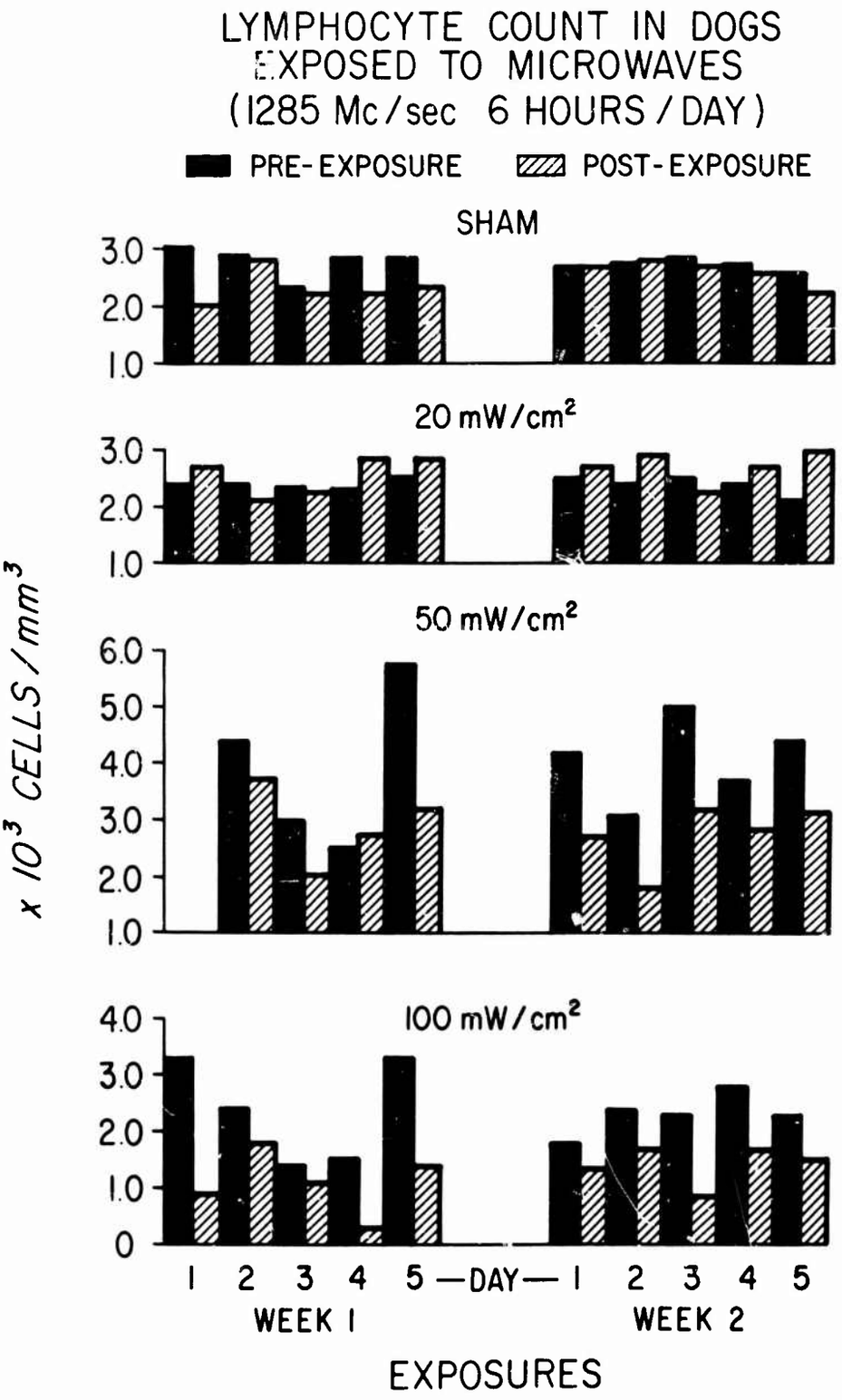

LYMPHOCYTE COUNT IN DOGS
EXPOSED TO MICROWAVES
(1285 Mc/sec 6 HOURS / DAY)
PRE-EXPOSURE
POST-EXPOSURE
SHAM
20 mW/cm²
50 mW/cm²
100 mW/cm²
x 10³ CELLS / mm³
3.0
2.0
1.0
6.0
5.0
4.0
0
1 2 3 4 5 —DAY— 1 2 3 4 5
WEEK 1
WEEK 2
EXPOSURES

Fig 19

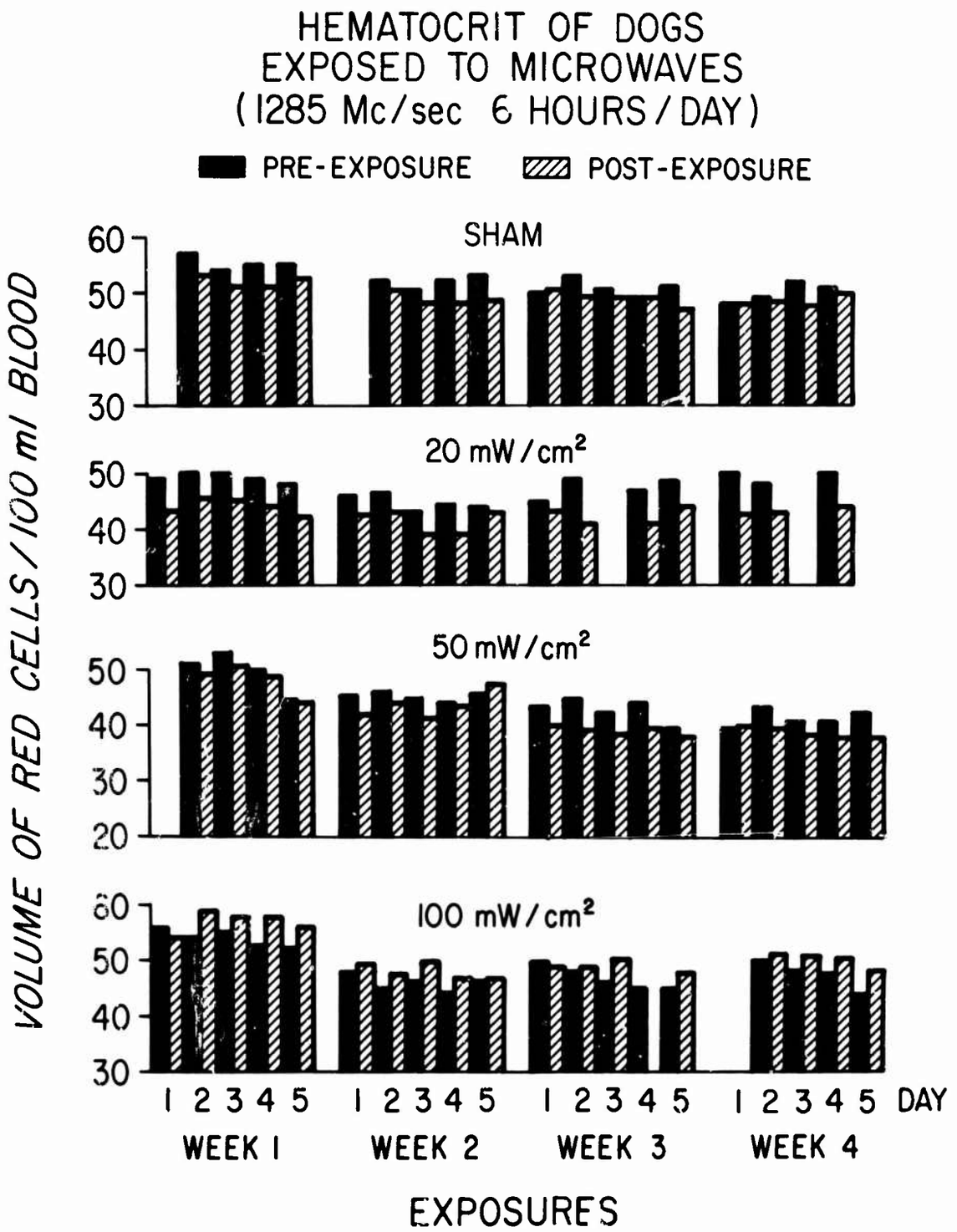

HEMATOCRIT OF DOGS
EXPOSED TO MICROWAVES
(1285 Mc/sec 6 HOURS/DAY)
PRE-EXPOSURE
POST-EXPOSURE
SHAM
20 mW/cm²
50 mW/cm²
100 mW/cm²
VOLUME OF RED CELLS/100 ml BLOOD
60
50
40
30
20
1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 1 2 3 4 5 DAY
WEEK 1
WEEK 2
WEEK 3
WEEK 4
EXPOSURES

Fig 20

RETICULOCYTE VALUES IN DOGS
PRIOR TO MICROWAVE EXPOSURE
(1285 Mc/sec 6 HOURS/DAY)

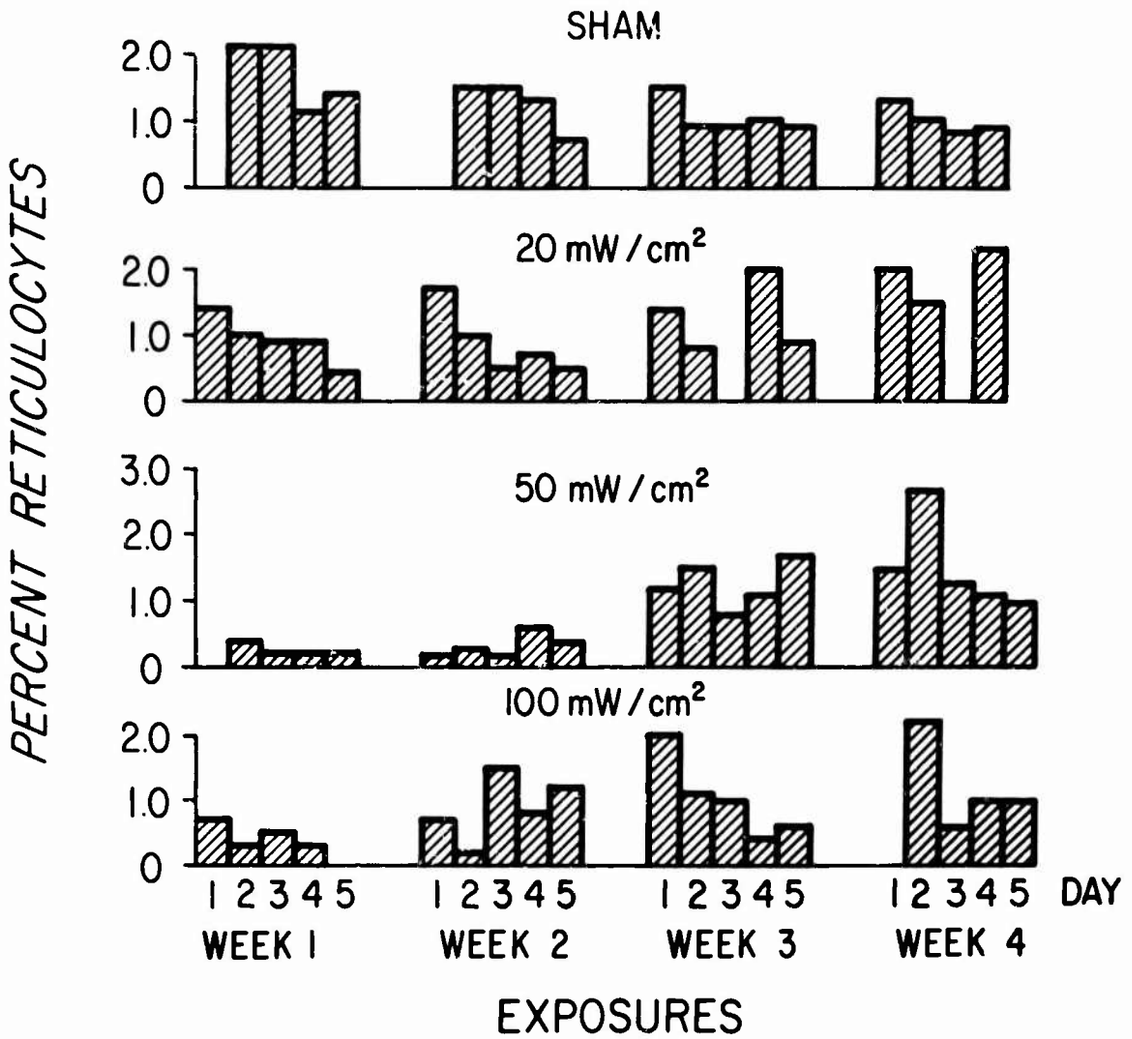


TABLE III

Erythrocyte Fragility Test Values for Normal and Microwave Treated Animals

No. of Dogs	Group	Hemolysis (Percent Salt Solution) Initial	Complete
22	Normal	0.46 ± 0.007 [a]	0.35 ± 0.005
	" 12 months later	0.49 ± 0.012	0.32 ± 0.008
9	Microwave Treated [b]	0.51 ± 0.012	0.37 ± 0.019
	" " 12 months later [c]	0.49 ± 0.012	0.32 ± 0.008

[a]-Mean ± 2 Standard Errors of the Mean

b-1285 Mc/sec, 100 mW/cm^2 for 6 hrs., 10 to 12 months previous to fragility study

c- " " " 22 to 24 months " " " "

Fe^{59} uptake that progressed to a maximum at forty-five days after exposure. Following 50 mW/cm^2 exposure the erythrocyte Fe^{59} uptake remained relatively depressed. At three months after exposure the Fe^{59} uptake had returned to the normal value.

In dogs given sham or 1285 Mc, 20 mW/cm^2 exposures daily for two to four weeks, no significant difference in the time taken for Cr^{51} activity of erythrocytes (tagged in vivo) to decline to 50 percent of the initial value, or in the apparent Cr^{51} elution rate was noted. Seventeen days after 1240 Mc, 50 mW/cm^2, 6 hours daily, totaling 84 and 114 hours, incorporation of Fe^{59} in erythrocytes was more rapid and the maximum incorporation was higher than was noted prior to microwave treatment. The initial plasma clearance of Fe^{59} in two dogs, ten days and eighteen days after 60 hours and 138 hours of microwave exposure (6 hours per day) appeared prolonged when compared with data for normal dogs. Maximum incorporation of Fe^{59} occurred earlier in microwave treated dogs, and the maximum degree of Fe^{59} incorporation in the erythrocytes was in the upper limits of the range for normal animals.

Plasma total proteins were decreased and plasma chlorides increased in dogs exposed daily for six hours at 50 mW/cm^2, 1240 Mc (Table IV). Immediately following each exposure there was an increase in plasma chlorides; venous CO_2 content was mildly decreased. When exposure was extended to five weeks, change in pre- and post-exposure venous CO_2 content values were essentially negligible, and the values were comparable to those noted prior to the first microwave exposure.

Microwave exposure did not cause any significant changes in blood glucose, NPN, plasma BUN or calcium levels.

D. Hemodynamics

Hemoconcentration resulted when dogs were exposed to 2800 Mc pulsed microwaves at 165 mW/cm^2 for three hours. The hematocrit approximated the pre-exposure level at 24 hours after exposure. Hematocrit values were slightly increased or decreased from the pre-exposure level after two hours at 165 mW/cm^2, or six hours at 100 mW/cm^2. After five hours of 200 Mc CW, at 165 mW/cm^2 an increased hematocrit was noted. Increase in hematocrit resulted following repeated 1250 Mc pulsed, at 100 mW/cm^2 for six hours daily. As the exposures were repeated the pre-exposure hematocrit value gradually decreased from the initial value for the first exposure of each week (Fig. 19). Following 50 mW/cm^2 the hematocrit decreased after each daily exposure. The pre-exposure levels were lower than the initial value after the first week of daily exposures. After 20 mW/cm^2 there was a decrease in hematocrit, more marked than that noted after 50 mW/cm^2, but comparable to the response seen among the sham exposed dogs. One month after exposure there was an increase in hematocrit which was still evident at 6 months.

Normal dogs exposed at 165 mW/cm^2, 2800 Mc, had a 2 percent per hour body-weight loss (Fig. 21). At 100 mW/cm^2, the body-weight loss was 1.1 percent per hour. The weight loss was linear after the first 30 minutes of exposure. The relationship of percent body-weight loss during exposure to the

TABLE IV

Biochemical Changes Following 1240 Mc/sec Exposure

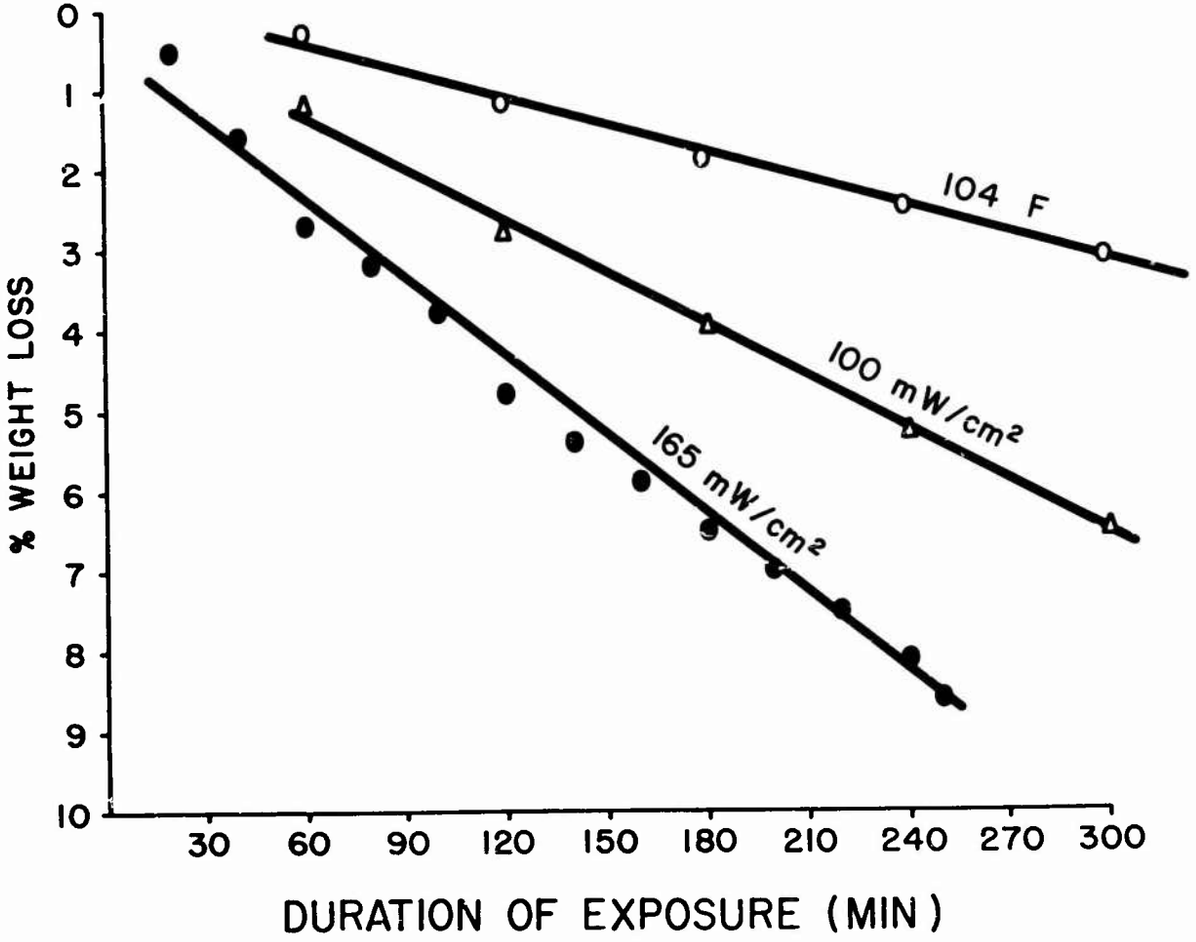
(50 mW/cm^2, 6 hrs, daily)

Determination	No. of dogs	Exposure No.	Pre-Value	Post-Value
Total Proteins (gm/100 ml)	4	1	6.90 ± 0.14*	6.95 ± 0.12
		2	6.65 ± 0.19	6.60 ± 0.24
		3	6.53 ± 0.15	6.60 ± 0.14
		4	6.50 ± 0.14	6.50 ± 0.14
	3	5	6.43 ± 0.09	6.30 ± 0.15
Plasma Chloride (mEq/L)	6	1	109.6 ± 1.9	121.3 ± 2.4
		2	110.7 ± 2.8	118.8 ± 2.0
		3	110.7 ± 2.6	117.6 ± 1.7
		4	110.3 ± 2.5	119.2 ± 2.9
		5	111.5 ± 2.9	116.2 ± 2.8

* Mean ± Standard Error of the Mean

Fig 21

EFFECT OF 2800 Mc/sec MICROWAVES
OR INCREASED ROOM TEMPERATURE
ON BODY WEIGHT OF DOGS



thermal burden imposed by microwaves indicates a direct correlation between power density and weight loss. Dogs maintained in an 103.5-106° F environment for comparable periods had a 0.6 percent body-weight loss per hour and variable hematocrit change.

The interrelationship of hematocrit change, rectal temperature increase, and weight loss between the normal dog and IR survivor is illustrated in Fig. 22. Rectal temperature in IR dogs increased more rapidly than in normal dogs although the period of thermal equilibrium was comparable. Weight loss was constant and linear. The normal dog had a biphasic hematocrit response.

A 5 to 6 percent reduction in hematocrit was evident in dogs immediately after microwave exposure administered four days after 400 R Co^{60} irradiation (Table V). The decrease in hematocrit was more marked one week after microwave exposure. The greater hematocrit change in dogs exposed to microwaves four days after Co^{60} irradiation suggests a vascular effect from ionizing radiation, or a microwave effect that amplifies ionizing radiation injury to the hematopoietic system.

To determine the extent to which compensatory fluid adjustments occur during microwave exposure with particular attention to abnormal or pathological sequelae, dogs that had been exposed to large doses of ionizing radiation several months to years previously, were studied. Comparison was made with normal dogs of comparable age. The response after water intake, starting at the time of critical temperature increase, is shown in Fig. 23. Immediately following ingestion of water, the rectal temperature rapidly decreased toward the pre-exposure level. The lowering of rectal temperature facilitated prolongation of the exposure. These animals did not develop a critical temperature in the six-hour experimental period, yet most of them exposed for such a prolonged period at 165 mW/cm^2 developed burns over the rib cage, which were more extensive and frequent than in the animals not given water. In contrast, ingestion of water by IR dogs, did not permit a prolongation of permissible exposure.

E. Heart

Electrocardiograph tracings were obtained on anesthetized dogs exposed at 165 mW/cm^2, 2800 Mc pulsed. The findings on one dog that were representative of the general response were interpreted by Thomas B. Watt, Jr., M.D. and are summarized in Table VI. EKG tracings are illustrated in Figure 24. There was a strong suggestion of an ischemic myocardial change during the latter part of the 30 min. exposure.

F. Central Nervous System

There was impaired locomotion in animals during or after microwave exposure indicating an effect on the central nervous system.

Head irradiation of an anesthetized dog with 2800 Mc, 165 mW/cm^2, resulted in a more rapid and greater rectal temperature increase than when an equivalent body surface area on the rib cage was exposed (Fig. 25,26). The

Fig 22

PHYSIOLOGIC RESPONSE IN DOGS
EXPOSED TO MICROWAVES
2800 Mc/sec 165 mW/cm^2

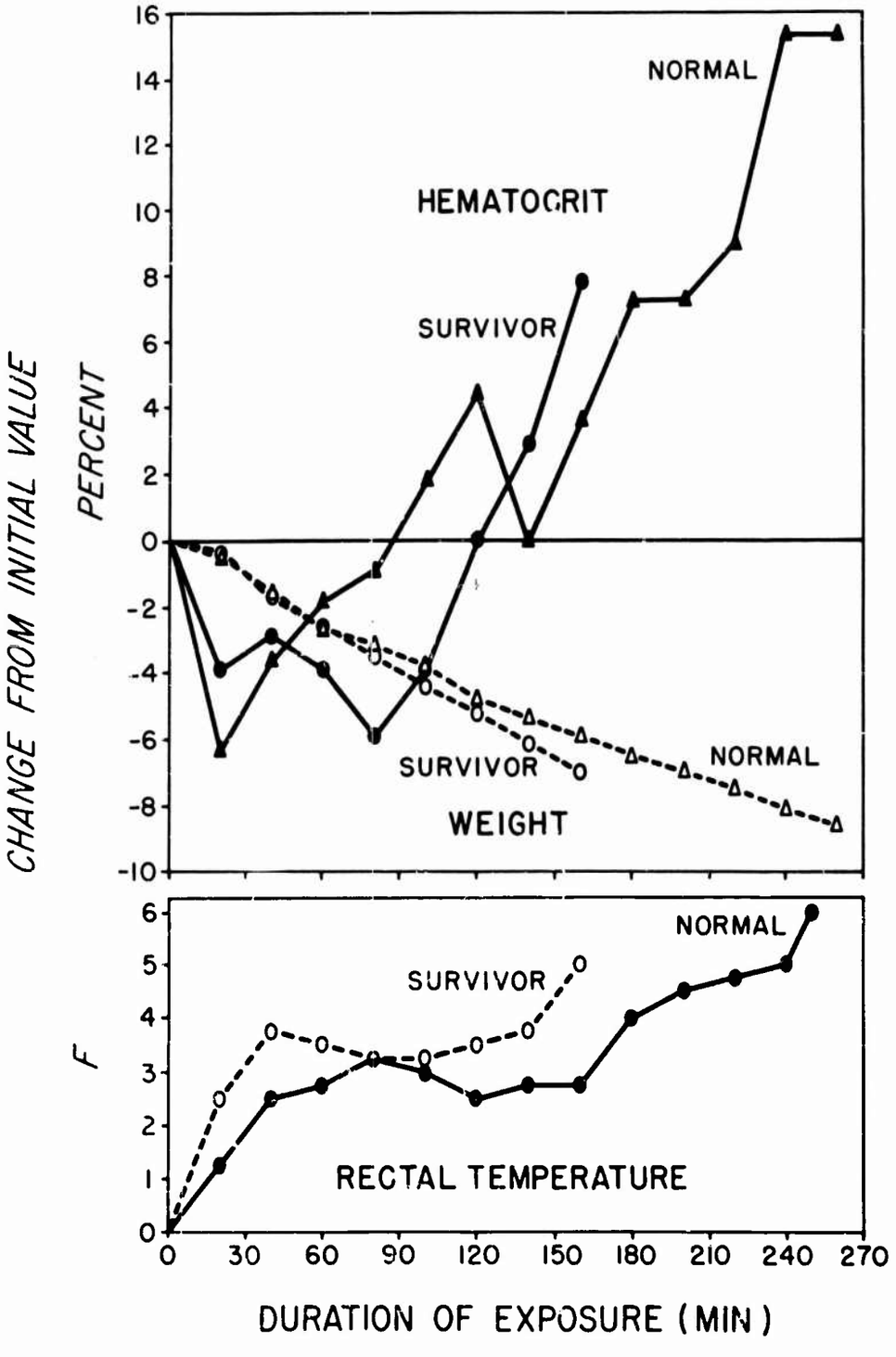


TABLE V

Effect of 2800 Mc/sec on Temperature and Hematocrit in Dogs

Immediately Post-Exposure					
Power Level	100 mW/cm^2		165 mW/cm^2		Control
Duration of Exposure	6 Hours		2 Hours		
History	Normal (10)	Co-60 400 R (5)	Normal (10)	Co-60 400 R (6)	Co-60 400 R (6)
Temp. change ^{o}F	+ 3.0	+ 1.4	+ 3.4	+ 3.8	
Hct. % change	+ 3.0	- 5.0	+ 3.1	- 6.0	
1 Week Post Exposure					
Hct. % change	- 3.1	- 19.7	- 4.5	- 21.0	- 10.1

( ) Number of Dogs

Fig 23

EFFECT OF HYDRATION ON RECTAL TEMPERATURE
IN DOGS EXPOSED TO MICROWAVES
2800 Mc/sec 165 mW/cm²

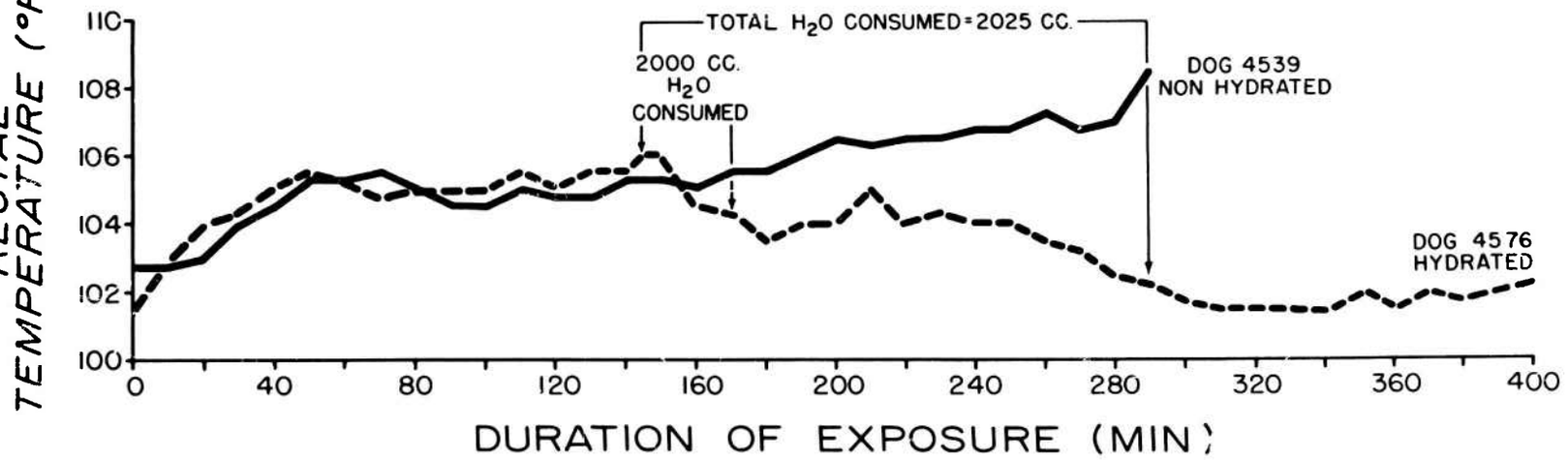


TABLE VI

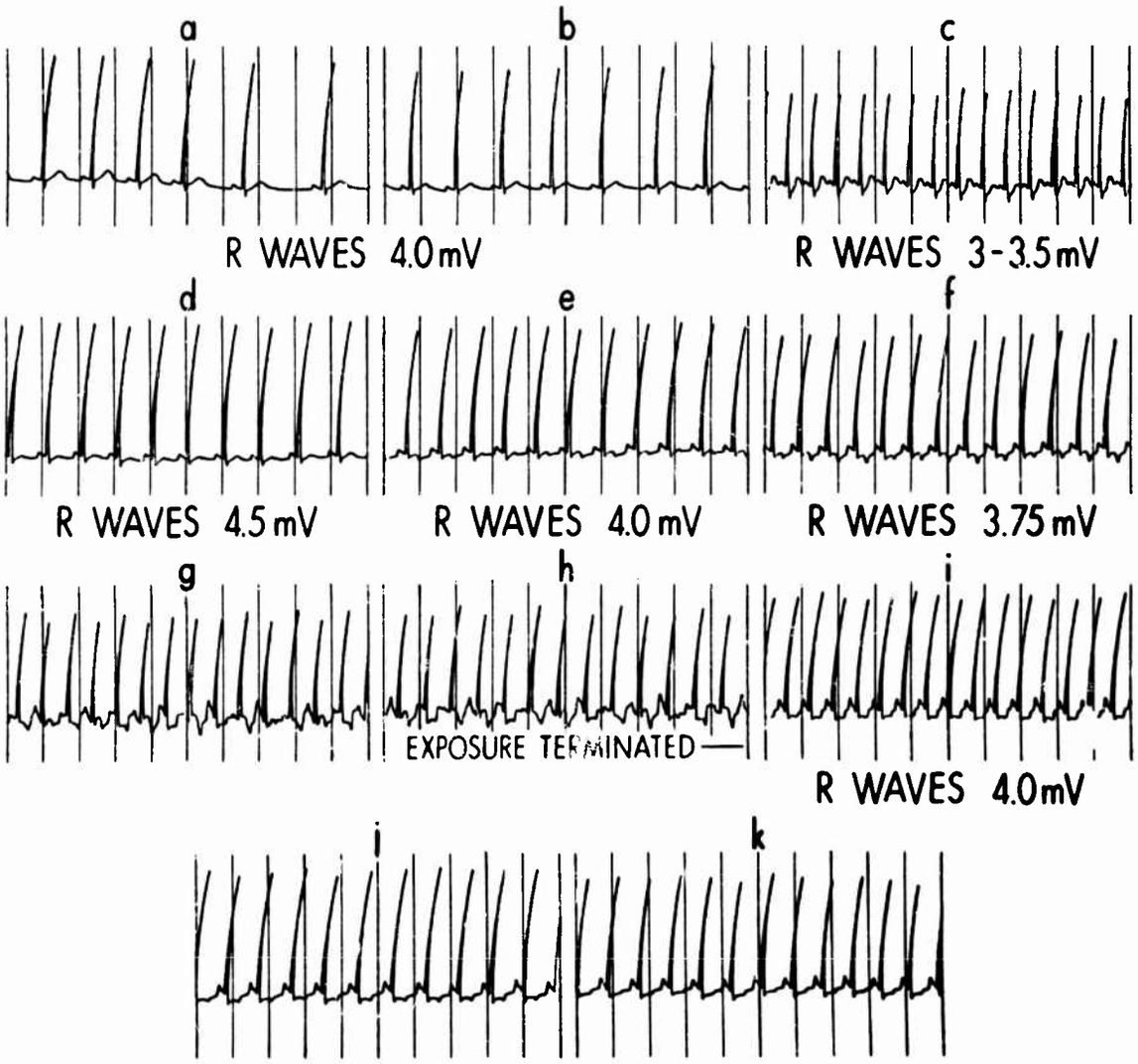
Heart Rate and Temperature Response in Dogs to 2800 Mc/sec Pulsed Microwaves-165 mW/cm^2

Time after onset of exposure (minutes)	Rectal Temp. (^oF)	Heart rate (beats/min.)	Comment	EKG figure 24
Control	100.5	107	Some sinus arrhythmia	a b
1-5	100.5	225	Depression of takeoff point of ST segment; slightly taller T wave	c
10	101.5	148	Accomodation	d
15	102.8	167	Inverted T wave	e
20	104.0	193		f
25	105.2	210		g
30*	106.2			h
60	105.2	232		i
85	102.8	168		j
110	102.2	175		k

* power off

Fig 24

EKG OF DOGS EXPOSED TO MICROWAVES
2800 Mc/sec 165 mW/cm^2



*a-k refer to Table VI

Fig 25

TEMPERATURE RESPONSE IN DOGS FOLLOWING MICROWAVE EXPOSURE TO THE HEAD
(2800 Mc/sec 165 mW/cm^2)

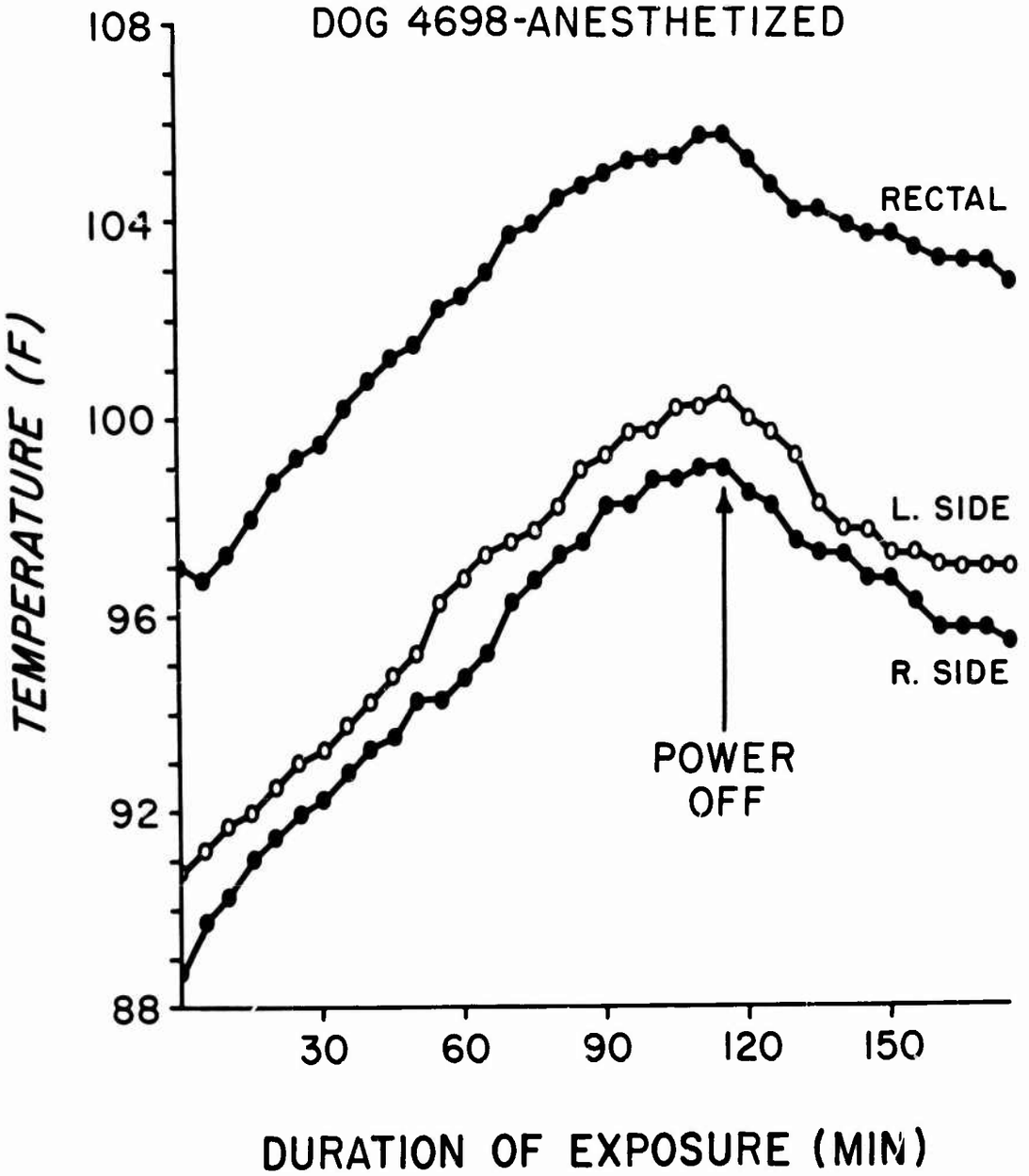
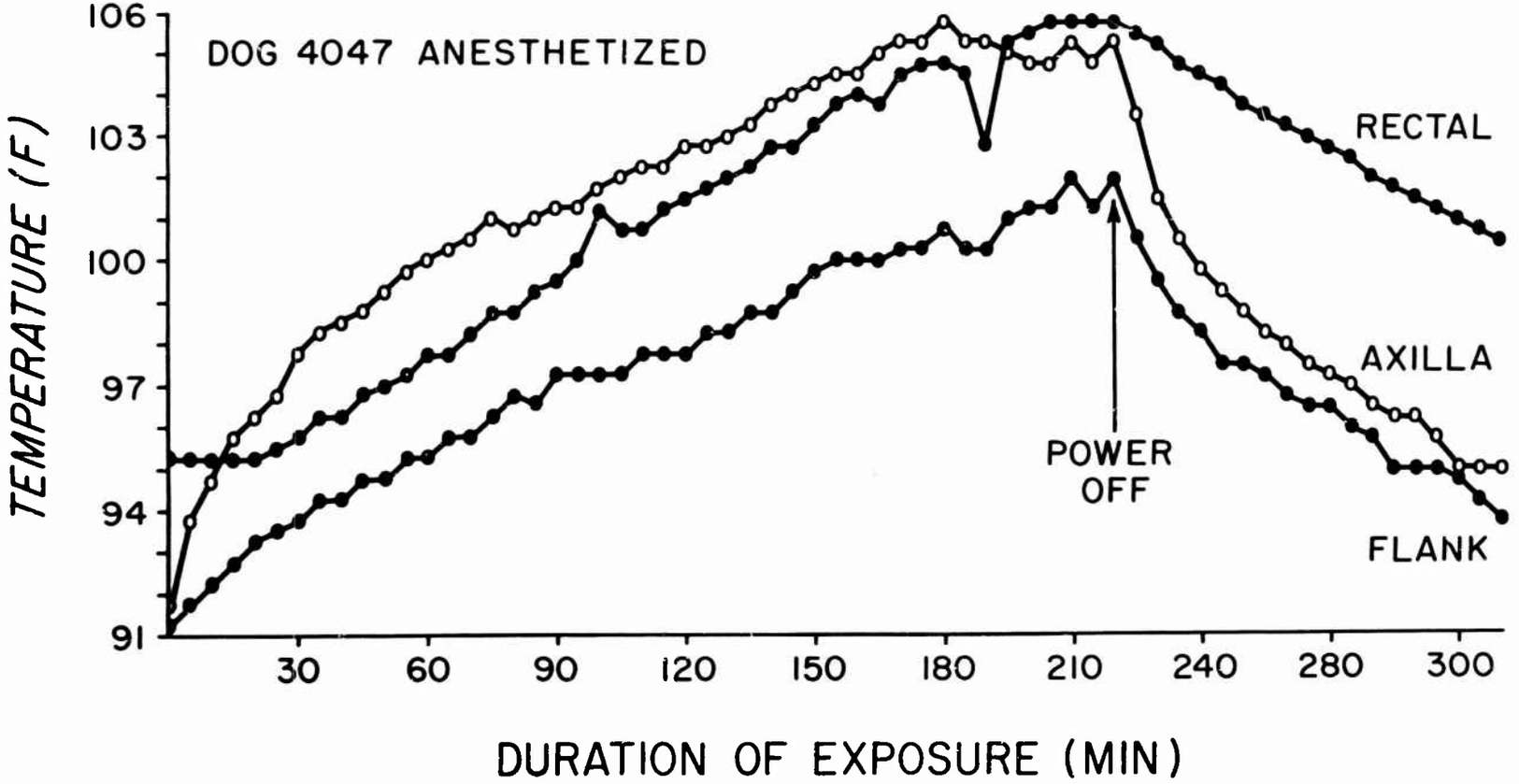


Fig 26

TEMPERATURE RESPONSE IN DOGS
FROM PARTIAL-BODY EXPOSURE TO MICROWAVES
(2800 Mc/sec 165 mW/cm^2)



difference in rectal temperature that resulted from these two partial body exposures may indicate altered brain function in the control of thermal regulation when the head is subjected to microwaves.

Neurologic manifestations (loss of equilibrium, opisthotonus, nystagmus and salivation), that were observed following 25,000 R X-irradiation to the head, were less severe in dogs with a history of microwave exposure than in dogs not exposed to microwaves. There was diminution in the acute X-irradiation effects in the microwave treated animals prior to death, whereas in the other dogs it was unchanged. Dogs had persistent vomiting up to five hours following 10,000 R X-irradiation to the head, while similarly X-irradiated microwave pre-treated dogs did not vomit. Emetic response in other studies indicates a microwave effect on the central nervous system. A significant difference in vomiting time at the 5% level, following intravenous apomorphine administration, between normal and microwave treated animals was noted. Mean vomiting time was 98 seconds in normal dogs and 197 seconds in the microwave treated animals. Retching and vomiting were noted in dogs that survived whole-body and upper-body X-irradiation when they were subjected to 1240 Mc microwaves, but were absent during exposure at 2800 Mc. Emesis never occurred in normal dogs during microwave exposure. Vomiting which occurred in exposures at 1240 Mc but not at 2800 Mc may be related to the greater tissue depth through which the 1240 Mc energy is absorbed, added to a residual biologic change in dogs from the previous X-irradiation.

Differing responses were seen in mice during acute exposure at 1240 Mc and 2800 Mc. Mice, while subjected to 1240 Mc were less active than those exposed at 2800 Mc. When the lethal limit at 1240 Mc was approached the mice convulsed for less than 30 seconds before death ensued. At 2800 Mc a more violent and intermittent convulsive pattern (clonic-tonic seizures) of about one minute duration preceded death.

Hyperpyrexia developed when one side of a rabbit's head was exposed to 2800 Mc CW, while the rest of the body was shielded. At 180 mW/cm^2 or higher, the pupil was dilated in the exposed eye and constricted in the non-exposed eye. At field intensities less than 180 mW/cm^2 the pupil was constricted in the exposed eye, and normal in the unexposed eye. No effects were observed following exposures at 54 mW/cm^2 or less.

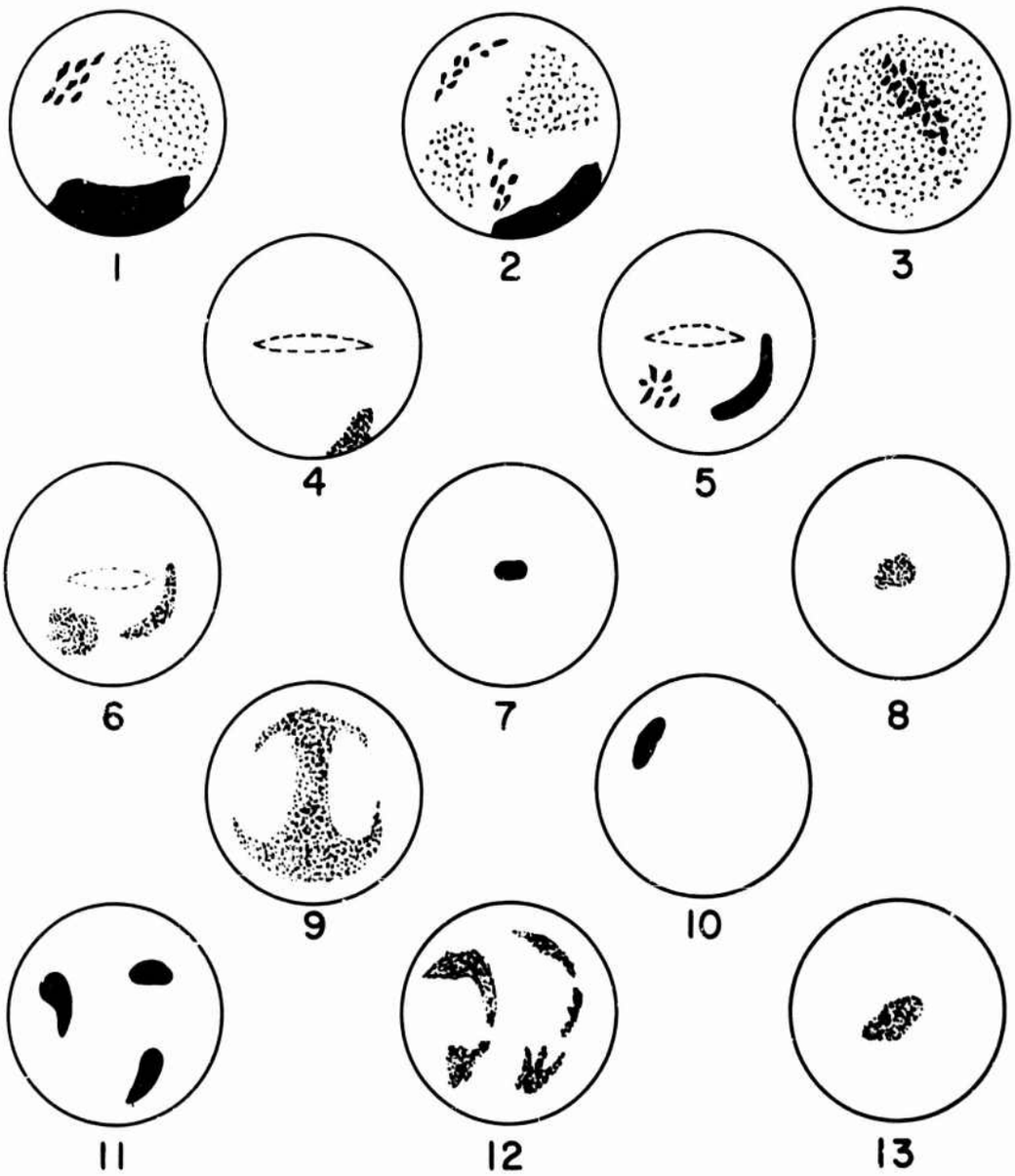
G. The Eye

The sensitivity of the eye to microwaves was studied by exposing rabbits and dogs to CW or pulsed 2800 Mc.

Representative lens changes in rabbits are shown in Figure 27. Exposure at 220-240 mW/cm^2 for 30 min. produced some conjunctival congestion and miosis which subsided the next day. No lenticular changes were noticed over a period of 5 months. Exposure for 45 min. at the same flux density caused a marked conjunctival reaction and purulent discharge from the eye. The animal salivated considerably during exposure and was very restless. Ten days after exposure the lens showed a nonhomogeneous dark opacity in the upper quadrant of the posterior cortex (Fig. 27-1). This progressed into a denser and more

Fig 27

LENS CHANGES IN RABBITS AFTER EXPOSURE TO MICROWAVES (2800 Mc/sec CW)



diffuse opacity (Fig. 27-2), which gradually became white. At 20 days after exposure, the lens was completely opaque (Fig. 27-3). This remained unchanged for 2 1/2 months, after which the opacity started to diminish and the retinal light reflex could be seen at the periphery. The change continued over the next 2 months.

Three animals exposed at 220-240 mW/cm^2 for 1 hr. were very agitated and sallvated profusely during exposure. Their eyes showed marked conjunctival injection and edema after exposure. This subsided after 3-4 days. These animals developed suborbital skin burns. In one animal the lens appeared hazy within 24 hours and subsequently became white. No further change was noted over the next 1 1/2 months, at which time the animal died. Another rabbit had prominent suture lines of the lens and a small dark opacity 12 days after exposure (Fig. 27-4). The dark spot progressed into a denser, crescent-shaped area by 36 days post-irradiation (Fig. 27-5). In addition, some vacuoles appeared in the lower segment for a few days and then regressed. The dark area and suture line started fading at 45 days (Fig. 27-6) and by the fifty-sixth day, the lens was completely clear. A week later a small dark spot appeared in the center of the lens (Fig. 27-7), which progressed into an irregular but well-defined opacity by the seventy-fifth day after exposure (Fig. 27-8). This lesion began to regress in size and density and did not disappear until 165 days after the irradiation. The third animal showed a small, dark lenticular opacity in the lower segment of the lens 2 days after exposure, which developed into an anchor-shaped opacity at 15 days (Fig. 27-9). This remained unchanged until the animal died 45 days after exposure.

A rabbit exposed at 220-240 mW/cm^2, 10 min. daily for 3 days showed marked agitation and salivation during exposure. There was a marked effect on the eye; 2 days after the last exposure a severe uveitis developed and a complete pearly opacity of the lens resulted. The eyeball decreased in size, with the lenticular changes unaltered throughout the observation period of 6 months.

In one rabbit, a single 30-min. exposure at 160-170 mW/cm^2 produced only mild conjunctival reaction which subsided by the following day. A tiny dark speck that appeared in the posterior cortex of the lens remained unchanged for 5 months (Fig. 27-10). Exposure of another animal at the same flux-density for 45 min. did not produce any lenticular changes.

Six rabbits exposed at 160-170 mW/cm^2 for 60 min. showed moderate conjunctival reaction. Four had lenticular changes; in three of these diffuse nonhomogeneous dark opacities appeared in the posterior cortex 1-2 days after exposure. This change was transitory and disappeared in four days. At 5-7 days after exposure, well defined small opacities appeared (Fig. 27-11). These completely regressed in one animal, remained stable in the second, and varied in size and opaqueness over a 4 month period in the third rabbit (Fig. 27-12). The fourth animal developed a small central lenticular opacity at 20 days that was unchanged for 3 months (Fig. 27-13).

Four rabbits were exposed 30 min. daily at 160-170 mW/cm^2 for 5 days. One rabbit developed a diffuse nonhomogeneous darkening of the lens on the

third day of exposure. This opacity disappeared by the following day. No other lenticular changes were observed over a period of 3 months.

Dr. Sidney Lerman, of Strong Memorial Hospital, studied biochemical changes in the lens of rabbits' eyes after 30 to 45 minutes exposure at 175 mW/cm^2, 2800 Mc CW. The following is an excerpt from Dr. Lerman's report:

"Protein metabolism was investigated by means of isotopically labeled amino acid incorporation and turnover studies in the normal and irradiated lens. These experiments were performed both in vitro and in vivo. Nucleic acid metabolism was also investigated by means of isotope studies and physicochemical determinations on the individual RNA fractions. In vitro experiments with posterior capsules obtained from irradiated and control lenses were utilized to determine the effects of microwave irradiation on the permeability of the posterior capsule. Labeled sulfate was also employed in order to study mucopolysaccharide metabolism in the lens capsule. The results of these experiments indicated that the only discernible alteration in lenses exposed to microwave irradiation occurred in the posterior lens capsule. A significant increase in the permeability of the capsule was shown, as well as an impairment of labeled sulfate turnover. The extensive studies on protein and nucleic acid metabolism failed to reveal any significant differences between the experimental and control lenses. The results of these experiments indicate that the initial effect of microwave irradiation is mainly directed towards the lens capsule resulting in a marked alteration in the permeability of that structure and impairment in metabolism. These effects are probably of a thermal nature."

In collaboration with Dr. Milton Zaret of New York University, the eyes of dogs were selectively exposed to 2800 Mc pulsed at 350 mW/cm^2 or 700 mW/cm^2. Exposures were for 10, 20 or 40 minutes, or for a total of 20 minutes with two or three brief periods of interruption. Periodic examination over an eighteen month period following exposure revealed varying degrees of cataractous changes in some of the microwave irradiated eyes. Most of the changes occurred during the twelve month period following exposure. Lens changes were more marked and always resulted from 20 minutes of exposure at 700 mW/cm^2. The general finding was initially, tumescence of the lens, keratoconjunctivitis, iridocyclitis, miosis, and haziness of the lens. Later, as slowly developing changes occurred, increased opacity, broadening and arborization of the posterior suture marking was observed.

H. The Thyroid

Changes in thyroid I-131 uptake were noted in dogs following microwave exposure (Table VII, VIII). Between 881 and 985 days after a single 1280 Mc exposure at 100 mW/cm^2 for six hours, thyroid uptake of I-131 was greater in dogs subjected to microwaves than in normal dogs. The difference in uptake at 72 hours between microwave exposed and normal dogs was significant at the 5 percent level. The microwave effect on thyroid response may be temporary (Table IX). Repeated thyroid I-131 uptake studies reveal a return in I-131 uptake values to normal levels. After daily microwave exposure of one to five weeks duration at 20 or 50 mW/cm^2 some dogs had an increase in I-131 uptake.

TABLE VII

Thyroid Uptake of I-131 in Dogs Following 1280 Mc/sec Microwaves

(100 mW/cm^2, 6 hour exposure)

Dog No.	Time (days) after exposure	I-131 Uptake(%) at 72 hours
5387	881	31.6
5152	896	34.3
5428	900	41.4
5401	928	44.4
5405	929	42.3
5347	942	47.0
5262	952	37.0
5057	985	30.1

TABLE VIII

Thyroid Uptake of I-131 by Dogs 881-985 Days After Microwave Exposure

Time after I-131 Injection (Hours)	Normal Dogs (8)	1280 Mc/sec 100 mW/cm^2 Single Exp. (8)
24	24.9±2.3 *	30.0±1.5
48	29.0±3.2	35.9±2.5
72	30.3±2.5	38.5±2.2

*percent uptake Mean ± Standard Error of the Mean; ( ) number of dogs

TABLE IX

Thyroid Uptake of I-131 by Dogs Exposed to Microwaves

Normal					Microwave Exposure				
Dog number	Days between tests	%Uptake (hours) 24	48	72	Dog number	Days after exposure	%Uptake (hours) 24	48	72	1280 Mc/sec
V-9	Initial	25.0	31.2	33.0	5385	724	33.6	43.6	46.6	20
	56	25.3	31.9	33.8		920	26.1	31.9	35.6	mW/cm^2
V-11	Initial	23.7	26.7	25.6	5405	929	32.5	40.8	42.3	100
	112	25.2	24.6	25.2		986	23.3	34.1	35.5	mW/cm^2
V-12	Initial	24.4	29.5	29.1	5262	952	34.7	36.7	37.0	100
	120	22.6	23.0	22.5		1051	22.6	23.0	22.5	mW/cm^2
V-4	Initial	23.7	17.3	25.0	5401	928	30.7	41.4	44.4	100
	140	14.8	15.9	11.9		1065	19.4	23.3	23.7	mW/cm^2

The relationship of I-131 uptake to elapsed time following 20 and 50 mW/cm^2 was ill-defined, whereas following a single 100 mW/cm^2 exposure the I-131 uptake indicated a time-related effect (Table VII).

Increased I-131 uptake occurred between 4 and 25 days after microwave exposure in normal, and whole-body X-irradiated animals (Table X). Between 173 and 194 days after microwave exposure there was no increase in I-131 uptake values from the pre-exposure level. In one upper-body X-irradiated dog there was increased uptake 11 days after microwave exposure (90 days after X-irradiation). In two other upper-body X-irradiated dogs, one had a normal uptake 12 days after microwave exposure (49 days after X-irradiation); the other had a decreased uptake 33 days after microwave exposure (83 days after X-irradiation).

I. Immune Response

Peak antibody titer and half-life values in 2800 Mc 100 mW/cm^2 exposed rabbits were reached later than in control animals (Table XI). Passively transferred antibodies were more rapidly eliminated or metabolized in the microwave exposed rabbits than in the controls (Table XII).

J. The Influence of Drugs on Microwave Response

Comparison of the thermal response in medicated and unmedicated dogs that were treated alternately in random order prior to 2800 Mc, 165 mW/cm^2 is shown in Table XIII. The medicated animals exhibited a more rapid increase in rectal temperature than unmedicated animals. During the initial 30 minutes of exposure, animals in the third stage of pentobarbital induced surgical anesthesia, developed the greatest increase in rectal temperature, 4.56°F. When Chlorpromazine and morphine sulfate were given, an increase of 2.5°F. and 2.13° F resulted during exposure. Unmedicated animals had a mean increase of 1.31° F from the pre-exposure temperature. The time (28.8 minutes) for the initial 4° F increase in rectal temperature to occur indicated that the pentobarbital sodium treated animals were least tolerant to microwaves. The rectal temperature in morphine sulfate medicated animals increased 4° F in 66.3 minutes; following Chlorpromazine the time required was 108.8 minutes. Rectal temperature increase was more rapid in morphine sulfate than in Chlorpromazine treated animals after the first 30 minutes of exposure. The time required for a 4° F decrease in rectal temperature after exposure, appeared to be dependent on the rapidity of the animal's temperature increase during the exposure. A rapid increase in rectal temperature of 4° F was associated with a prolonged cooling period to control levels.

Chlorpromazine administration suppressed the adaptation that results from daily microwave exposure (70).

K. Partial-Body

When head and rib-cage surface areas of comparable size in an anesthetized dog were subjected to pulsed 2800 Mc, 165 mW/cm^2, different rectal temperature changes occurred (Fig. 25, 26). The more rapid and greater rectal

TABLE X

I^{131} Uptake in Dogs After Electromagnetic Radiation

Exposure	Dog Number	% Uptake (24 hrs) (1) Pre Exposure	(2) Post X-ray	(3) Post Microwaves	Days Post Exposure (2) Post X-ray		(3) Post Microwaves
Microwaves[a]	5460	18	-	40	-	4	-
	5363	21	-	16	-	194	-
X-ray[b] Whole Body 300 R(MAD)[c] + Microwaves[a]	5470	23	-	34	-	18	(90)
	5364	24	-	33	-	25	(90)
	5159	18	20	20	10	173	(260)
	4938	18	17	12	10	180	(260)
X-ray[b] Upper Body 1800 R(MAD) + Microwaves[a]	5627	15	-	30	-	11	(90)
	5160	21	-	22	-	12	(49)
	5634	20	22	10	32	33	(83)

a = 1240 Mc/sec pulsed 50 mW/cm^2; 6 hrs/day; 5 consecutive days

b = 1000 kVp 55 R/min ( ) = days after X-rays for 3rd determination

c (MAD) = Midline Air Dose

TABLE XI

Effect of Exposure on Primary Antigen Stimulus (Rabbits)

(2800Mc/sec,100 mW/cm^2)

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 XII

Effect of Exposure on Disappearance Rate of Passively Transferred Antibodies

(2800 Mc/sec, 100 mW/cm^2)

Group	Number of Rabbits	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.

TABLE XIIIA

Thermal Response of Dogs Exposed to Microwaves After Medication

Drug	Dose	Thermal Equilibrium (minutes after onset of exposure)	Thermal Breakdown (minutes after onset of exposure)	Initial 30 Minute Heating(°F Increase Mean ± s.e.)	Initial 30 Minute Cooling(°F Decrease Mean ± s.e.)
Control		36-154	154	1.31 ± 0.16	3.75 ± 0.30
Pentobarbital	30 mg/kg	0	36	4.56 ± 0.75	2.25 ± 0.40
Chlorpromazine	2 mg/kg	43-173	173	2.50 ± 0.21	3.63 ± 0.29
Morphine	4 mg/kg	23-123	123	2.13 ± 0.40	2.63 ± 0.33

TABLE XIII B

Thermal Response of Dogs Exposed to Microwaves After Medication

Analysis of Variance

Drug	Dose	Heating		Cooling
		Maximum ^{o} F Increase Initial 30 Min. Heating	Time (Minutes) For Initial 4^{o}F Increase	Time (Minutes) For Initial 4^{o}F Decrease
Control	----	1.31 ± 0.47 *	172.5 ± 22.0	35.0 ± 8.45
Pentobarbital Sodium	To effect ca. 30 mg/kg iv	4.56 ± 0.47	28.8 ± 22.0	85.0 ± 8.45
Chlorpromazine	2 mg/kg im	2.50 ± 0.47	108.8 ± 22.0	39.5 ± 8.45
Morphine Sulphate	4 mg/kg sc	2.13 ± 0.47	66.3 ± 22.0	51.3 ± 8.45

* Mean ± Standard Error of the Mean

temperature increase from head irradiation in comparison with irradiation to the rib cage would indicate that the head is more sensitive to microwaves. An alteration in the thermoregulatory mechanism of the central nervous system is suggested as a possible explanation.

Rabbits that received 2800 Mc CW, to one side of the head developed hyperpyrexia (Table XIV). Lethality occurred in 30 minutes, at 180 mW/cm^2. Selective exposure to the eye at 90 mW/cm^2 or higher field intensity resulted in constriction or dilatation of the pupil.

L. Microwaves and Ionizing Radiation

Modification of response to ionizing radiation was observed in animals that received microwave exposure prior to, during, or after X or gamma irradiation. In 6-8 week old mice observed for 44 days following 750 R whole-body X-irradiation, the mean survival time was longer, and lethality least, in animals previously exposed to 2800 Mc CW, at 10 mW/cm^2 (Table XV). Survival time and lethality in mice given microwave treatment before and after X-irradiation were intermediate between microwave pre-treated and normal animals following X-irradiation. Survival time was shortest and lethality maximum when microwave exposure was given after X-irradiation.

Pulsed microwave pretreatment at 100 mW/cm^2 resulted in a generally prolonged survival time in 13 to 15 week old mice following 700 R, 800 R, or 900 R whole-body X-irradiation, and a significant reduction in lethality after 800 R exposure was observed (Fig. 28, Table XVI). Mice that received only microwave exposure had a 10 to 20% longer mean life-span than those not subjected to microwaves (Table XVII).

Lethality (40%) in rats of 200 to 250 g body weight that received 2800 Mc CW, at 39 mW/cm^2 for 38 minutes, before, during or after 700 R, 250 kVp X-rays, 18 R/min to the whole-body, was less than the lethality (80%) in rats that received only X-irradiation (Table XVIII).

Dogs subjected to microwaves several months to years after X-irradiation exhibited defective thermal regulation, as indicated by a greater sensitivity than that of normal dogs, to the thermal effect of microwaves (Fig. 22, Table XIX). These animals had a greater loss in body weight than did normal dogs after the microwave exposure. Leukocytosis was more persistent in IR dogs than in normal dogs following 2 hours of exposure at 165 mW/cm^2, 2800 Mc (Table XX). Following 1240 Mc, 50 mW/cm^2 exposure, whole-body X-irradiated dogs had lymphocytosis while normal dogs had lymphocytopenia. Mortality (30%) in dogs exposed to 400 R Co^{60} whole-body irradiation and four days later to microwaves, was unchanged following 100 mW/cm^2 exposure for 360 minutes, but was increased to 70 percent when 120 minutes of exposure at 165 mW/cm^2 was given. In Cr^{51} studies of red cell life span, the increased disappearance rate of tagged red cells was greater in animals that received Co^{60} and microwave (165 mW/cm^2) irradiation, than in animals exposed only to Co^{60} or microwaves. Retching or vomiting was observed only in IR animals during 1240 Mc exposure.

Dogs that were simultaneously exposed to microwaves and X-irradiation had

TABLE XIV

Response of Rabbits to Head Exposure From 2800 Mc/sec CW Microwaves

Rabbit No.	Distance From Horn (cm)	Field Intensity (W/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>+6F, 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. +3F, 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. +4.5F, 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. +3.75F.
R-41	25	0.041	30	No effect.

TABLE XV

Lethality in Mice Exposed to Microwaves

No. of Mice	Group	Mean Survival Time	44 Day Lethality
10	x-ray *	18.9 ± 6.2 days [a]	100%
10	x-ray - microwave **	12.8 ± 7.6	100%
10	microwave - x-ray - microwave	21.7 ± 11.9	80%
10	microwave - x-ray	26.0 ± 4.3	67%

a Mean ± Standard Deviation of the Mean

* 750 R Whole-body, 21 R/min, 250 kVp

** 2800 Mc/sec, 10 mW/cm^2, 1 hour daily for 3 days pre-X-irradiation and/or 30 days following X-irradiation

TABLE XVI

Thirty-Day Mortality in Mice after Whole-Body X-irradiation
14 or 30 Days After Microwave Treatment

Group	X-irradiation	Mortality	Percent	Mean survival time (days)
		Single Microwave Treatment		
Untreated	700 R	17/58	29.3	13.6 ± 0.67[a]
Treated		10/40	25.0	15.1 ± 0.66
Untreated	800 R	44/58	75.9	12.9 ± 0.47
Treated		30/60	50.0	14.2 ± 0.55
		$P < 0.01$		$P < 0.1 \geq 0.05$
Untreated	900 R	54/57	94.7	12.5 ± 0.36
Treated		52/58	89.7	14.1 ± 0.37
				$P < 0.01$
		Daily Microwave Treatment		
Untreated	800 R	16/20	80.0	11.7 ± 0.75
Treated		10/20	50.0	11.6 ± 1.33
		$P < 0.1 \geq 0.05$		
Untreated	900 R	18/20	90.0	12.7 ± 0.71
Treated		17/18	94.4	14.5 ± 0.85

[a] Mean ± Standard Error of the Mean

Fig 28

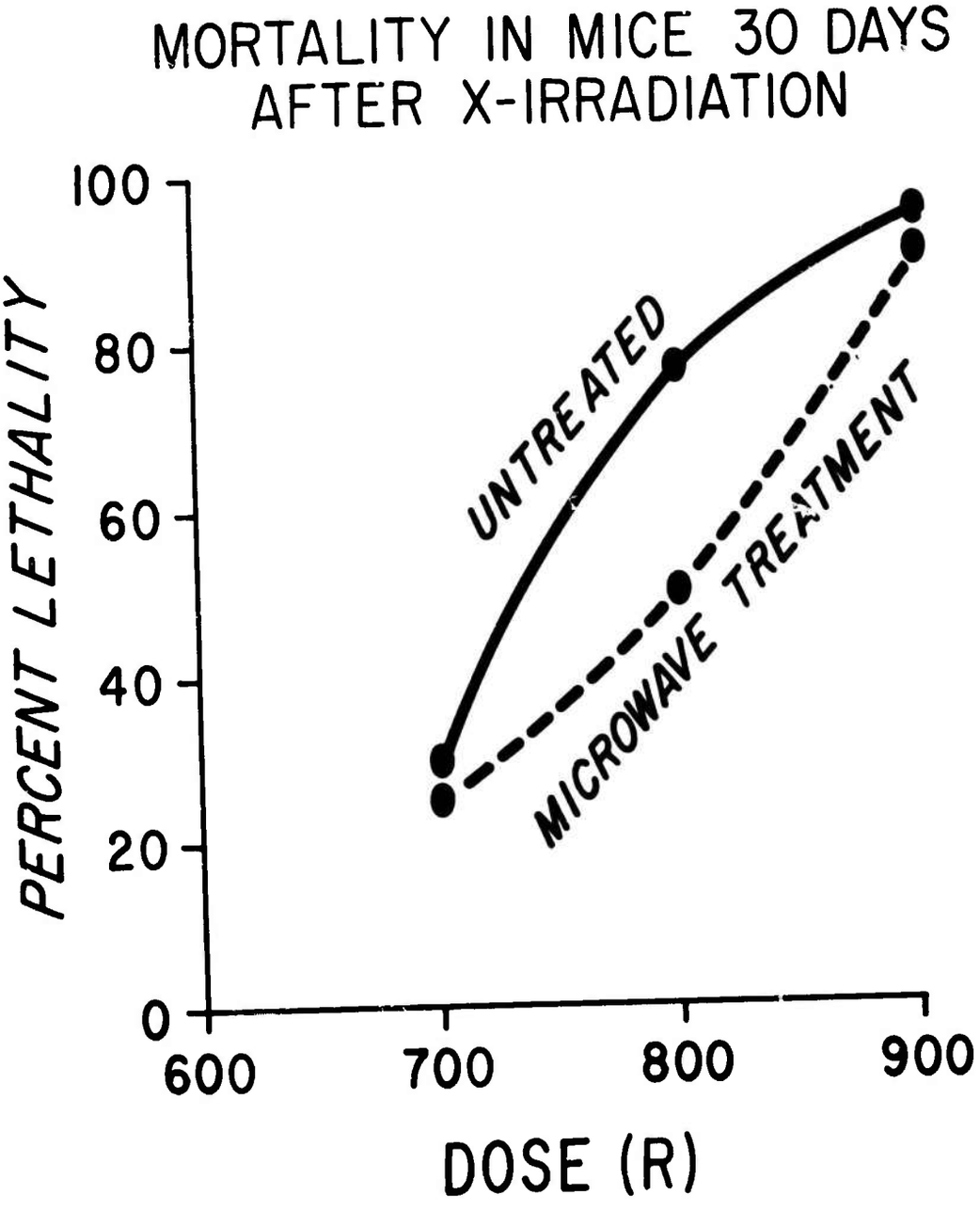

MORTALITY IN MICE 30 DAYS
AFTER X-IRRADIATION
PERCENT LETHALITY
100
80
60
40
20
0
600
700
800
900
DOSE (R)
UNTREATED
MICROWAVE TREATMENT

TABLE XVII

Survival Time (Days) of X-irradiated Mice[a]

Mice	Group				
	Control	700 R	720 R	750 R	800 R
Normal	532 (70)	312 (68)	211 (36)	375 (6)	257 (12)
	± 21.6*	± 17.7	± 16.9	± 54.7	± 52
Microwave	622 (31)	337 (58)	246 (33)	301 (6)	347 (19)
	± 33.2	± 21.5	± 21.0	± 61.0	± 8

	Pooled Data
X-irradiated	280 (122)
	± 14.9
Microwave and X-irradiated	311 (116)
	± 13.7

[a] Excludes data during acute post-X-irradiation (30 day) period

* Mean ± Standard Error of the Mean

( ) Number of animals in group

TABLE XVIII

Mortality in Rats Following Exposure

to Microwaves and X-radiation

Group	Number of Animals in Group	Mean Rectal Temperature °F	Days Post Exposure	30-day Mortality (%)
Controls	5	-----	------	0
Microwave plus X-ray	5	104	14, 11	40
X-ray plus Microwave	5	108	21, 10	40
X-ray[1]	5	-----	10, 15, 16, 17	80
Microwave[2]	5	107.5	------	0
X-ray and Microwave simultaneously	5	104.5	12	40

1 - 700R, 250 kVp

2 - 2800 Mc/sec CW, 39 mW/cm^2, 38 min

TABLE XIX

Effect of Time Lapse and Total Ionizing Radiation Exposure on Dog Response to Microwaves

Whole Body Ionizing Radiation				Microwaves 2800 Mc/sec, 165 mw/cm^2		
Number of Dogs	Total Dose (r)	Number of Exposures	Months Before Microwaves	Duration of Exposure (Min)	Rectal Temperature (F) Terminal	T
7	1057* (400-1324)	1-4	37 (7-59)	103 (65-185)	107.5 (106.0-111.0)	+6.2 (+4.50-+9.25)
4	825 (595-1416)	1-4	24 (11-36)	140 (120-175)	106.1 (105.0-107.0)	+4.3 (+3.50-+5.25)
6	399 (350-594)	1	12.5 (10-16)	128 (55-195)	106.3 (104.0-107.5)	+4.3 (+2.00-+5.50)
6	365 (332-400)	1-7	3 (2-4)	232 (120-360)	105.8 (104.00-107.25)	+4.3 (+2.00-+6.00)
31	Normal			165 (95-290)	106.3 (102.75-110.8)	+4.4 (+1.25-+8.75)

* Mean

( ) Range

TABLE XX

Hematologic Changes in Dogs Exposed to Microwaves

Time After Exposure	% Change WBC				
				Survivors	
	Sham	100 mW/cm^2 6 hr.	165 mW/cm^2 2 hr.	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
48 hr.	---	+ 10.4	- 3.1	+ 28.4	+ 30.6
1 wk.	+ 5.1	+ 3.2	- 1.8	+ 12.4	- 23.1

differing responses to that which results from X-irradiation alone. Earlier neutrophil recovery and delayed hematocrit recovery resulted from simultaneous 2800 Mc pulsed, 100 mW/cm^2, and 720 R (250 kVp X-rays, 2 R/min) exposure for six hours. Death did not result from the X-ray dose used. Simultaneous exposure at a lethal X-ray dose level, 1656 R (250 kVp, 4.6 R/min), caused an increase in lethality from LD 30/60 to 100/60 (Table XXI). Microwave treated dogs, X-irradiated (340 R, 1000 kVp, 55 R/min) one month to thirty-two months after microwave exposure, had a varying mortality response, which suggested that lethality from X-irradiation may be affected by the total microwave exposure as well as the time interval between exposures. X-irradiation lethality in normal dogs was LD 68/60 while in microwave pre-treated dogs, it ranged from LD 17/60 to LD 40/60 (Table XXII). In another study, where the X-irradiation was 1656 R (4.6 R/min, 250 kVp) lethality was LD 70/60 in microwave pretreated dogs and LD 30/60 in dogs not exposed to microwaves (Table XXI, Fig. 29). The data revealed that the animals with the least number of consecutive microwave exposures survived the X-irradiation.

None of nine dogs previously exposed to microwaves succumbed to 950 R, MAD, 1000 kVp, 50 R/min, lower-body (xiphoid process caudad) X-irradiation, while three of eight normal dogs died within four days. Intestinal hemorrhage was less severe in the animals pretreated with microwaves.

Following 25,000 R X-irradiation to the head, survival time was significantly increased from a mean of 22 hours for animals not treated with microwaves to 43 hours in animals pretreated with microwaves (Table XXIII). Neurologic changes following X-irradiation were less pronounced in the animals that received microwave treatment.

4. DISCUSSION

With the progressive improvement and increased power of radar units for military and civilian use, questions as to their potential hazard have appropriately been voiced. During the past 25 years numerous experiments on animals and several human surveys have been performed in the U.S.A. and the USSR to answer some of these questions. It has been shown that microwaves affect a variety of organisms from protozoa to mammals. Effects may occur at low intensities of irradiation and are characterized by responses that involve reaction of the entire organism, to changes at the molecular level (137).

A. Thermal Effects

Microwaves exhibit the general properties of electromagnetic waves. They can be reflected, scattered, or absorbed depending on the size and composition of the biologic material interposed in their path. When tissues are exposed to microwaves, the radiant energy absorbed is transformed into increased kinetic energy of the absorbing molecules, thereby producing a general heating of the entire medium. This effect on biologic systems is modified by mechanisms of heat loss primarily neurocirculatory in type. When the thermoregulatory capability of the animal is exceeded, death can result.

Microwave absorption in tissues is a function of ionic conduction and of

TABLE XXI

60 Day Lethality in Dogs Following 1656 R Whole Body X-irradiation

Exposure	Lethality		Survival Time (Days)			Mean
X-ray	3/10	30%	11,12	17		13.3
X-ray 9 Months Post Microwave Exposure	7/10	70%	12,12,12,13	21,23,25		16.9
Simultaneous Microwave and X-ray	5/5	100%	9,9,11,11	15		11.0

Fig 29

RELATIONSHIP OF MICROWAVE EXPOSURE AND LETHALITY IN DOGS FOLLOWING X-IRRADIATION

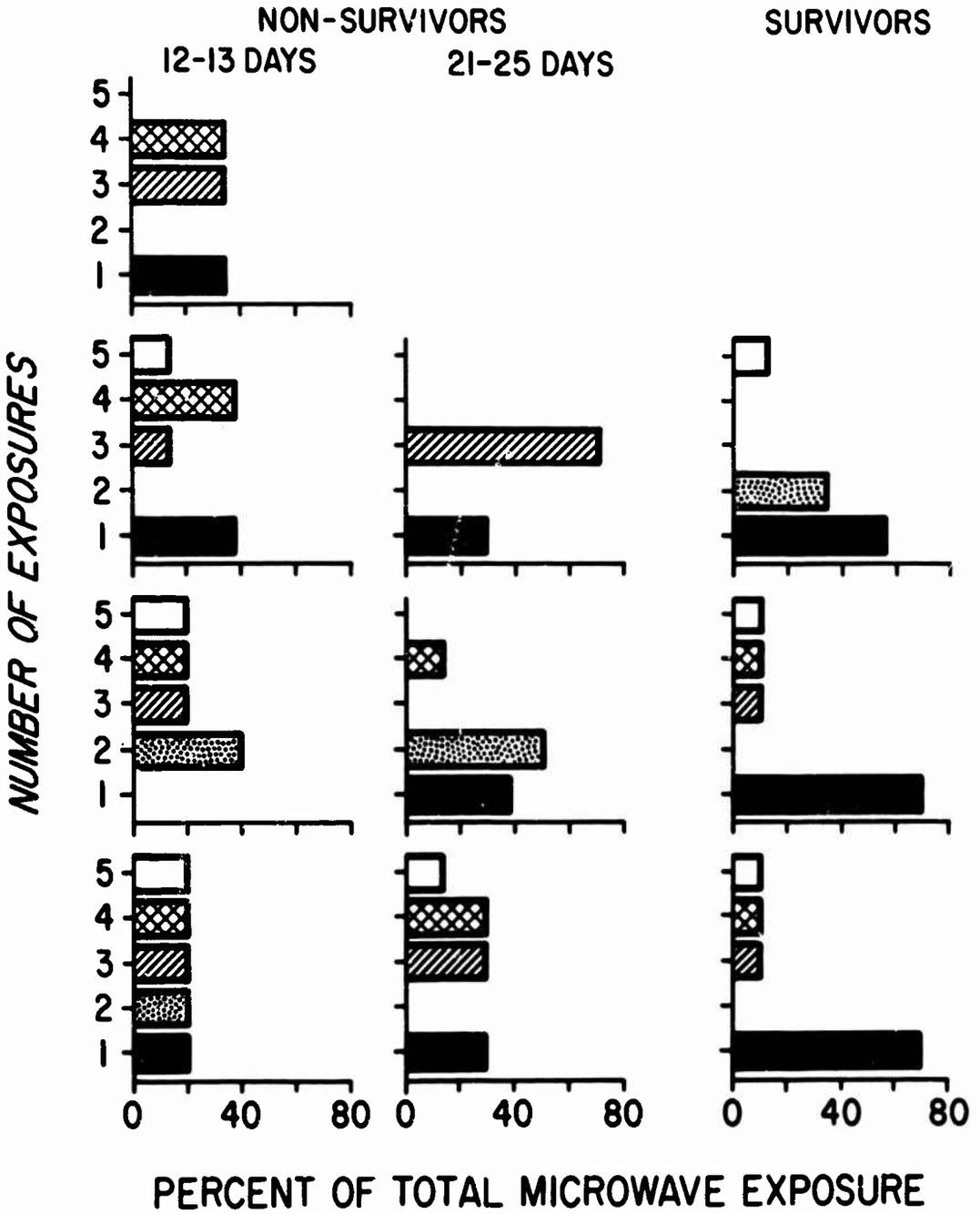


TABLE XXII

Lethality in Microwave Treated Dogs Exposed to 340 R MAD Whole Body Ionizing Radiation

(30-Day Lethality)

2800 Mc/sec Microwave Radiation				Ionizing Radiation		
Group	Exposure	Duration (Min)	Months Pre-Ionizing Radiation	Mortality		Survival Time (Days)
1	Normal (X-ray only)	-	-	23/34	67.6%	14.9 (±0.62)*
2	100 mW/cm^2 (single)	180-360	5-32	1/6	16.7%	11.0
3	100 mW/cm^2 165 mW/cm^2 (multiple)	270-2730	7-30	4/10	40%	17.5 (±1.50)
4	165 mW/cm^2 (single)	120-450	4-21	6/10	60%	17.3 (±1.02)
5	100 mW/cm^2	30-90	1-15	3/4	75%	16.7 (±1.18)

* Mean (± Standard Error of the Mean)

TABLE XXIII

Survival Time of Dogs That Received Ionizing Radiation to the Head

Midline Air Dose (R)		No. of Dogs	Survival Time
25,000	Normal	10	22.0 (±1.37)* hours
	Microwave	10	43.1 (±6.70) hours
10,000	Normal	5	13.8 (±0.73) days
	Microwave	5	15.4 (±1.03) days
5,000	Normal	5	40.6 (±4.68) days
	Microwave	5	43.6 (±15.4) days

* Mean (± Standard Error of the Mean)

relaxation of dipole molecules. Both processes account for the transformation of microwaves into thermal energy. The shorter the wave length the greater is the amount of energy absorbed by tissue due to dipole loss.

The percentage of microwave energy absorption ranges from 50% at 10 cm to 98% at 3 cm wavelengths. Tissues with a high water content, such as skin, muscles, and the brain are penetrated 2-4 cm by microwaves in the meter range, 1-2 cm in the decimeter range, and 6-8 mm in the centimeter and millimeter ranges (137). Absorption coefficients of body tissues depend on their water content, i.e. fat, bone, and yellow marrow will absorb relatively less than muscle tissue (154, 155). Skin, muscle, and internal organs have high water content, high dielectric permittivity, and low resistivity; fat, bone, bone marrow have low water content, low dielectric permittivity and high resistivity (94). Besides the differences in absorption of various tissues, factors such as physical characteristics, angle of incidence of the energy and the anatomical configuration of the subject are important considerations. The physiologic state of the subject and the enviroment also influence the biologic response. Non-thermal effects of microwaves, if they occur, are likely due to resonance. The relationship of frequency to proportion of microwave energy absorbed is shown in Table XXIV. Frequencies greater than 3000 Mc approach infrared frequencies and surface heating predominates. Since the skin contains most of the body's nerve endings or sensory elements, heating of this layer is perceived immediately. It is important not to equate the biological effects of microwaves and infrared radiation because the processes of transformation of energy from these two forms of radiation into heat differ.

Heat produced, is in turn, diffused from the irradiated portions of the body by the vascular system. The breakdown of protective mechanisms for heat control causes an uncontrolled rise in body temperature.

An excessive increase in body temperature produces damage indistinguishable from fever of other origin. The increase in temperature in irradiated tissues during local exposure occurs linearly for short periods (1-3 minutes) and is related to the quantity of the microwave energy absorbed. With exposures in excess of 3 minutes the magnitude of the thermal effect and distribution of heat in tissues is determined by heat-regulating mechanisms (120). Intermittent exposure can be better tolerated for longer periods, than constant exposure at comparable power levels, owing to processes of heat dissipation. The rate at which the heat is dissipated will vary in different species depending on heat-regulating mechanisms or metabolic characteristics. Water intake during exposure enables normal animals to tolerate longer exposure.

A cumulative effect from microwave exposure has been noted (21). Intermittent exposure for brief periods allows evaluation of the heating and cooling capability of the subject. Such a technique may be used for the determination and assessment of alterations in vascular or nervous function which might occur from injury or senescence.

The development of skin burns over the rib cage suggests a differential sensitivity of specific body areas. Because water readily absorbs microwave

TABLE XXIV

Biological Characteristics of Microwaves

Frequency Mc/sec	Wavelength cm	Energy eV	Relative Absorption Cross Section %		Tolerance Levels (incident power) density
			Fat Thickness 1-3 cm / Skin Thickness 0.2-0.4 cm	Body	
400	75	10^{-7}	43-73		
900	33	10^{-6}	51-89	50-100	10 mW/cm^2
3000	10	10^{-5}	18-50	20-100	20 mW/cm^2
10,000	3	10^{-4}	41-50	50	

Adapted from Anne, A. (ref:4)

energy, the distribution of fluid in the exposed subject is an important factor in the development of burns.

An early manifestation of any heat stress for the mammal is hemodilution, which occurs during the first 30 min. of exposure prior to temperature increase. As the exposure is prolonged the hemodilution is reversed as a result of dehydration, and hemoconcentration follows. The early hemodilution possibly reflects an influx of extravascular fluid as a result of the extensive peripheral vasodilation. This permits dissipation of absorbed heat.

Exposure to microwaves produces a response similar to that from other heat stresses. In the dog, signs of discomfort and distress are less evident during 1280 Mc than during 2800 Mc exposures. At 2800 Mc greater discomfort and temperature rise is noted than at 200 Mc for the same field intensity and comparable time periods. These findings suggest that the peripheral nerve endings are more affected by the shorter wavelengths which produce surface heating, as contrasted with the longer wavelengths with their greater depth of penetration. Studies on nerve stimulation support this conclusion (185).

Microwave energy is reflected at the boundary between media: skin-fat and fat-muscle. The thermal effect can increase or decrease depending on the angle of reflection. If reflection occurs at the fat-muscle boundary, fat will absorb more energy in spite of the fact that it is more "transparent" than is muscle. No reflection will occur at the fat-muscle boundary if the "electrical length" (thickness) of the fat adjacent to the muscle equals a quarter or an odd number of quarters of the wave-length (120).

When dogs were exposed to 3000 Mc at 10 mW/cm^2 for 20 minutes, body temperature did not increase (181). In rats, comparable exposures were thermogenic. This difference indicates either the influence of body size or inherent thermal regulatory capability of the animal.

The thermal effects of 10,000 Mc has been studied (13,75,120,134,181). In rats exposed to 10,000 Mc the temperature of all organs increases simultaneously from the beginning of irradiation with linear rise during the initial 3-4 minutes. After 5 minutes of exposure the temperature increase is less rapid. In mice exposed to 10,000 Mc the body temperature rise is proportional to absorbed energy during the first 3 minutes, after which it plateaus. The height of the plateau is related to microwave intensity and the adaptation to exposure.

Tolgskaya (178) studied the effects of pulsed and CW 3000 and 10,000 Mc microwaves on rats at various intensities. Emphasis was placed on morphologic changes. The more pronounced morphologic changes in the nervous system following 3000 Mc than 10,000 Mc at 1-10 mW/cm^2 was interpreted as evidence of a non-thermal effect. Pulsed waves are more effective than CW. The greater effectiveness of pulsed microwaves was also noted by Marha (106).

Species sensitivity to 2800 Mc decreases from rabbit, to rat, to dog. Repeated exposures to 2800 Mc at 100-165 mW/cm^2 shows some form of adaptation. This process may indicate a "physiologic memory" capable of being blocked by

Chlorpromazine (70).

The thermal response in dogs exposed to 1280 or 2800 Mc is characterized by three phases: (a) initial heating, (b) equilibrium, and (c) breakdown. Varying degrees of disruption of equilibrium related to the increase in rectal temperature are noted. Most animals have an increased desire for water, those with most marked changes refuse water. Rectal temperature decrease after termination of exposure is exponential; recovery occurs within one hour. Exposure at 165 mW/cm^2 for three hours causes hyperthermia which may be lethal. The animal develops thermal equilibrium during exposure of up to six hours' duration at 100 mW/cm^2; rectal temperature increase is slight. Repeated exposure to 100 mW/cm^2, results in adaptation as evidenced by the ability to prolong exposure time. Initial temperature and the elevation during irradiation decrease with each exposure. Repeated exposure can be tolerated for extensive periods and is directly related to the interval between exposures. This provides sufficient time for recovery.

Addington (2) noted that heating occurred more rapidly and to a greater degree in dogs exposed to 200 Mc CW when the longitudinal axis of the body was parallel to the polarization plane of the microwave field. Such positioning of the animal permits greater absorption of the energy in the exposed mass, in relation to the wavelength.

Exposure of the head of rabbits to 10,000 Mc (220 mW/cm^2 for 20 min.) causes a 1.5 - 2.0°C rise in the temperature of jugular blood (120). Localized exposure to the skin of rats at 10,000 Mc, 350 mW/cm^2 shows a linear temperature rise during the initial 3 to 4 minutes at a depth of 1 - 2 mm. Measurement at 2.5 to 3.0 mm indicates a non-linear increase which reflects heat transfer from the skin to deeper tissues. During exposures at 350, 220, and 100 mW/cm^2 for 5-20 min., thermal equilibrium is established (120). Tissue heat is diffused deeper into the organism with increase in blood volume and flow velocity to dissipate the heat. Experiments using phantoms reveal that the linear portion of the temperature curve continues up to 9 minutes and the equilibrium phase does not develop. The difference in pattern of temperature increase in animals and phantoms points to the thermal-regulation capability of the organism which enables a lowering of body temperature in order to maintain thermal equilibrium. Drugs such as pentobarbital sodium, morphine sulfate, or Chlorpromazine which affect the thermoregulatory center increase the susceptibility of animals to microwaves (110). Caffeine is reported to increase brain sensitivity to microwaves (56).

Tolerance to microwave exposure decreases as the ambient temperature increases (108). Rats subjected to 24,500 Mc, 250 mW/cm^2 show a two-fold increase in survival time when the environmental temperature is reduced from 35° C to 15° C. Increased air circulation also results in a prolongation of survival time (36).

The exact nature of the biological interactions of microwave exposure is not known. Evidence suggests that microwaves can act as a "stressor" agent, with effects on regulatory and integrative mechanisms of the body resulting in alteration of homeokinesis. Studies in this laboratory indicate a

hypophysial-hypothalmic-adrenal effect. A sharp decrease in eosinophils and lymphocytes, and leukocytosis suggests such an action. The general adaptation syndrome (stress) is defined as the integration of the adaptative reactions of the organism to any insult (heat, chemical, psychic). These reactions occur in the following sequence: the acting agent (stressor) stimulates the hypothalamus which in turn through the hypophysis stimulates hormonal activity. The above described blood changes are characteristic of the early manifestations of thermal stress.

As interpreted by Pressman (137, 139) the stress reactions to average and high-field intensity microwaves arise through the direct action on all skin receptors (not only the heat receptors) and on the brain. The effects produced by irradiation at low-field intensities originate only through action on the central nervous system. When man is exposed to low frequency microwaves (600-1000 Mc) the effect on the subcortical structures predominates over the effect on skin receptors. At higher frequencies only the skin receptors are affected.

In experiments on rats, the authors and others report studies in which death occurs during irradiation or at certain periods after exposure. It is impossible to establish a simple lethality: intensity: duration: frequency relationship. This must depend on the combination of all conditions of irradiation as well as the state of the animal.

The influence of wave-length on lethality has been noted by several investigators (137, 153).

At 200 Mc CW one dog died after 31 min. exposure to 200 mW/cm^2; in guinea pigs death occurred after exposure to 432, 500,or 680 mW/cm^2 for 28, 40, or 18 min., respectively (2).

Exposure to 400 Mc resulted in death in rabbits after 30 - 40 min. at 50 mW/cm^2 (13) and in rats after 30 min. at 100-120 mW/cm^2 (101).

Lethality in rats occurred after 15 minutes at 2450 Mc, 70 mW/cm^2 (143).

At 10,000 Mc, 400 mW/cm^2 rats died in 13 - 14 minutes (120). During irradiation at 200 mW/cm^2 survival time was 25 - 30 minutes (181).

Exposure at 24,000 Mc pulsed, 300 mW/cm^2 was lethal to rats when comparable areas of abdomen, hind quarters, or head were irradiated for 12, 15, or 19 minutes (38).

The appearance of local lesions of the skin and underlying tissues from microwave exposure has been observed (2,13,14,47,59,70,77,110,125,130,131, 143,163). Development of the lesions is related to the thermal effect of microwaves. Coagulation necrosis of the skin of rats was observed after exposures which caused a local temperature elevation to 50-55° C in the irradiated area (47).

Skin lesions in rats and rabbits exposed to 10,000 Mc at 50 mW/cm^2 were

described in detail by Slabospitskiy (163). Skin biopsy immediately after irradiation revealed multiple hemorrhages, unevenly distributed in foci of various sizes chiefly at the boundary of the derma and the muscular layer. Most of the blood vessels were engorged. Leukocyte reaction was negligible; cellular infiltration, absent. Biopsy at twenty-four hours after irradiation revealed more marked changes. The hemorrhagic foci contained many leukocytes. In almost every nerve trunk, damaged nerve fibers were visible. The myelin sheath was especially involved. Five days after irradiation, brownish foci of mummified areas of necrosis of the skin were visible. Isolated areas of coagulation necrosis penetrating to the subcutaneous tissue were noted. In such areas the cells of the epidermis were indistinctly outlined, their protoplasm vacuolized, the nuclei pyknotic, and a homogeneous appearance noted. The affected section of the skin was considerably thickened. According to the author, microwave induced skin lesions do not resemble those induced thermally at comparable skin temperatures. Because of the depth of tissue injury, the microwave lesions of the skin should be compared with fourth degree burns. Higher temperatures from other thermal sources are needed for the development of burns comparable to those caused by microwaves. Minimally expressed symptoms of inflammation are a peculiarity of microwave effects. Lesions of the skin may develop without signs of the blisters which are characteristic of second and third-degree thermal burns. The absence of pain and tactile sensitivity of the skin area irradiated by microwaves is also noteworthy. Increase in the skin temperature to $55\pm3^{\circ}$ C during a comparatively short interval (three minutes) indicates heat induction that exceeds the rate of heat loss. Exposure to microwaves of the same intensity but with skin heating reduced did not produce pathological changes. The skin lesions appear to be the result of the action of heat given off during the absorption of microwave energy (130).

In warm-blooded animals the heat emission by conduction, radiation, and evaporation becomes of practical importance for heat regulation. Almost 90 percent of heat loss is through the skin. At high temperatures, heat radiation through the skin is the primary mechanism for maintenance of body temperature. The thermal effect in the skin during microwave exposure depends on the physical properties of skin as a dielectric. Microwave injury to nerve elements and blood vessels of the skin may lead to denervation and irreversible stasis of the blood within the irradiated area. This results in the typical microwave lesion. Subsequent changes lead to a necrobiotic process with mummification of the tissues. Disturbance of the innervation of the skin area caused by intense microwave absorption is seen as one of the chief aspects of microwave pathology. This can partially explain development of stasis in the blood vessels.

Burn production from microwave exposure has been described in rats exposed to 24,000 Mc, 40 mW/cm^2 (77). Acute exposure of mice to 2450 Mc and 10,000 Mc resulted in hyperemia, congestion, and hemorrhage; repeated exposures for 30 minute periods resulted in hemorrhage and progressive fibrosis in the brain, lungs, liver, and kidneys (143). Gastric ulcers have been produced in rabbits following exposure of the epigastric region to Super High Frequency (SHF) at 70-160 mW/cm^2 (131). Mice given 10,000 Mc, 400 mW/cm^2 to the whole body developed necrosis in the myocardium and liver (59). Focal lesions have been produced in the cerebral cortex of rabbits exposed to 2450

Mc (125). Degeneration of neurons in the cerebral cortex and retrograde changes in the kidneys and myocardium of rabbits have been produced by exposure to 200 Mc (14). Subcutaneous burns developed in a dog exposed to 200 Mc CW, 220 mW/cm^2 for 22 minutes and in a goat exposed to 165 mW/cm^2 for 40 minutes (2).

Schaffer (149) reviewed experimental data involving microwave heating of small animals and proposed experiments to equate temperature-time responses to microwave irradiation. These data reveal wave-length, animal species, competence of the thermo-regulatory mechanisms, the area peculiarities, and environmental conditions to be important factors affecting the thermal threshold.

B. Non-thermal Effects

The possibility that microwaves may interact with biological material without the production of significant heating is pointed out by several investigators. Some of these effects are analyzed by Sher (161) and Schwan (151). Resonance absorption in living cells is considered to be a non-thermal effect of microwaves (49, 135, 146). Paramagnetic resonance analysis is used to determine dipole moments of protein molecules and to elucidate the structure of crystalline proteins (140). Pearl-chain formation is demonstrated in organic and inorganic particulate suspensions (68). Hall effects in water and the existence of protonic semiconductors in biological materials are reported (170). Microorganisms move in directions associated with the field (172). Organisms such as planaria and snails react to the polarity of weak electric and magnetic fields (15, 16).

Some fish are able to navigate by detecting distortions in a self-generated electric field (93). Neuromuscular responses occur in birds in a rf field (171). Changes in the paper electrophoretic pattern of human gamma globulin after isothermal irradiation by microwaves are described (6). Inactivation of RN-ase and DN-ase with increased RNA and decreased DNA are reported (78). Reduction in size of tumors results following 2000 Mc and 2450 Mc irradiation (183). Garlic root-tips mutate when grown in a high-frequency field (67). Visual stimuli occur in humans from fields too weak to depolarize visual neurons in the usually understood way (86); people hear sounds corresponding to the frequency of modulation of the incident beam (50, 51).

Increase in rectal temperature does not result from whole-body exposure at 20 mW/cm^2, 1280 Mc pulsed (111). At 3000 Mc, CW or pulsed, and 10,000 Mc pulsed, 10 mW/cm^2 or less, increase in body temperature does not occur (46, 57). Mirutenko (119) observes no increase in skin temperature at a depth of 0.5 to 0.8 mm from 10,000 Mc at field intensities below 1.5 mW/cm^2. Gordon (56) states that radiation under industrial conditions over a period of one to two months at intensities producing minimal thermal effect can cause severe functional changes in the central nervous system.

According to Presman (139) who states a Soviet view, intensities below 10 mW/cm^2 may be considered non-thermal for pulsed and CW microwaves of biologically effective frequencies in either whole-body or local irradiation.

With intensities of 10 mW/cm^2 or less, conversion of microwave to thermal energy does not exceed the heat loss from 1 cm^2 of body surface under normal environmental conditions.

Schwan (151) and Sher (161) report on the effect of microwave fields on particles dispersed in liquids. Four non-thermal effects of alternating electrical fields are assessed for possible biological significance. The effects, which are the result of mechanical forces on particles dispersed in a liquid are: (a) pearl-chain formation (linear orientation) - does not occur in humans exposed to microwaves at an incident power density of less than 10 mW/cm^2; (b) orientation of nonspherical particles - is improbable since biologic structures are insufficiently superficial, large, or free to be so oriented; (c) forces in inhomogeneous fields appear to have no biological significance, since skin has no freely movable, micron-size particles; (d) the effect of AC (alternating current) electrophoresis at microwave frequencies is infinitesimal. These forces may have biological manifestations on a cellular scale but are ineffective for sizes smaller than those microscopically visible. None of these four non-thermal effects, therefore, has biological significance for humans exposed to a physiologic "thermally safe" microwave field.

C. Biomedical Aspects

The eye, testes, thyroid, bone marrow, cardiovascular and central nervous system are subject to structural or functional alteration by microwaves.

1. The eye

The eye is one of the few organs that can be directly exposed to microwaves without the influence of intervening skin and other tissues. The superficial location and structural characteristics of the eye make it particularly susceptible to microwave injury. The lens of the eye is sensitive to ionizing, rf, or infrared energy, each of which causes the development of characteristic opacities. Considerable experimental work is reported on cataract production by microwaves (1,11,17-23,27,29-33,46,73,101,128,133,143-145, 148,159,188). Investigations to 1956 indicate: a) 500-600 mW/cm^2 produces cataracts in rabbits; b) the threshold value for injury from a single 270 minute exposure is between 120-200 mW/cm^2; c) the temperature in the vitreous humour near the posterior lens capsule in which structural changes occur is 49-53°C.

Richardson, et al (145) note clouding of the lens after heating of rabbit eyes to 50° C with 10,000 Mc. With this superficial energy the changes occur in the anterior segment, with clouding of the cornea. Irradiation of excised cow's eyes with 2450 Mc results in maximum temperature increase in the location where damage appears when eyes are irradiated in situ (73). Salisbury et al (148) find that microwave radiation produces lens changes more rapidly than infrared, ultraviolet, or X-irradiation.

Addington et al (1) report no eye changes in guinea pigs, dogs, sheep, or mice from chronic exposure to 200 Mc CW. Lens changes do not occur in rabbits given 400 Mc whole-body exposure even if radiation times are

extended to the lethal period (101).

Carpenter and associates (17-23) find that repeated irradiation of rabbit's eyes with 2450 Mc can cause clouding when the lens temperature increases 4° C. No discomfort is noted until the eye temperature increases 7°C. The greater the microwave field intensity the less is the time required to produce lenticular clouding. The microwave power level required to produce these opacities is a constant (power $\times\sqrt{\text{time}}$ = constant). Latency time observed for lens changes averages 3.5 days. Immediately after exposure hyperemia in limbal and iris vessels, contraction of pupils, swelling and chemosis in the conjunctiva, and thread-like formations in the lens occur. Changes initially are temporary and slight. The greater the radiation intensity the higher the temperature. Lens clouding appears with repeated exposure at those intensities which produce no observable effect with a single exposure. The first change appears in the posterior subcapsular cortex as concentric lamellar opacities; some of these clearing in several days.

Cataracts produced by microwave frequencies near 2450 Mc resemble those of ionizing radiation with respect to location (posterior cortex of the lens) and morphology, but show a shorter latent period (26,27). The important factor in production of a cumulative lens change is the interval between exposure. Ascorbic acid content of the lens decreases 6-18 hours after irradiation. With the development of a cataract, water content is increased. The induction of an opacity is not related to critical intraocular temperature in these experiments. Other experiments suggest that pulsed microwaves may have a more injurious effect than CW. The thermal effects of pulsed waves are directly proportional to the average field intensity. On the basis of his findings, Carpenter believes that the cataractogenic effect of microwaves at 2450 Mc is not thermal. Microwaves do not shorten the latency of X-ray induced lens changes (26). Infrared radiation sufficient to raise the temperature in vitreous humour to the same level as microwaves (4°C) produces clouding of the lens localized to the anterior cortex. Pol (133) notes that 10,000 Mc causes opacities on the anterior lens surface and 2450 Mc, on the posterior surface.

A major variable in the interpretation of these results is the validity of the reported power levels (185). A discrepancy exists between Carpenter's results and those in this laboratory in which CW exposure at similar intensity for as long as one hour did not result in permanent lenticular changes (159). Repeated exposures for short periods did not produce a cumulative effect. Experimental techniques and differences in power level measurements may account for the variations in results. Carpenter conducted his experiments in an anechoic chamber, while those in this laboratory were performed in "free air."

Microwave induced opacities in the posterior cortex, may result as an interface effect at lens cortex-posterior capsule boundary or at capsule-vitreous body boundary, with concentration of the energy in the posterior cortex from reflection of microwaves. Thus, the temperature could be higher in the cortex than in the vitreous immediately behind the lens where ocular temperatures are usually recorded.

Following microwave irradiation of sufficient intensity to result in

early cataract formation, a loss of phosphatase activity of the lens occurs (33). At low exposure levels, with delayed cataract formation, no significant reduction of adenosine triphosphatase or pyrophosphatase is noted. Rabbits with alloxan induced diabetes are more susceptible to microwave induced cataract formation than are non-diabetic rabbits (32). The results suggest that microwave irradiation produces a "metabolic imbalance."

Studies employing C^{14} labeled adenine in lenses from rabbits' eyes exposed to 2800 Mc CW indicate an increase in turnover of the albuminoid RNA fraction. Ionizing radiation of the lens causes a similar but more marked increase in apparent turnover of albuminoid RNA (90). Further studies show this fraction is derived from soluble RNA of the lens. A significant increase in the permeability of the capsule occurs. Impairment of labeled sulfate turnover is demonstrated. Extensive studies on protein and nucleic acid metabolism fail to reveal significant differences between experimental and control lenses. Such effects appear to be thermal in nature.

Cataracts are reported in workers exposed to rf (70, 115, 162). Barron et al (9) and Daily (28) did not find changes in eyes of persons working with radar.

Minecki (115) found a haziness in the posterior lens suture among workers exposed to high field intensities. The incidence of such changes relates to duration of employment as follows: 38% of individuals working less than 1 year, 55% up to 3 years, and 67% up to 5 years.

Zaret (189-191) also reports eye opacities in persons working with radar. After a survey of a large number of exposed individuals, occurrence of opacities was compared with a control population of similar age groupings. A statistically significant difference between the eyes of microwave exposed and control populations is stated. Posterior polar lens changes are more prominent in microwave workers. The number of defects increases linearly with age. Although an apparent statistical difference in the score of lens changes between the exposed and control groups exists, the difference is considered not significant from a clinical standpoint. The extent of minor lenticular imperfections does not serve as a useful clinical indicator of cumulative exposure. A relationship can be established between the dose of microwave radiation delivered to the eye and the appearance of cataracts (189). From repeated exposure at 5 Watts/cm^2, cataracts are formed in two months. At 500 mW/cm^2, several months elapse prior to appearance of posterior capsular opacification. At this level, several years are required for production of formed cataracts.

Information concerning microwave effects on the eye is based primarily on acute and sub-acute studies because chronic exposure studies are few, inconclusive and to date do not provide a basis for estimating the minimal exposure dose for military and civilian radar workers. A paucity of information exists on those characteristic changes that are microwave frequency dependent. In the absence of more specific information concerning chronic exposure and frequency dependent changes, an increase in the 10 mW/cm^2 maximum permissible exposure (MPE) is not recommended.

2. Hematopoiesis

Analysis of blood changes provides a basis for evaluating the biologic effects of microwaves. Most data are based on studies using rodents, rabbits,and dogs under controlled exposure conditions, or in man where field intensities and duration of exposure are not easily measured (8,9,12,34,35,37, 38,62,65,72,80,84,92,102,110,111,114,117,137,165,167-169,173).

Leukocyte changes are independent of the hematocrit or temperature increase. Leukocytosis, lymphocytopenia, eosinopenia,and alteration in red blood cell life span and bone marrow function are noted. The time of onset and degree of hematopoietic change are dependent on the wave-length, field intensity and duration of exposure (72, 84, 111, 114). It is suggested that the leukocyte response is related to hypothalamic-hypophysial-adrenal stimulation (110). Lymphocytopenia and neutropenia are noted in rats exposed to 24,000 Mc, 20 mW/cm^2 for 7.5 hours (37). Kitsovskaya (84) subjected rats to 3000 Mc according to the following schedule: 10 mW/cm^2, 60 min, 216 days; 40 mW/cm^2, 15 min, 20 days; 100 mW/cm^2, 5 min, 6 days. At 40 mW/cm^2 and 100 mW/cm^2, total RBC, WBC, and absolute lymphocytes were decreased; granulocytes and reticulocytes were elevated. At 10 mW/cm^2, total WBC, and absolute lymphocytes decreased, and granulocytes increased. Bone marrow examination revealed erythroid hyperplasia at the higher power levels.

In mice irradiated with 10,000 Mc at 450 mW/cm^2 for 5 minutes, decrease in erythrocyte, leukocyte, and hemoglobin values were noted immediately, and at 1 and 5 days. Hematologic recovery was evident 10 days after exposure. Convectional heat produced less distinct changes, with more rapid recovery than exposure to microwaves (62).

In surveys of military and industrial radar personnel, minimal to significant hematologic changes are reported (8,9,12,28,65,92,102,117,165). Reticulocytosis is noted in some studies (92, 165). Reticulocytosis has also been found in dogs exposed to 2800 or 1280 Mc (111, 114).

Early and sustained leukocytosis in dogs exposed to microwaves may be related to stimulation of the hematopoietic system or recirculation of sequestered cells. Eosinopenia and transient lymphocytopenia with rebound or overcompensation when accompanied by neutrophilia may indicate increased adrenal function.

Erythrokinetic studies in dogs suggest that microwaves produce effects on the bone marrow which could be of clinical significance (114). The increase in disappearance rate of Chromium ^{51} tagged red cells after exposure, with gradual return to the normal, suggests a hemolytic process. Partial sequestration of red cells with gradual recycling may constitute a factor in this change. The hematologic and ferro-erythrokinetic data in dogs exposed to 50 mW/cm^2 indicate some depression of the bone marrow which slowly returns to normal. The depressed iron turnover rate after 100 mW/cm^2 exposure implies depression of the total erythropoietic acitvity of the bone marrow. The reticulocytosis and increased effective utilization of Fe^{59} in red cell production suggest, however, that the productive capacity is relatively more

efficient.

3. Endocrine response

Evidence of a microwave effect on the testes is indicated in several studies (44,60,63,74,158). Reports of infertility or sterility in man as a result of exposure to microwaves are questionable.

Ely and Goldman (44) irradiated the testes of dogs with 2880 Mc and found that the testicular temperature during irradiation varied from 36° C to 44° C. This temperature was maintained in most cases for 60 minutes. They conclude that the response of the testes to heating from a radar source is similar to that from other sources. The sensitivity of the testes is due to their physical location relative to the body surface, and a poor ability to dissipate heat. Heating of the testes results in tubular injury directly related to total temperature elevation. The limit for the "steady state" dose rate that produces testicular injury in experimental animals is 10 mW/cm^2 at 2800 Mc. It should be noted that determination of temperature threshold for testicular damage is difficult because of the many variables such as duration and type of exposure, CW or pulsed, that affect interpretation of findings.

Gunn et al (63) exposed the testes of rats to 24,000 Mc at 250 mW/cm^2, for 5, 10, or 15 minutes. Examination on the 6th, 13th, and 29th days revealed injury related to the duration of exposure. A five minute exposure resulted in edema and enlargement of the testes. Other changes were atrophy, fibrosis, and coagulation necrosis of seminiferous tubules. Repair was apparent 13 days after exposure. A pituitary effect is postulated. Infrared radiation that produces the same temperature rise (41°C) as microwaves, does not cause comparable injury.

Comparison of microwave (2450 Mc) and infrared radiation on the testes of rats was investigated by Imig et al (74). For similar injurious effects, a higher tissue temperature is required for infrared radiation exposure than for microwaves.

Seguin (158) suggests that irradiation of the testes of rats with 24,000 Mc microwaves, to a temperature of 41°C, results in endocrine disorders without visible testicular change; infrared radiation producing the same increase in temperature causes no endocrinopathy.

Exposure of 2-3 month old mice to 10,000 Mc, 400 mW/cm^2 for 5 minutes, causes a decrease in the number of estral cycles with increase in duration of individual cycle stages (61). One month after exposure, the estral pattern is re-established. Mating of normal females and microwave irradiated males results in a decrease in number of progeny, lower average weight of offspring and increase in number of stillborn (60). When mated with normals, microwave treated females produce weaker offspring than do similarly treated males. Deformed offspring are observed only in the females. Histologic studies reveal cumulative dystrophic changes in the germinal epithelium. In the ovaries, follicular epithelial cells are dystrophic with pyknotic nuclei. Comparison of changes in testis and ovary, indicate the female gonads to be

more sensitive. Similar but less pronounced changes result from convectional heating. Decreased breeding efficiency is noted in rabbits exposed to 2450 Mc diathermy (105).

Thyroid gland enlargement and increased I-131 uptake are reported in microwave workers (40,42,147,164). Alteration in thyroid function is noted in dogs after exposure to 1280 Mc and 2800 Mc (113). The changes suggest increased thyroid activity presumably due to increased thyroid stimulating hormone (TSH) from the hypophysis. This is consistent with microwave induced stimulation of hypothalamic-hypophysial activity (110).

Adrenocortical changes, increased blood sugar, and alteration in creatinine, lactic, pyruvic, and blood ascorbic acid levels are reported in animals and man exposed in the 2000-3000 Mc range (10, 89).

Reduced serum cholinesterase activity in the liver, heart, and brain stem of rabbits and rats is reported (122). The degree of reduction, percent incidence and time of onset are wave-length dependent. Decimeter (dm) and centimeter (cm) waves produce the most marked effect.

Observed alteration in growth rate (60, 122, 143) may reflect an endocrine imbalance. Exposure of mice to 20 mW/cm^2 and 60 mW/cm^2, 10,000 Mc causes increased growth rate after initial suppression. Repeated exposure to 3000 Mc results in a 4-6% increase in growth rate in young animals (121). Rats given repeated 2450 Mc exposures gain weight more rapidly than do controls (143). Increased protein synthesis after exposure is reported (118). Increase in basal metabolic rate is noted when the head of cats are exposed to UHF microwaves (180).

4. Immunity

Microwave irradiated animals either take up antigen more slowly, form antibodies more slowly after absorbing antigen, or form antibodies that are metabolized or eliminated more slowly than in controls. Passively transferred antibodies are also more rapidly metabolized in microwave exposed rabbits than in controls (70). 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.

C_x reactive protein is found in rabbits exposed to microwaves in the UHF range, (300-3000 Mc) at a field intensity of 10 mW/cm^2 (25). Increase in serum histamine level and total protein, and a decrease in the albumin globulin ratio are noted (52, 137).

The effects of high frequency radar waves on phagocytic activity of reticuloendothelial cells in mice are reported. A period of increased activity is followed by depression of phagocytosis during chronic exposure over a 20 day period (132).

5. Cardiovascular effects

Several investigators report that exposure of animals or man may result in direct or indirect effects on the cardiovascular system (3,40,41,48, 55,58,87,91,122,124,126,127,129,132,138,140,141,143,166,177,181). Some authors suggest that exposure to microwaves at intensities that do not produce appreciable thermal effect may lead to functional changes. These changes are observed with acute as well as chronic exposures (55,58,140).

In rats exposed to 70 mW/cm^2, 2450 Mc, right auricular pressure is increased from 2 to 10 minutes, then decreases precipitously before death at 15 minutes. Respiration becomes labored 7 minutes after the exposure is started; dyspnea develops prior to death (143).

Decreased arterial pressure and bradycardia were noted in rabbits during microwave irradiation (3). In rabbits and dogs whole-body irradiation (100-200 mW/cm^2) causes brief constriction and subsequent dilation; especially in the veins of the pia mater (166).

Dogs exhibit slowing of heart rate with alteration in the electrocardiogram during UHF irradiation. More marked and persistent EKG changes are seen in dogs with experimentally induced myocardial infarction than in normal dogs when exposed to microwaves (181). Changes in the heart rate, coagulation time, and a fall in blood pressure occurs in dogs exposed for 30 minutes at 5 mW/cm^2 UHF microwaves (140).

Functional damage to the cardiovascular system indicated by hypotonus, bradycardia, delayed auricular and ventricular conductivity, and decreased height of EKG waves in persons that work in rf fields is reported (40, 124,126,127). The changes do not diminish work capacity, and are reversible.

Wakim, et al (187) report irradiation of the thigh of healthy persons with 12 cm waves for 5 to 30 minutes and note acceleration in blood circulation, and a gradual increase in temperature in all layers of tissue. The increase in blood circulation becomes more marked as the exposure time is lengthened, and the irradiation intensified.

Tolgskaya and Gordon (177) suggest that receptors of the reflexogenic zone of the curve of the aorta, the carotid sinus,and all layers of the auricular wall are highly sensitive to microwaves. They observed morphological modification of receptors after one exposure to microwaves. These changes decreased with repeated exposures.

6. Central nervous system effects

Direct or indirect effects on the CNS or autonomic nervous system of animals and man exposed to microwaves are reported (7,34,41,43,50,51,54,56,58, 64,76,77,79-83,91,95-99,103,104,115,116,125,137-140,143,157,171,179).

One of the earliest studies on neurologic effects of microwaves is by Oldendorf (125) who found evidence of focal coagulation necrosis in the

brain of rabbits exposed to 2450 Mc. Irritability and altered equilibrium in animals during and shortly after microwave exposure are noted (34,96,143). Such symptoms are attributed to direct CNS action and are independent of hypothalamic temperature regulation.

McAfee (103) reports a nociceptive reflex in cats after 10,000 Mc, 200 mW/cm^2 focal irradiation that produces a temperature of about 45° C in the nerve fibers. The head and feet in animals and man are sensitive areas for production of this reflex which is dependent on the orientation in the microwave field and maximal stimulation of sensory nerve endings. These effects are similar to those produced by direct heating. The frequency range most likely to evoke direct stimulation is between 200 Mc to 400 Mc (185).

Neurologic effects follow head irradiation of Macaca rhesus monkeys at 225-400 Mc (7). The response depends on the orientation of the head in the microwave field and reflections from the surrounding enclosure.

When the head of pentobarbital anaesthetized dogs is exposed to 2450 Mc CW for 1-7 hours at 500 - 800 mW/cm^2, the temperature measured in the cisterna increases more rapidly and reaches a higher level than does that of other portions of brain or the rectum (157). In our studies head irradiation of anesthetized dogs results in increased rectal temperature (71).

In conditional response studies in dogs irradiated with 50 Mc to specific zones of the cerebral cortex, exposure at 7-14 W output power elicits no response; 20-25 W, causes defensive reactions and deterioration of discrimination (95).

Rats given head irradiation at 24,500 Mc tried to avoid the microwave field suggesting awareness of a stimulus (77). The most conspicuous effects are muscle spasms, tremors, and clonic convulsions. This stimulation is capable of arousing a rat from deep surgical anesthesia. In this laboratory, avoidance behaviour and clonic convulsions are noted during whole-body exposure of mice at 2800 Mc. These responses are less evident during 1240 Mc exposure.

In mice exposed to 10,000 Mc, 450 mW/cm for 5 minutes a partial decrease in conditional responses is noted up to three days after exposure; recovery occurs within one day following convectional heating (62).

Brief exposure of rabbits to 10 mW/cm^2 (VHF) intensifies conditional responses to different stimuli, whereas proionged exposure produces an inhibitory effect. Selective sensitivity of the brain to this frequency is demonstrated by reversible structural changes in the cerebral cortex and the diencephalon (80).

Organ and tissue response to UHF exposure are characterized by local cell and tissue changes. The reaction of the organism to UHF involves the nervous system. Reflex mechanisms play an important role in this respect (96).

Presman et al (140) state that chronic exposure to low intensity "non-thermal" microwave energy causes alteration in CNS function manifested by modification of inhibitions.

Frey (50,51) reports that the human auditory system can respond to at least a portion of the rf spectrum (200-3000 Mc). The response is instantaneous and occurs at a power level as low as 400 mW/cm^2. Subjects report perception of a buzzing or knocking sound. Deaf subjects perceive the sound as readily as those with normal hearing. Frey concludes that this perception is not due to acoustic energy external to the tympanic membrane, but represents an effect on the auditory nerve.

Vogelman (personal communication) notes, "Significant though extremely inefficient rectification of microwave energy has been noted by me in various junctions of partially conducting materials (the most notorious being solder-flux and metal). If we accept this as possible in vivo even with extremely poor efficiencies, we can explain some of the "non-thermal", neurologically related observations, particularly where we hear the PRF."

Individuals chronically exposed in rf fields have altered EEG patterns (43).

Morphological and histochemical studies of the nervous system show changes in cellular protein metabolism following 3,000-30,000 Mc exposures at a "non-thermal" intensity (10 mW/cm^2) (123,179).

Kamenskiy (76) finds, using comparable temperature rise as an index, a greater increase in excitation from pulsed microwaves than from CW or convectional heat. He suggests that the effect of pulsed microwaves can be considered non-thermal (specific) whereas changes observed during CW radiation might be attributed to a thermal effect. Tanner (171) finds 16,000 Mc exposure at a "non-thermal" level to cause neuromuscular responses in birds. Neuromuscular effects are also noted in chicks exposed at 24,000 Mc (38).

McAfee (104) shows the physiological effects of 3 and 12cm microwaves in dogs, cats, rabbits, and rats to be duplicated by heating peripheral nerves with a warm water or resistance wire thermode. The physiological effects occur independently of a significant increase in skin temperature or of total body heating.

Several Soviet investigators state that the central nervous system is the most sensitive of all body systems to microwaves at intensities below thermal thresholds. The reaction of the nervous system to microwaves is due to the spontaneous effect of the energy on skin receptors or brain cells. Receptor cells react to external stimuli while brain cells react to specific coded signals transmitted by receptor cells. The response is dependent on wavelength. Direct effects on the brain are intensified with increase in wavelength. Reactions attributed to skin receptor stimulation increase as the wavelength is decreased. When the reactions are due to a combination of peripheral and CNS stimulation, it is impossible to correlate the degree of reaction with wavelength (56,139).

D. Interaction with Ionizing Radiation

The stress of microwave exposure is useful in demonstrating the latent or residual effects of ionizing radiation. Increased susceptibility to microwave induced hyperthermia, indicates a lowered threshold to the stress of microwaves and/or inefficient physiologic function in X-irradiated animals (109). Dogs subjected to microwaves (whole-body) show significant increase in survival time when exposed to 25,000 R, 1000 kVp X-rays to the head. Mortality after whole-body irradiation with 1000 kVp X-rays is substantially lower among microwave pretreated dogs. Simultaneous exposure to pulsed 2800 Mc, 100 mW/cm^2, and 250 kVp X-rays, 720 R (2 R/min.) results in earlier leukocyte recovery from the radiation induced leukopenia. Such enhanced recovery is not observed at the higher rate of 4.6 R/min.(112,175). In rats that received 2500 Mc CW, 10 mW/cm^2, 30 minute exposures for 25 days, mortality from subsequent gamma-irradiation (600 R) is one-third that in the controls; irradiation with pulsed microwaves under similar conditions produces no effect (142). A reduction in X-irradiation lethality in rats and mice previously subjected to 2450 Mc CW, as well as in mice exposed to 2800 Mc pulsed is noted (174). Microwaves (UHF) influence leukopoiesis and thrombopoiesis in rabbits previously subjected to whole-body X-irradiation (184).

Microwave modification of response to X-irradiation is related to the duration and sequence of the microwave exposures as well as the time interval between the microwave exposure and the X-irradiation. Microwave exposure during the acute post X-irradiation period enhances the effect of X-irradiation (176). Cavallero (24) reports a similar finding. Except for increase in survival time and less severe neurologic response in microwave treated dogs receiving X-irradiation to the head, modification of the X-ray injury has most demonstrable effect on the hematopoietic system. Other potential microwave effects such as hypoxia, molecular alteration, thermogenesis, and stress could modify this sensitivity to X-rays. Livshits (96) interprets the reaction of the nervous system to UHF and ionizing radiation as similar.

A possible mechanism, may be that hypoxia reduces the formation and toxic effect of free radicals. Any molecular alteration may modify target molecules. Thermogenesis and stress may influence metabolism and endocrine response. The severe injury from X-irradiation at lethal dose levels, may exceed or nullify any modifying capability of microwaves.

The potential of microwaves to increase or reduce the injurious effects of X-irradiation could have application in space operations.

E. Hazard Evaluation

After a comprehensive review of the literature on microwave effects it becomes apparent that promulgation of safety standards is still extremely difficult. The rapid improvement and expansion in microwave technology and use, will most likely increase rather than decrease exposure risks.

The biologic effects of microwaves are related to microwave frequency, field intensity, and duration of exposure. Other important factors are

environmental temperature, the influence of any medication on the subject, and mode of exposure (pulsed or CW).

In comparative studies on dogs at 1280 and 2800 Mc, frequency dependent biologic responses are revealed. Exposures at 1280 Mc result in greater and more sustained leukocyte response than is noted from 2800 Mc (111). Lymphocytosis is observed for several months after 1280 Mc irradiation. Indications of a hematopoietic effect, reticulocytosis, decrease in erythrocytes and hemoglobin have been substantiated by others (84,92,102,165).

Changes are observed from 1280 Mc at 20 mW/cm^2, in the absence of increased rectal temperature; at 50 mW/cm^2 changes are more pronounced (111). Data on long term chronic exposure at 20 mW/cm^2 or less are inadequate.

The occasional reports of headache, lassitude, stomach pains, sleeplessness, irritability, and other highly subjective symptoms among workers in the vicinity of microwave generating equipment (9,42,98,100,102,117,137,147) have not been thoroughly investigated. These findings should not be ignored as similar vague, mild, and undefined symptoms have been experienced in the course of microwave studies in this laboratory. Such symptoms could indicate a basic microwave effect.

Lens changes can be induced by microwaves. Alteration in metabolic processes, and cataract formation result from irradiation of the eye. Rabbit eyes are more susceptible than dog eyes to microwave induced lenticular changes.

The heating effects of microwaves can impair testicular function, but the limits of permanent damage are not known.

Cutaneous burns can result from exposure at 2800 Mc, 100 to 165 mW/cm^2.

Soviet investigators suggest that chronic irradiation of animals with low intensity microwaves (<15 mW/cm^2) may induce functional changes in the nervous and cardiovascular systems without a rise in tissue temperature. The changes are observed both in chronic and in single exposures. Higher field intensities produce a thermal effect that can damage brain and testes, cause internal hemorrhage, burns, necrosis of tissues, and produce lenticular opacities. A Soviet report describes the effect of microwaves on humans over a 6 year period. Vascular hypotonia and bradycardia were noted in 800 persons exposed to dm-mm microwaves of "nonthermongenic intensities" (≤ 10 mW/cm^2). The most pronounced changes were observed in subjects exposed to mm waves. Most of the functional disturbances were reversible after several weeks to months (137). General weakness, diminished work capacity, increased irritability, headaches, dizziness, unpleasant sensations in the heart region, and functional cardiac changes were noted in persons chronically exposed to rf (102). In individuals working under conditions of chronic rf exposure in a hot climate, asthenia and autonomic disruptions tended to be increased; the EKG indicated sinus arrhythmia with bradycardia. Diencephalic changes were observed only in individuals working in the hot climate (100).

Synergism of high temperature and rf reduces heat tolerance of the

organism. Data of Tolgskaya and Gordon (179) in which injury of the hypothalamic region is detected under the influence of "nonthermogenic" microwave radiation in the 3000 Mc range indicate that microwaves at "non-thermal" intensities disturb processes of thermal regulation.

The reaction of animals to heating from a radiant source or microwaves suggests that awareness of heating is relative to the ambient temperature. We have found from personal exposure at 2800 Mc that there is immediate and acute perception of microwave energy at normal room temperatures. At environmental temperatures in excess of normal body temperature perception decreases. As a synergistic effect results from microwave exposure in a hot environment, and microwave injury is related to induction of thermal energy, the ambient temperature is important in the promulgation of safe exposure levels.

To our knowledge there are no substantiated reports of death from exposure to microwaves under normal conditions of operation. Death of a man from radar exposure has been reported (107). Qualified opinion indicates no relationship between the death of this individual and microwave exposure (85).

F. Comment on Soviet Investigations

Soviet and U.S. interest in the biologic effects of microwaves has increased since 1950. Although some Soviet investigators emphasize the thermal nature of the biologic effects of microwaves, several report "non-thermal" and specific microwave effects at the molecular and cellular level (39). It is suggested that microwave exposure at a field intensity which does not produce any appreciable thermal effect may cause functional disturbances of the nervous and cardiovascular systems (41,42,55,137).

Studies performed in the U.S. generally reflect the physiologic response of the organism to the thermal burden imposed by microwaves.

Pressman (137) believes that the stress stimulus from microwaves comes not only from the thermal receptors of the skin, but also from other sensory skin receptors. Microwave irradiation results in a more intense flow of afferent impulses and more intense stimulation of hypothalmic-hypophysial activity than does thermal irradiation at equivalent energy levels. The apparent adaptation to repeated microwave exposures is referable to stimulation of skin receptors.

It should be emphasized that Soviet studies are oriented towards the Pavlovian concept of "nervism". This concept presumes that all body responses are influenced by the central nervous system. Experimental support for this approach is obtained by conditional response studies. Experimental studies that rely on conditional responses are subject to criticism because of limited statistical anaylsis of data, inadequate controls, and difficulty in objective interpretation of the findings. Although the concept of "nervism" may cloud many experimental or human survey findings, the Soviet position on the biologic effects of microwaves should not be overlooked. For details of Soviet studies, the survey by Dodge (39) is recommended.

G. Maximum Permissible Exposure

To establish maximum permissible levels for human exposure to microwaves the necessary concepts and criteria which apply to the entire spectrum of microwave radiations must be developed. The effects of microwaves appear to be more complex than those simply due only to heating. The effects are dependent on frequency, power level, pulse relationship, and wave form characteristics. The biologic response is related to the distribution and absorption of the energy which in turn is influenced by the anatomical configuration of the irradiated subject, duration of exposure, air circulation rate, ambient temperature and humidity. The response of the subject is the result of the integration of all these factors as well as the physiologic status of the individual.

The decision as to how much risk is acceptable is a difficult one and depends on many factors: biological, engineering and operational. This requires biological and medical studies of the specific effects, engineering studies of the behavior of the equipment, field surveys of the power levels actually encountered,and development work on means of protection where necessary.

For significant biological effects to occur, the size of the body must be equal to or greater than 0.1λ (wavelength). At high frequencies, small organs become significant in terms of relative size. High frequencies (>3000 Mc), approach the infrared spectra where surface heating provides a warning mechanism. A considerable portion of this incident energy is reflected by the skin. Between 1,000 Mc and 3,000 Mc, varying degrees of penetration and energy absorption occur, depending on the frequency of the energy and dielectric properties of the body. Below 1,000 Mc, approximately 40% of the incident energy is absorbed primarily in deep tissue and converted into heat with little warning, as the thermal receptors are located in dermal tissue.

During the past 15 years several suggestions for maximum permissible exposure (MPE) have been made (45,153,182,186). The U.S. Armed Forces have accepted 10 mW/cm^2 for all frequencies without specifying duration of exposure. Schwan and Li (152) have suggested frequency-dependent MPE: 30 mW/cm^2 for frequencies below 1000 Mc, 10 mW/cm^2 for frequencies between 1000 and 3000 Mc and 20 mW/cm^2 for frequencies greater than 3000 Mc. Ely and Goldman (45) have designated critical organs in consideration of MPE: 150 mW/cm^2 for the eyes and 5 mW/cm^2 as the threshold for testicular damage at 3000 Mc. For frequencies greater than 1000 Mc some electronics industries have suggested 1 mW/cm^2 as the MPE (186).

These recommendations are subject to criticism because microwave frequency and time factors are not incorporated. No attempt has been made to equate power levels in terms of the duration of exposure, ambient temperature or the influence of orientation of the subject in the electromagnetic field.

In the USSR the maximum levels of high-frequency radiation permitted for radar personnel are: a) not more than 0.01 mW/cm^2 over an entire workday, b) not more than 0.1 mW/cm^2 for more than two hours during a work day

(protective glasses required), c) not more than 1.0 mW/cm^2 for not more than 15-20 minutes during a working day (protective glasses required) (54,88,136, 137).

The United States of America Standards Institute recommended in 1966, "For normal environmental conditions and for incident electromagnetic energy of frequencies from 10 Mc/sec to 100,000 Mc/sec the radiation protection guide is 10 mW/cm^2 as averaged over any possible 0.1 hour period. This means the following:

Power Density: 10 mW/cm^2 for periods of 0.1 hour or more

Energy Density: 1 mWh/cm^2 (milliwatthour per square centimeter) during any 0.1 hour period

This guide applies whether the radiation is continuous or intermittent.

These recommendations are qualified as follows:

Body temperature depends in part on sources of heat input such as electromagnetic radiation, physical labor and high ambient temperature and on heat dissipation capability as affected by clothing, humidity, etc. People who suffer from circulatory difficulties and certain other ailments are more vulnerable. It is also known that details of anatomy, the frequency of radiation, and its penetration affect the percentage of absorbed radiation. Hence, the power levels established by the radiation protection guide numbers are related in a complicated way to power levels at which damage occurs. The guide numbers are appropriate for moderate environments. Under conditions of moderate to severe heat stress the guide number given should be appropriately reduced. Under conditions of intense cold, higher guide numbers may also be appropriate after careful consideration is given to the individual situation. These formulated recommendations pertain to both whole-body irradiation and partial-body irradiation. Partial-body irradiation must be included since it has been shown that some parts of the human body (e.g. eyes, testes) may be harmed if exposed to incident radiation levels significantly in excess of the recommended levels."

5. CONCLUSIONS AND RECOMMENDATIONS

Sufficient factual data are not available to establish a comprehensive safe level for microwave exposure because of factors related to microwave frequency which influence the biologic response to this energy.

Considerable progress has been made in characterizing the overt effects of microwaves, 2800 Mc/sec (165 mW/cm^2 and 100 mW/cm^2), and 1280 Mc/sec (100 mW/cm^2, 50 mW/cm^2 and 20 mW/cm^2). The described effects are for the most part inseparable from responses which could result from thermal stress. Certain findings, such as a possible effect on bone marrow and the thyroid must be carefully evaluated because of their subtle nature and questionable relationship to thermal effects. Soviet reports allude to asthenic reactions, cardiovascular and thyroidal changes from radar exposure. Such findings should be

experimentally verified and evaluated under defined conditions of operation. Modification of X-ray effects in experimental animals has been reported by us and others. The significance of these responses should be elucidated.

Further knowledge of microwave hazards especially those of a subacute nature is required to establish safety regulations. The changes noted in our studies and those of other investigators suggest areas for further exploration. The possible introduction in the near future of more powerful radar units is cause for additional concern. Caution should be observed and specific controls adopted as dictated by the type of radar set used. The lack of definitive information on the existence of non-thermal effects might be attributed to the absence of appropriate detection and monitoring techniques.

The development of standardized detection and monitoring equipment and techniques for the entire spectrum of microwave energies for use in personnel exposure, field and laboratory measurements forms a critical need. This should be given the highest of priorities in future planning. Only in this way can definitive interpretation and comparisons of microwave field exposures and biological effect be made. Consideration must be given not only to the physical characteristics of the energy but also the interaction of the physiologic and anatomic makeup of the individual, and external factors such as temperature, humidity and air currents.

The present "safe" level of 10 mW/cm^2 for whole-body exposure, which is based on average power density for all frequencies, has remained unchanged since its adoption. The fact that effects can occur from 1280 Mc/sec, 20 mW/cm^2 exposure should alert us that 10 mW/cm^2 may not be safe for all conditions of radar operation. High peak power can be associated with low field intensities. Thus, it should be noted, that the 10 mW/cm^2 permissible exposure level does not take into consideration peak power associated with a given average power, the pulse repetition rate, ambient temperature and humidity, or the physiologic state of the individual. The part these parameters play in the biologic effect of microwaves need to be more fully investigated.

Although exposure to low intensities over prolonged periods has not produced any overt effects, the development and use of radars with many times the present power capabilities may change our concept of the biological potentials of this energy. In the meantime, we should not be complacent. For the present and near future any attempt to raise the present MPE of 10 mW/cm^2 should be approached with extreme caution.

Until more information is available on the effects of long term, low intensity (<20 mW/cm^2) exposure, we would not recommend increasing the MPE of 10 mW/cm^2.

Analysis of all data would indicate that the 1000 Mc/sec - 1500 Mc/sec range is the most hazardous. At such frequencies biological absorption is maximal and cutaneous sensibility reduced particularly with lower power levels. It is suggested that if such frequencies are operational, monitoring must be carried out within this range.

The following general rules should be observed to prevent unnecessary exposure to microwaves:

(a) The supervisory staff should be responsible for mapping out areas of unsafe power densities, i.e., above 10 mW/cm^2, either by means of power density meters or by calculation. Appropriate warning signs should be posted.

(b) Personnel should be prohibited from working in the field of radiation of any energised antenna, wave-guide, feed-horn structure, or transmission line where the power density is unsafe. Ingress into hazardous areas by personnel should be permitted only under emergency conditions; exposure in these areas should be kept to an absolute minimum, commensurate with proper maintenance and operating procedures.

(c) The practice of discharging rf output from high-power generators into the surrounding area should be discouraged. Dummy-loads, water-loads or absorbent material should be used to reduce the energy output of such equipment during testing.

(d) Where test or operational procedures require free-space radiation, the antenna should be positioned to direct the radiating beam away from residential or working areas.

All microwave workers should undergo a thorough pre-employment medical examination. The eyes should be subjected to detailed examination with ophthalmoscope and slit-lamp. Because of indications of bone marrow and thyroid sensitivity to microwaves, the hematologic and thyroid status of each individual should be considered. Persons having cardiovascular, thyroid or hematologic problems or lenticular defects should not be accepted for this work. Medical examination of all radar workers should be conducted at specified intervals.

Medical complaints of persons exposed to microwaves should be directed to a central source. A detailed history of microwave exposure should be obtained to facilitate correlation of effects with exposure.

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Arlington, Virginia 22314

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Department of Commerce
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FTD - Foreign Technology Division
Translation Services Branch
Wright-Patterson AFB
Ohio 45433

JPRS - Joint Publications Research Service
Building Tempo E
Adams Drive, 4th and 6th Streets, S.W.
Washington, D.C. 20443

NASA - National Aeronautics and Space Administration
Scientific and Technical Information Facility
P.O. Box 33
College Park, Maryland 20740

RADC - Rome Air Development Center
Griffiss AFB
New York 13440

UDC - Consultants Bureau
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227 West 17th Street
New York, N.Y. 10011

Glossary

BUN - blood urea nitrogen

cm - centimeter (wavelength)

CNS - central nervous system

CO_2 - carbon dioxide

Co^{60} - radioactive isotope of cobalt

Cr^{51} - radioactive isotope of chromium

CW - continuous wave

dm - decimeter (wavelength)

DNA - desoxyribonucleic acid

EKG - electrocardiogram

Fe^{59} - radioactive isotope of iron

IR - ionizing radiation

iv - intravenous

I^{131} - radioactive isotope of iodine

kVp - kilovolt peak (energy output)

(λ) - lambda (wavelength)

LD - lethal dose

MAD - midline air dose

Mc;Mc/sec - megacycles per second

MHz - megahertz (cycles per second)

mm - millimeter (wavelength)

MPE - maximum permissible exposure

mW/cm^2 - milliwatts per square centimeter

NPN - non-protein nitrogen

R - röntgen (unit of exposure dose of X- or γ-radiation)

(R) - registered trademark

RBC - red blood cells

rf - radio frequency

RNA - ribonucleic acid

SHF - super high frequency (1 cm - 10 cm wavelength)

TSH - thyroid stimulating hormone

UHF - ultra high frequency (10 cm - 1 meter wavelength)

VHF - very high frequency (1 meter - 300 meters wavelength)

APPENDIX I

Reports Prepared Under Contract USAF - 30(602)-2248

A. Publications

Howland, J. W., R. A. E. Thomson, and S. M. Michaelson. 1961. Biomedical aspects of microwave irradiation of mammals. p. 261-284. In Biological effects of microwave radiation. Plenum Press, New York.

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13. ABSTRACT

The exact nature of the biological effects of microwaves is not completely understood. Evidence indicates that microwave energy can act as a "stressor" agent, and has an effect on regulatory and integrative mechanisms of the body with resultant alteration in homeokinesis.

Animals exposed to microwaves at specific frequencies and flux densities experience thermal stress. Duration of exposure, environmental temperature, and drugs that affect the central nervous system (CNS) and temperature regulation influence the response of animals. High ambient temperature exaggerates the thermal response, while exposure in a cold environment prolongs the time interval for an increase in body temperature. Microwave effects on hematopoiesis, thyroid function and interaction with ionizing radiation are discussed.

Sufficient data are not available to establish a comprehensive safe level for microwave exposure because of microwave frequency related factors which affect biologic response. The subtle nature of some of our findings such as modification of response to X-irradiation and effect on bone marrow and thyroid require careful evaluation. Soviet reports allude to asthenic reactions, CNS cardiovascular, and thyroid changes from radar exposure. The described effects are for the most part inseparable from responses which could result from thermal stress. Further knowledge of microwave hazards especially those of a subacute nature is needed to establish safety regulations. The changes noted in these studies and those of other investigators suggest areas for

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further exploration. Controlled conditions of operation will be essential to verify and evaluate experimental findings.

It is recommended that all microwave workers should undergo a thorough pre-employment and periodic medical examination. Persons with cardiovascular problems or lenticular defects should be considered as risks. Because of indication of bone marrow and thyroid sensitivity to microwaves, hematologic and thyroid function studies should be incorporated in the medical examination.

An increase from the present maximum permissible exposure level of 10 mw/~~cm~~^{2} sq cm could introduce risks which might not be readily recognized.

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