Biological Effects of Microwave Exposure—An Overview*

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ABSTRACT

In October, 1968 the U.S. Congress passed, and former President Johnson signed, Public Law 90-602, The Electronic Products Control Law. To carry out the provisions of this law the Bureau of Radiological Health (BRH) of the United States Public Health Service was given the responsibility of setting standards and maintaining surveillance over all electronic products that may emit hazardous levels of electromagnetic radiation. In addition to the problem of ionizing radiation, a very large area of concern involves exposure to microwaves. The exact nature of the biologic effects of microwaves is not completely understood. Although most of the experimental data support the concept that the effects of microwave exposure are primarily a response to local or general hyperthermia, there are large areas of confusion, uncertainty and actual misinformation. This paper reviews the present state of knowledge on biologic effects of microwaves and attempts to (1) differentiate between the known and substantiated from the speculative and unsubstantiated effects and (2) provide a realistic perspective on the nature of microwaves and the possible effects of exposure to this form of energy. Unless this is done, the tremendous potential of electromagnetic energy in the microwave range for Radar, communications, biomedical, industrial, and consumer use and applications will be hampered. According to the best evidence available, the most important effect of microwave absorption is the conversion of the absorbed energy into heat. Exposure of various species of animals to whole-body microwave radiation at levels of 100 mW/cm² or more is characterized by a temperature rise which is a function of the thermal regulatory processes and active adaptation of the animal. The end result is either reversible or irreversible change depending on the conditions of the irradiation and the physiologic state of the animal. The thermal response induced by microwave exposure in an animal with thermal regulatory capability comparable to that of man (such as the dog) is characterized by three phases: (a) initial thermal response, (b) period of thermal equilibrium and (c) period of thermal breakdown. In areas in which relatively little blood circulates, the temperature will rise considerably since there is little means for the interchange of heat. Consequently, tissue damage is more likely to occur in those areas where proportionately a greater rise in temperature can occur. Thus the lens of the eye and testes are readily susceptible to thermal damage, since these organs do not posses an adequate vascular system for the exchange of heat. The greatest need today in the assessment of Biological Effects of Microwave exposure is to maintain a realistic perspective on the nature of microwave fields and the

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possible effects from exposure. The mechanisms by which cell damage is produced, the biological tolerance of the most susceptible tissues and safe levels of intensity must be established in an organized fashion. Ultimately, a clear differentiation between hazard and biologic effect must be made.

With passage in October 1968 of Public Law 90-602—the "Radiation Control for Health and Safety Act of 1968"—there has been a resurgence of interest in the biologic effects of microwave exposure.

To put the question of microwave bioeffects in its proper perspective, a critical analysis of the published literature is essential to differentiate between the known and substantiated from the speculative and unsubstantiated effects. In addition, an appreciation of the nature of microwaves and the possible effects of exposure to this form of energy is required. Unless this is done, the tremendous potential of microwaves for radar, communications, biomedical, industrial, and consumer use and application will be hampered.

Although most of the experimental data support the concept that the effects of microwave exposure are primarily a response to local or general hyperthermia, there are large areas of confusion, uncertainty and actual misinformation.

The electrical properties of tissues must be known for a complete understanding of the mode of interaction of electromagnetic energy with organisms. Absorption coefficients, reflections at interfaces between different tissues, absorption of incident power, and scattering properties of biologic materials are related to certain composite features such as water content, macromolecular and lipid content (Schwan, 1957, 1958; Schwan and Piersol, 1954).

The energy content for microwaves is approximately 10^{-5} electron volts (eV) per photon in contrast to X or gamma rays, the energy of which is rated as thousands or millions of electron volts per photon. Since it takes about 34 eV to produce an ion pair, this energy level can be reached in the short wavelength ultraviolet range and with X or gamma rays. For microwaves, however, the energy value is too low to produce ionization on a single event basis.

Since microwaves do not cause ionization, those effects resulting from dissociation of chemical bonds, such as mutations, cannot be induced directly by microwaves as they can by X-rays or gamma rays. Generally speaking, the effects of microwave exposure are not associated with the disturbing effects of ionizing radiation. The effects of microwaves are a manifestation of thermal conversion. Ionizing radiation, on the other hand, causes little thermal effect (Pollard, 1969).

According to the best evidence available, the most important effect of microwave absorption is the conversion of the absorbed energy into heat. Exposure of various species of animals to whole-body microwave radiation at levels of 100 mW/cm^2 or more is characterized by a temperature rise which is a function of the thermal regulatory processes and active adaptation of the animal. The end result is either reversible or irreversible change depending on the conditions of the irradiation and the physiologic state of the animal. The thermal response induced by whole-body high-level ($>$ 100 mW/cm^2) microwave exposure in an animal such as the dog, with thermal regulatory capability comparable to that of man, is characterized by three phases: (a) initial thermal re-

sponse, (b) period of thermal equilibrium and (c) period of thermal breakdown.

In areas in which relatively little blood circulates, the temperature may rise more rapidly than in vascular parts of the body since there is little means for the interchange of heat. Consequently, tissue damage is more likely to occur in those areas where proportionately a greater rise in temperature can occur. Thus the lens of the eye may be more susceptible to thermal damage since this structure does not possess an adequate vascular system for the exchange of heat.

A great deal of controversy has arisen concerning the relative importance of thermal and non-thermal effects of microwave radiation. Thermal effects have been well demonstrated and documented but the evidence for a non-thermal effect is at best only suggestive.

Evidence presented for a non-thermal effect has generally been in one of several areas; microscopic, biochemical, cataract production, and neurological. Pearl chain formation with blood cells and bacteria has generally been held to be insignificant biologically (Kalant, 1959; Saito and Schwan, 1961). The bactericidal effect and chromosomal aberrations described by Heller and Teixiera-Pinto (1959) are probably thermal in nature (Kalant, 1959). Several metabolic defects, especially in relation to the lens of the eye, have been described (Carpenter, 1962; Daily et al, 1951; Merola and Kinoshita, 1961; Richardson et al, 1952). These alleged metabolic defects may in fact be a function of the early and transient tumescence of the lens which occurs after exposure to high power densities (> 100 mW/cm^2) rather than a biochemical alteration. The suggestion of a non-thermal effect in cataractogenesis comes from the work of Carpenter and associates (1960, 1962). He bases this conclusion on the fact that repeated "subthreshold" exposures will produce cataracts with a smaller rise in intraocular temperature than a single larger exposure with a greater rise in temperature which does not produce a cataract. He concludes that no critical intraocular temperature for cataract production nor a very appreciable temperature rise is necessary, therefore, the cataractogenic effect must be nonthermal (Carpenter et al, 1960). The possibility of cumulative damage to the lens from repeated "sub-threshold" exposures of rabbits, eyes to microwaves has also been suggested by Carpenter and associates (1959, 1960).

Most investigators agree that there is a critical intraocular temperature which must be reached before opacities develop. This temperature, as reported by various authors, ranges from 45 to 55°C. Obviously, no cumulative rise in temperature can occur if the intervals between exposures exceed the time required for the tissue to return to normal temperature. The cumulative effect to be anticipated, therefore, is the accumulation of damage resulting from repeated exposures each of which is individually capable of producing some degree of damage (Kalant, 1959).

According to Zaret (1959) Carpenter's results do not necessarily indicate a non-thermal cumulative effect. Acute injury of the lens leads first to hydration, and this is reversible providing no lens protein denaturation has taken place despite the fact that banding, striations and opacification are evident. Hydration of lens fibres may last for many days. If the excess water leaves the lens before denaturation has occurred, no permanent residua results. If other thermal injury intervenes, however, at a time when the lens is partially damaged, there

may be a summation of effect. Baillie (1969) used a hypothermic technic to investigate the postulated nonthermal mechanism for cataractogenesis from multiple microwave exposure at subthreshold levels. His data do not support the existence of a nonthermal cataractogenic property of microwave radiation. According to Baillie (1969) the cataracts which developed during the course of his study can only be explained on the basis of thermal coagulation of lens protein. There is therefore, adequate evidence to incriminate heat as the initiating mechanism leading to cataract formation during or following a single exposure to microwave radiation. This study suggests that microwave cataractogenesis is, directly or indirectly, a thermal phenomenon. At subthreshold power levels, there is still some question regarding the cumulative effects on the lens. Differences in patterns of peak pulse levels and off time between pulses may be critical factors (Roth, 1968).

It should be understood that a cumulative effect is the accumulation of damage resulting from repeated exposures each of which is individually capable of producing some degree of damage. Since this has not been conclusively shown, the suggestion of cumulative effects from microwave exposure is untenable.

It is important at this point to define the cumulative effect produced by ionizing radiation to put this question in its proper perspective. It has been suggested (and there are some experimental data to support the concept) that injury incurred from exposure to ionizing radiation is cumulative. This cumulative effect is a manifestation of the *irrepairability of a certain fraction of ionizing radiation injury* which has been designated as *Residual Radiation Injury*. This component of Residual Radiation Injury is additive with frequency of exposures and is not dependent on intervals between exposures once the full recovery potential has been realized (Blair, 1964).

A number of retrospective studies have been done on human populations exposed to microwave energy. These have been, for the most part, either radar operators and repairmen or personnel involved in production and testing of microwave equipment, primarily radar. The studies may be divided into essentially 2 categories; those seeking general effects, and those specifically seeking changes in the lens of the eye. Barron and Baraff, in 1958, studied 335 microwave exposed workers and compared them with a control population. No differences were found in physical inventories of the two groups nor were any differences in death, disease, sick leave, or subjective complaints found. No blood or urine changes and no increase in ocular pathology of the microwave exposed group was noted. Both groups were equally fertile as determined by reproductive history.

A number of effects in man have been described by Soviet workers. These reports have been reviewed by Dodge (1965, 1970) and Turner (1962) and analyzed by Michaelson and Dodge (1971). Most of the reported effects are subjective, consisting of fatigability, headache, sleepiness, irritability, loss of appetite, and memory difficulties. Cardiovascular effects consist of lability of the pulse and blood pressure, heart enlargement and murmurs, and ECG changes. Increased I^{131} uptake by the thyroid, changes in serum proteins, decrease in olfactory sensation, falling hair, and disruption of sexual potency have also been reported. Cataracts have been observed, although not to the extent that has been reported in the United States. Other reports describe psychic

changes including unstable mood, hypochondriasis, and anxiety. The nervous and cardiovascular disruptions are noted to be benign and do not lead to loss of work capacity.

On the basis of these reports the Soviets have set their exposure standard at 0.01 mW/cm² for an entire work day in contrast to the U.S. limit of 10 mW/cm². Pazderova (1968), in Czechoslovakia, has reviewed the Soviet bloc and Western literature in this field and has pointed out that apparent discrepancies perhaps are not so great as would appear. She points out that the Soviet literature presents very little data and cannot be statistically analyzed; that the Soviet work is based a great deal on subjective rather than objective findings; and that dosimetry in both cases is rather poor and not comparable from worker to worker. She states that "In order to judge the significance of the occupational hazard of electromagnetic radiation more accurately, it will be necessary to correlate medical findings obtained from long-term observations of workers exposed to electromagnetic radiation with extent of exposure".

Surveys have been performed in the U.S. of lenticular opacities among microwave workers to assess the significance of possible lenticular changes. Zaret and Eisenbud (1961) reported no late lens defects peculiar to microwave exposure. They did, however, note a statistically significant increase in posterior polar defects. Zaret et al (1961) reported a study of 475 exposed personnel and 359 controls in which a slight, but statistically significant increase in lenticular defects were noted in the exposed group. These consisted of posterior polar defects, opacification, minute defects, and relucency. In 1966, Cleary and Pasternack reported a study of 736 microwave workers and 559 controls. They too reported a statistically significant increase in certain types of lens defects. They suggest that this may represent an aging effect and note that there is no relationship to loss of visual acuity or cataract production (Cleary and Pasternack, 1966). Majewska (1968) studied 200 Polish microwave workers and 200 controls. He too noted a statistically significant increase in lenticular defects. Again an aging effect is suggested.

While all of these reports have found a statistically significant increase in lenticular defects in microwave workers, none has reported any clinically significant defects in terms of decreased visual acuity. The scoring methods used both for degree of exposure and lenticular defects in all cases is not particularly sound and their validity may be questionable.

In 1957 McLaughlin reported "Tissue Destruction and Death from Microwave Radiation (Radar)" (McLaughlin, 1957). This is the only case of death reported in association with microwave exposure, and it is doubtful whether the microwave exposure had a significant effect, if any, in causing the death. It was, in fact, a case of acute appendicitis in which evisceration of the wound occurred on the tenth post-operative day leading to profound shock and death. Kanuf (1958) and Kalant (1959) both note that no other deaths from microwave exposure had ever been recorded and that deaths due to appendicitis and shock are not uncommon. As noted by Ely (1971), this case was thoroughly discounted in an Armed Forces Institute of Pathology Memorandum of July 25, 1957.

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fects from exposure. The mechanisms by which cell damage is produced, the biological tolerance of the most susceptible tissues and safe levels of intensity must be established in an organized fashion. Ultimately, a clear differentiation between hazard and biologic effect must be made.

The only effect of microwave energy absorption which is of pathophysiologic consequence is the dielectric heating which could present a thermal hazard to the body. Safety from this hazard is assured by the proper observance of the American National Standards Institute (ANSI) C95.1 Standard (1966) which specifies a maximum exposure level of 10 mW/cm^2 under normal environmental conditions averaged over any possible 0.1 hour. This has been and continues to be the generally accepted safety standard in the Western world for individuals in microwave radiation fields.

The ANSI C95.1 standard of 10 mW/cm^2 is roughly a factor of ten below thresholds of damage by thermal effects, assuming a long duration of exposure—i.e., one quarter hour or more. The 10 mW/cm^2 level is based on thermal equilibrium conditions for whole-body radiation. Temperature rise is determined primarily by the body's ability to dissipate heat; factors affecting this would be significant in terms of the consequences of whole-body irradiation. Heat dissipation capabilities are better for partial-body radiation; higher levels of irradiation would therefore be acceptable. This is the case in medical diathermy, where the levels may be at 100 mW/cm^2 or higher.

The value of 10 mW/cm^2 has been generally accepted throughout industry and the armed services without qualification with regard to frequency, pulsed or continuous transmission, partial or whole-body exposure. The British adopted the 10mW/cm^2 level for the general public as well as the military and industry after careful consideration by many government and independent organizations (BPO, 1960). Sweden, in 1961, after an extensive review of all the information available recommended "the maximum permissible intensity (average irradiation) within areas where personnel are occasionally to be found is 10 mW/cm^2 for all occurring frequencies" (Clemedson, 1961). The Federal Republic of Germany, France, and M. V. Philips in the Netherlands have also established a 10 mW/cm^2 as a maximum safe level (Körner, 1967, Swanson et al, 1969).

While the limit of 10 mW/cm^2 served as a practical exposure level in the military in the U.S. for several years, it was felt that the duration of exposure was important, and that higher levels could be tolerated for shorter periods. Applying toxicological criteria (i.e., the duration of exposure to a toxic agent multiplied by concentration of that agent during exposure represents the hazard), new guidlines were developed and published as an Army-Air Force Manual in 1965 (AFM 161-7, 1965). In this document, exposures of personnel within limited occupancy areas is permitted only for the length of time given by the following equation:

$$Tp = \frac{6000}{w^2}$$

Where: Tp = permissible time of exposure in minutes during any 1-hour period.

w = power density in area to be occupied in mW/cm^2

The equation is useful only for power densities up to 100 mw/cm^2, and because exposures of less than 2 minutes are operationally impractical, its use for power densities above 55 mW/cm^2 was not recommended.

It is often said, usually informally, that the exposure level limit of 10 mW/cm² was little more than a guess on the part of early investigators, was founded on scanty research evidence, and must be scrapped for a much lower level in light of "new" questions being raised on effects of RF radiation. The Standard originally set in 1957 and later established by ANSI C95.1 in 1966 was based on very sound experimental evidence gathered by military and civilian scientists. The latter group included many now recognized as international authorities on the subject. Thus, the limit of 10 mW/cm² cannot be challenged on the premise that its formulation had no basis in fact, or was the arbitrary decision of individuals having no insight into the problem. And the current questions being asked on effects of microwave radiation are really not "new" (Odland, 1970).

In November 1956 a report issued by the Air Research and Development Command (ARDC) Microwave Panel observed "there is no substantial evidence of injury having resulted from accidental exposure to r-f radiation under either field or laboratory conditions" (AF, 1956). To the present day, there is no documented evidence of injury to military personnel from the operation and maintenance of Radars within the limits of the microwave exposure standard. The guidelines and exposure levels published in 1965 are in force today, and appear to be entirely safe.

The question of microwave hazard evaluation presents a very considerable problem. There are many areas in which presently available data are questionable, contradictory, or inapplicable. Many of the present deficiencies in microwave hazards investigations must be eliminated before good sound data will be available on which to base precise microwave exposure standards. The present U.S. standard of 10 mW/cm² has been in effect for about 15 years. The present Soviet maximum permissible exposure is 0.01 mW/cm² for a 6 hour work day which, however, can be raised to 1 mW/cm² for a 15-20 minute exposure during a work day. It is questionable whether the Soviets can, in fact, function within this limit which was reached, to a great extent, on the basis of psychological and neurasthenic responses in exposed personnel which have not been observed in this country. In comparing the Western and Soviet standards, note should be taken that the Soviets do permit an increase to 1 mW/cm² for 15-20 minutes. Also similarities and differences in distance from the microwave emitting source and measuring techniques, as well as differentiation between personnel exposure and product performance standards have to be considered.

There are presently groups in this country seeking changes in the MPE, some seeking to lower the present limit and others seeking to raise it. There is not now in the Western literature any evidence of injury occurring at the 10 mW/cm² level. Neither is there evidence to demonstrate that there is no long-term, low-dose effect. To make effective any legislated limits or hazard evaluation, a precise and accurate system of dosimetry is essential. It would be fruitless to set a limit which cannot be enforced for lack of a device to measure microwave field strengths accurately and in many different situations. Likewise, hazards cannot be evaluated if they cannot be quantitated.

It is highly questionable whether any of the presently available experimental data can be validly applied to humans and used as arguments favoring lowering of the present Maximum Permissible Exposure of 10 mW/cm². Only in the coming years, with the

development of better equipment for generating, measuring, and recording microwave energy will this problem be solved.

Probably the best solution at present is to keep the 10 mW/cm^2 maximum permissible exposure rather than making arbitrary decisions with insufficient information. A greater effort should be made to obtain the data necessary to make rational and informed decisions concerning the hazards of microwave exposure.

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