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THE INFLUENCE OF RADIOFREQUENCY/MICROWAVE ENERGY ABSORPTION ON PHYSIOLOGICAL REGULATION

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Summary.—Physiological regulation represented by thermoregulation, neuroendocrine function, neurochemical activity, and immune responses is a composite of exquisitely "tuned" interrelated systems that constitute sensitive indicators of body responses to environmental stimuli or absorbed physical energies. Exposure to microwave/radiofrequency fields may affect such physiological regulation. Study of the integration and correlation of many body functions relative to the altered homoeostatic status of the microwave/radiofrequency-exposed subject is thus indicated.

Microwave-induced physiological changes cannot be dissociated from increases in tissue temperature. Such responses are considered to be essential in defence against environmental changes as a febrile response is essential for host immune defence. These responses can also be considered to reflect the utilization of physiological function to maintain regulations or adjustments. These are not necessarily adverse reactions to environmental stimuli. These responses can be transient or persistent, beneficial or detrimental.

Assessment of the integration and correlation of these functions relative to the thermal inputs and homoeokinetic reactions of the individual subjected to microwave/radiofrequency energy should permit differentiation between potential hazards which might compromise the individual's ability to maintain normal physiological function and effects which are compensated by physiological redundancy.

EXPOSURE to microwave/radiofrequency (MW/RF) fields may elicit thermoregulatory responses, neuroendocrine, neurochemical modulations and behavioural reactions. These physiological regulatory processes are exquisitely tuned, interrelated functions that constitute sensitive indicators of strains on the homoeostasis of the individual. Assessment of the integration and correlation of these physiological regulatory processes relative to the thermal inputs and homoeokinetic reactions of the individual exposed to MW/RF energy should permit differentiation between responses which might compromise the individual's ability to maintain normal physiological function and reactions which are compensated by physiological redundancy.

Physiological integration

The main integrator of mammalian regulatory systems is the brain which through the autonomic nervous system and hypothalamus mediates thermoregulatory, behavioural, neuroendocrine, cardiodynamic and immunological reactions to factors that may impose a strain on the homoeostatic mechanisms of the body.

To best describe physiological regulation we might consider principles of control theory in the regulation of body temperature. Several thermoregulatory models have been developed of which the closed-loop concept of Stolwijk & Hardy (1966, 1977) is most useful. In this model the feedback control system is divided into a regulated system (body heat capacitance) and the regulating system (regulator) (Stolwijk, 1970). The regulated system can be subjected to a disturbance from environmental heat or cold or metabolic heat. The disturbance causes a change in the controlled variables (body tempera-

ture or combination of temperatures) that are measured by thermal receptors (a transducer) which generate related neural or hormonal information. This information (the feedback) is compared with reference information. The difference between the feedback and the reference, termed the error, is a measure of the effect of the disturbance on the controlled variable. The error activates a control centre which provides a control action in such a way as to oppose the effect of the disturbance. In thermoregulation the control actions are means of modifying heat loss, heat production, or heat conservation by sweating, shivering or vasomotor activity.

Body heat content is equilibrated by balancing two overall processes, gain and loss. Heat production and heat loss are both locally and integrally controlled in a complex manner and many sensory and central factors are intricately involved. The hypothalamus is the major site of thermosensitivity in the brain. Thermoregulatory responses to both cooling and heating can be elicited in the anterior hypothalamus (Hammel, 1968). Temperature receptors are distributed over the entire body surface. In addition, certain cells deep within the brain are capable of responding directly to local changes in temperature; additional deep receptors are also located elsewhere within the core of the body (Brengelmann, 1974).

The "set point" temperature for mammals is maintained by physiological and behavioural processes that appropriately balance heat production or gain, and heat loss (Kluger, 1979). When an animal experiences a thermal burden, it will institute physiological and behavioural manoeuvres to increase heat loss, unless the stimulus had produced alteration of the set point. If the thermal load can be dissipated by the individual's physiological and behavioural responses, an increase in core body temperature may not be detectable. Alternatively, if the subject cannot completely compensate for heat gain, a temperature rise would ensue.

Integrative structures which presumably compare sensed temperature to "normal" temperature (the *set point* or *reference temperature*), determine whether existing temperature is too high or too low, and activate the appropriate motor response. Incidental changes in central temperature set in motion a sequence of events which eventually affect (or feed back upon) central temperature. A spontaneous deviation of central temperature in one direction sets into play mechanisms which tend to displace that temperature in the opposite (negative) direction; a rise in central temperature activates heat-loss mechanisms, while a fall in central temperature activates heat-conservation and heat-production mechanisms (Brengelmann, 1974).

To maintain homoeostasis, mammals also possess control mechanisms that react to multidimensional changes in the internal and external environments (stimuli or stressors). In the adult mammal, external and internal factors modify the activity of the endocrine system *via* the central nervous system by influencing the output rate of releasing hormones from the hypothalamus. Hypothalamic–hypophysial–adrenocortical (HHA), hypothalmic–hypophysial–thyroidal (HHT), hypothalmic–hypophysial–somatotrophic (HHS) prolactin and opiocortin neuroendocrine/neurotransmitter systems participate in the maintenance of homoeostasis through negative feedback mechanisms. As an example, adrenergic neurones in the hypothalamus stimulate thyrotropin-releasing hormone (TRH), which acts on the anterior pituitary to increase thyrotropin (TSH), which in turn enters the general circulation to stimulate thyroid secretion of triiodothyronine (T_3) and thyroxine (T_4). The T_3 and T_4 then act by negative feedback control on hypophysial TSH secretion.

Microwave hyperthermia and physiological regulation

The organs and organ systems susceptible to microwave/radiofrequency (MW/

RF) exposure manifest functional disturbance and/or structural alterations relative to the absorption and distribution of the energy. Some reactions to MW/RF exposure may lead to measurable biological effects which remain within the range of normal (physiological) compensation; or may improve the efficiency of certain physiological processes and thus be used for therapeutic purposes. Some reactions, on the other hand, may lead to effects which may be health hazards.

Most MW/RF responses are explained by thermal energy conversion of the absorbed electromagnetic energy. The non-uniform distribution of absorbed energy may give rise to temperature increases and rates of heating in local "hot spots" that could result in unique biological effects. Neurophysiological responses to raised local tissue temperature may be elicited by direct effects on nervous tissue functions and/or reflex effects operating through sensors or transducers of these thermal changes and then through effector mechanisms in the central nervous system (Adey, 1981). Since resonant absorption within the cranium may result in the focusing of energy and the production of electromagnetic "hot spots" in the brain (Johnson & Guy, 1972) some observed effects may relate to higher absorption in such regions as the hypothalamus than in other tissue.

The spatial distribution of electric field strength and absorbed energy in tissue spheres and phantoms has been investigated (Barber, 1977; Johnson & Guy, 1972; Kritikos & Schwan, 1976, 1979; Shapiro *et al.*, 1971; Weil, 1975). The uniqueness of the inhomogeneity of microwave-induced temperature distributions may, however, be more apparent than real since the mammalian body normally is not a uniform incubator and contains significant temperature gradients (Hardy, 1970) as well as diurnal temperature excursions.

Specific organ or tissue systems may "function" at a significantly different rate if local thermal gradients are altered. Relatively large changes in circulation can be provoked by small deviations from neutral temperature (Thauer, 1965) as in exercise- or work-related temperature increase (Saltin, 1970). The physiological significance of raising cerebral temperature by 1–2°C is not known (Adey, 1981), although general as distinct from focal increments in brain temperature up to 2°C occur in normal subjects during exercise and in moderate heat stress without evidence of major alterations in higher nervous function (Baker & Chapman, (1977).

Exposure of animals to MW/RF energy leads to body temperature elevation when the rate of energy absorption exceeds the rate of energy dissipation. Whether the resultant temperature elevation is general or confined to specific anatomical sites depends on:

(a) the electromagnetic-field characteristics and distributions within the body and
(b) the passive and active thermoregulatory mechanisms available to the individual.

Thermoregulation

It has been shown that whole-body exposure of dogs to 2880MHz pulsed microwaves at power densities of 100 mW/cm^2 (3·7 W/kg) for 6 h or 165 mW/cm^2 (6·1 W/kg) for 2–3 h resulted in physiological responses consonant with thermal stress and characterized by 3 phases: (a) initial temperature rise, (b) period of thermal equilibrium, and (c) thermal regulatory failure (Michaelson, 1974; Michaelson *et al.*, 1961, 1967). Dogs displayed greater thermal susceptibility to microwaves while under the influence of pentobarbital sodium, morphine sulphate or chlorpromazine, which suggested impairment of thermoregulatory function.

An early manifestation of microwave-induced heat loading in the dog was an increase in blood volume, which occurred during the first 30 min of exposure and before colonic temperature increased. This

suggested fluid withdrawal from the extracellular space, leading to haemodilution. With prolonged exposure, haemodilution was reversed as a result of dehydration, and haemoconcentration followed. Haemoconcentration and temperature increase were prevented by alimentation with water. In all cases the animal returned to base-line function upon cessation of exposure.

Cardiodynamic activity

An increase in heart rate was noted as a consequence of irradiation of the dog's head at 80 mW/cm^2, 2450 MHz CW (Lu *et al.*, 1974; Michaelson, 1977). Exaggerated sinus arrhythmia with increased depth of respiration, leading to bradycardia, was noted. Ventricular conduction time increased with the respiratory rate.

Paradoxical cooling of the extremities accompanied by shivering was observed during cranial exposure, while tympanic temperature increased (Kinnen *et al.*, 1975; Lu *et al.*, 1974). Such reactive skin cooling was not noted within the skin of the inguinal, axillary or back regions of the dog. The data indicated that cranially localized microwave heating can influence peripheral effectors. Changes in heart rate and dP/dt (rate of change in isovolaemic intraventricular pressure) were found to correlate with body temperature. These cardio-respiratory responses are interpreted as an early increase in sympathetic activity followed by a parasympathetic response.

Neuroendocrine function

Neuroendocrine reactions to MW/RF energy absorption may reflect: (a) hypothalamic–hypophysial stimulation that results in changes in production, secretion or utilization of trophic hormones, (b) direct neural influence on a particular endocrine gland or (c) direct effect on the central nervous system. On the other hand, functional changes of the neuroendocrine system may represent a thermal effect on a particular endocrine gland itself, or a response to a more generalized thermal stress.

Hypothalamic–hypophysial–adrenal function

Plasma corticosterone (CS) levels in rats exhibited a variable power density/threshold pattern of response, with a different threshold for 120-min exposure than for 30- or 60-min exposure to 2450 MHz (CW) (Lotz & Michaelson, 1978). A strong correlation ($r = 0{\cdot}903$) was evident between mean colonic temperature and mean plasma corticosterone levels. The threshold power density was 50 mW/cm^2 for a 30- or 60-min, and 20 mW/cm^2 for 120-min exposure. These thresholds represented whole-body specific absorption rates (SAR) of 8·0 and 3·2 W/kg, respectively. Plasma CS increased within 15–30 min of the start of exposure and fell sharply within 15–30 min after termination of exposure. Thus the adrenocortical response is transient (Michaelson *et al.*, 1977).

Plasma CS levels did not increase in hypophysectomized rats exposed to 60 mW/cm^2 (9·6 mW/g) for 60 min or pretreated with dexamethasone and exposed to 50 mW/cm^2 (8 mW/g) for 1 h. Thus microwave-induced CS response is dependent upon ACTH secretion by the pituitary, rather than direct stimulation of the adrenal gland (Lotz & Michaelson, 1979).

In rats exposed to 2450 MHz CW at 0, 1, 5, 10 or 20 mW/cm^2 for 1, 2, 4, or 8 h (Lu *et al.*, 1977*a*), power density below 10 mW/cm^2 (SAR = 1·6 mW/g) accelerated the appearance of the peak colonic temperature to an earlier time of the day. Circadian elevation of serum CS increase was significantly blunted in rats exposed to 20 mW/cm^2 (3·2 mW/g) for 8 h. This blunting of CS circadian elevation was also noted in rats exposed to 0·1 and 1 mW/cm^2 (0·016 and 0·16 mW/g) for 4 h (Lu *et al.*, 1980*a*, *b*). Thus a dual action of microwaves on the HHA axis was demonstrated, in which "low-intensity" exposure (< 10 mW/cm^2 [1·6 mW/g]) inhibited

CS increase during the peak period of the CS circadian rhythm, while higher-intensity exposures (> 25 mW/cm^2 [4 mW/g]) stimulated CS secretion during any interval of the circadian periodicity. These divergent responses suggest that the functional reaction to microwaves represents 2 different types of action, depending upon intensity, eliciting different responses relative to the timing of the exposure with respect to circadian rhythm. Such periodic sensitivities suggest a dissociation of the modes of pituitary–adrenal activation for the stress response and rhythmicity at the level of the hypothalamic corticosterone-releasing-factor (CRF) neurones.

Thyroid function

Serum thyroxine levels were transiently elevated in rats after exposure to 2·45 GHz at 1 mW/cm^2 (0·16 mW/g) for 4 h (Lu *et al* 1977*a*). This transient increase was not accompanied by changes in serum TSH (Lu *et al.*, 1977*b*). Localized thyroid exposure to 2·45 GHZ, which resulted in thyroid temperature elevation, stimulated thyroid-hormone secretion in dogs (Magin *et al.*, 1977). Stimulation of thyroid function was also noted in rats exposed whole-body to 2·45 GHz, 70 mW/cm^2 [11·2 mW/g] for 1 h (Lu *et al.*, 1980*a*) or in rats in which thyrotropin (TSH) had been suppressed at the time of exposure by pretreatment with T3 (Lu *et al.*, 1980*b*).

Decreased serum thyroxine levels were noted in rats exposed to 2·45 GHz at 20 mW/cm^2 (3·2 mW/g) for 4–8 h (Lu *et al.*, 1977*a*). The thyroid depression apparently reflects the inhibition of hypophysial thyrotropin (TSH) secretion as evidenced by decreased circulating TSH before and accompanied by decreases in serum thyroxine (Lu *et al.*, 1977*b*). The TSH depression occurred in rats exposed to 2·45GHz microwaves (CW) at 10 mW/cm^2 for 1 and 2 h and 20 mW/cm^2 for 2 and 8 h (Lu *et al.*, 1977*b*). In rats exposed to 2·45 GHz at 13–60 mW/cm^2 for 30, 60 and 120 min there was no effect on TSH from a 30-min exposure but depressed TSH levels were noted in rats exposed at 30 mW/cm^2 (SAR = 4·8 mW/g) or higher for 60 min and 13 mW/cm^2 (SAR = 2·1 mW/g) or higher for 120 min (Lu *et al.*, 1980*b*).

Extrapolation from animals to man

In order to extrapolate observations in animals to predict results that might be obtained during human exposures, some method of scaling must be employed. The extent of the response induced by exposure to MW/RF energies depends on the amount of energy absorbed, which is a function of the wave-length, and on the geometry, orientation, and electrical properties of the object(s) being irradiated (Durney *et al.*, 1978). Maximum absorption (maximum SAR) during whole-body irradiation of small animals apparently occurs at frequencies between approximately 0·5 and 3 GHz and, for man, at around 60–100 MHz with a peak at about 80 MHz. At frequencies below 30 MHz, absorption drops off rapidly and is also much less at frequencies above about 500 MHz.

Specific absorption rate

MW/RF energy absorption is strongly dependent on polarization, frequency, and the immediate physical environment and is rapidly converted to thermal energy. This thermal energy is then rapidly redistributed by thermal conduction, convection (*e.g.* blood flow) and, to a lesser extent, radiation by the biological target. The tissue heat capacity and heat-transfer processes influence the dose and dose distribution within the body.

An effort is being made to standardize dosimetric measures of MW/RF exposure by employing a quantity called the Specific Absorption Rate (SAR). The unit-mass, time-averaged rate of MW/RF energy absorption is specified in SI units of watts per kilogram (W/kg) (National Council on Radiation Protection and Measurement, 1981). The SAR depends on a finite period of exposure to yield the amount of energy absorbed by a given

mass of material, which is termed specific absorption (SA), *i.e.*, Joules per kilogram = J/kg = W s/kg. Thus the *specific absorption* rate is the time rate at which radio-frequency electromagnetic energy is imparted to an element or mass of a biological body. The SAR is applicable to any tissue or organ of interest, or may be expressed as a whole-body average. SAR distributions are highly variable since they are dependent on wavelength, polarization, and zone of the incident field, and on mass and momentary geometry of the biological body.

It is now well established that the body as a whole exhibits frequency-dependent rates of absorbing radiofrequency energy (Durney *et al.*, 1978). Whole-body absorption rates approach maximal values when the long axis of a body is parallel to the E-field vector and is 0·4 × the wavelength of the incident field. At 2450 MHz, (λ = 12·5 cm) for example, Standard Man (long axis 175 cm) will absorb about half of the incident energy. At frequencies that result in maximal absorption, which defines whole-body resonance, the electrical cross-section of an exposed body increases in area. This increase occurs at a frequency near 70 MHz for Standard Man and results in an approximate 7-fold increase of absorption relative to that in a 2450-MHz field (Durney *et al.*, 1978).

Conclusion

Physiological regulation represented by neuroendocrine function, neurochemical activity, thermoregulatory, behavioural and immune responses is a composite of exquisitely "tuned" interrelated systems that constitute sensitive indicators of body responses to thermal incursions in the body. Exposure to microwave/radiofrequency fields may influence such physiological regulation.

Microwave - induced physiological changes cannot be dissociated from increases in tissue temperature. Such responses are considered to be essential in defence against homoeothermic imbalance as a febrile response is essential for host immune defence. These responses can also be considered to reflect the utilization of physiological function to maintain regulations or adjustments.

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