BIOLOGICAL EFFECTS AND EXPOSURE STANDARDS FOR NON-IONIZING ELECTROMAGNETIC ENERGIES*

Sol M. Michaelson
University of Rochester
School of Medicine
Department of Radiation Biology and Biophysics
Rochester, New York 14642

Abstract

Non-ionizing radiant energies at certain frequencies, intensities, and durations of exposure can produce biological effects which may be beneficial as well as harmful. For the general population and those persons exposed or with potential for exposure to these energies, personnel exposure guidelines and product emission standards have been promulgated for some of these energies. Personnel protection guides or exposure standards are usually those established by the American National Standards Institute (ANSI), American Conference of Governmental Industrial Hygienists (ACGIH) or Department of Defense. Some industrial organizations have standards of their own which may be modifications of the national standards. Legislation for personnel exposure and product emission levels for NIR are covered under the Occupational Safety and Health Act of 1970 and the Radiation Control for Health and Safety Act of 1968, respectively. It is important that distinction be carefully made between product emission standards and personnel exposure standards and how they relate to potential injury. A proper perspective and realistic assessment of the biomedical effects of these radiant energies is essential so that the individual or general public will not be unduly exposed nor will research, development and beneficial utilization of these energies be hampered or restricted.

Introduction

During the last quarter century there has been a marked development and increased utilization of equipment and devices for military, industrial, consumer use, and medical applications that emit a large variety of non-ionizing radiant (NIR) energies; these include ultraviolet, infrared, visible light, microwaves, and radio-frequency. Because of the biological consequences of these energies, the "Radiation Control for Health and Safety Act of 1968" (PL-90-602) and the "Occupational Safety and Health Act of 1970" (PL-91-596) (OSHA) were enacted to protect the general public as well as the worker.

---

*The work upon which this paper is based was performed pursuant to Contract No. FDA 73-30 with the Public Health Service, Food and Drug Administration, Department of Health, Education and Welfare, with support by the Bureau of Medicine and Surgery, U.S. Navy, and with the U.S. Atomic Energy Commission at the University of Rochester Atomic Energy Project and has been assigned Report No. UR-3490-247.

The Radiation Control for Health and Safety Act requires the Secretary of Health, Education and Welfare (HEW) to prescribe performance standards for U.S. produced and imported electronic products, if he determines that such standards are necessary for the protection of public health and safety. An electronic product, under the Radiation Control Act, is any product that uses an electronic circuit and that may generate ionizing or NIR, or sound waves. Any manufactured or assembled product is covered by the Act if it emits radiation and contains an electronic circuit or functions as part of an electronic circuit. Responsibility for day-to-day administration of the Act has been delegated to the Bureau of Radiological Health (BRH).

To assure safe and healthful working conditions, OSHA provides broad authority to the Departments of Labor and HEW to develop criteria for dealing with potentially toxic materials and harmful physical agents,such as NIR, indicating safe exposure levels for workers for various periods of time.

Some NIR energies at certain frequencies, intensities, and exposure durations can produce biological effects or injury depending on multiple physical and biological variables. Although devices which utilize or emit NIR provide immeasurable benefits to mankind, they may also create potential hazards to the individual through uncontrolled and excessive emissions. Consequently, questions are being raised such as: 1) Are there any problems, and if so, how serious are they? 2) How adequate is our present knowledge about hazard to personnel from these energies? 3) How can exposure be reduced? 4) How can better regulation be obtained to reduce exposure?

For the general population and those persons exposed or with potential for exposure to these enegies in the course of their occupations, personnel exposure guidelines and some product emission standards have been promulgated. Personnel protection guides or exposure standards are usually those established by the American National Standards Institute (ANSI), American Conference of Governmental Industrial Hygienists (ACGIH) or Department of Defense as well as BRH. Some industrial organizations have standards of their own which may be modifications of the national standards.

Standards

A summary of the various guidelines and standards is shown in Table I.[1-5] In spite of the fact that this compilation is oversimplified and many details are omitted, it does indicate the complexity and variety of protection guides for NIR.

TABLE I

Protection Guides and Standards for Non-Ionizing Radiant Energies

Energy	Wavelength	Guide Number	Duration of Exposure	Comments
Ultraviolet	200-315 nm	3 mJ/cm^2-1 J/cm^2	8 hr	radiation incident on skin or eye
		(3 mJ/cm^2-1 J/cm^2)*	10^{-3}-$3x10^4$ sec	direct ocular or skin exposure
Visible	400-700 nm	($5x10^{-7}$ J/cm^2)	10^{-9}-$1.8x10^{-5}$ sec	direct ocular exposure
		($2x10^{-2}$ J/cm^2)	10^{-9}-10^{-7} sec	skin exposure
Infrared	800-1100 nm	7.6 J/cm^2	----	corneal damage
	1200-1700 nm	2.8 J/cm^2	----	corneal damage
	800-1100 nm	10.8 J/cm^2	----	corneal dose causing iris damage
	700-1000 nm	(10^4 w/cm^2-$5x10^{-1}$ w/cm^2)	10^2-$3x10^4$ sec	direct ocular exposure
		(0.2 W/cm^2)	10-$3x10^4$ sec	skin exposure
Microwave	3 mm-100 cm	10 mW/cm^2	continuous	whole body (higher power permitted for localized exposure)
		25 mW/cm^2	10 min during any 60 min period	
		1-5 mW/cm^2 at 5 cm from external surface	continuous	microwave oven product emission standard
Radiofrequency	1 m-1000 m	0.3 mA/cm^2-1mA/cm^2 1000 V/m	continuous	whole body

* ( ) for coherent sources - laser.

To insure uniform and effective control of potential health hazards from exposure to NIR, it is necessary to establish standards or protection guides. Detailed discussion of exposure standards is presented by Matelsky[6], Michaelson[3,7], and Schwan.[8]

Ideally, effect or threshold values should be predicated on firm human data. If such data are not available, however, extrapolation from well-designed, adequately-performed and properly analyzed animal investigations is required. In discussing standards for NIR, it is necessary to keep in mind the essential differences between a "personnel exposure" standard and a "performance"

standard for a piece of equipment and how they relate to each other. An exposure standard refers to the safe (incorporating a safety factor of at least 10) level of whole-body exposure and exposure time. This standard is a guide to people on how to limit exposure for safety. An emission standard (or performance standard) refers not to people but to equipment and specifies the maximum emission close to a device which ensures that likely human exposure will be at levels far below this limit which essentially is several orders of magnitude below the personnel exposure standard. As an example, one can cite the standards for microwaves. For personnel exposure the standard is 10 mW/cm^2. For microwave ovens the emission or product performance standard is 1 mW/cm^2 at manufacture and a maximum of 5 mW/cm^2 throughout the life time of the oven. This level is measured at 5 cm from the external surface and should be considered in relation to a restricted field with only a small area of the body potentially exposed.

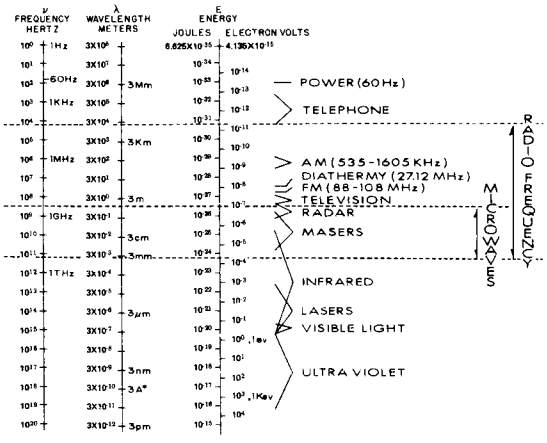
Conceptually, as well as practically, these guidelines bear no relationship to the use of these energies in the context of medical diagnosis and treatment and should not be applied for such purposes. These standards for product emission and personnel exposure are designed to protect the general public and the worker, and are based on entirely different criteria than one would apply for diagnostic and therapeutic purposes. In the medical context, on the basis of occupational and general personnel protection standards, individuals are grossly "over-exposed" to radiant energies to achieve a specific diagnostic or therapeutic result. Diathermy at 2450 MHz creates incident energy exposures on a watt level to achieve desired tissue heating;[9] ultraviolet erythemal doses are pushed to the limit to control serious cases of acne vulgaris and psoriasis;[9] Q-switched lasers are used by ophthalmologists to literally "cook" the back of the eye to restore a detached retina to a semblance of its normal anatomic position.[10] To draw a parallel with ionizing radiation, used therapeutically, the localized exposures of cancer patients to incident ^{60}Co gamma radiation grossly exceed current guidelines for general population and occupational exposures. This is brought out simply to emphasize that current standards are in no way applicable to medical uses of any of the non-ionizing radiant energies, nor should they be.[11]

Biophysics

To provide a basis for understanding the biologic effects of NIR, review of some fundamental aspects of electromagnetic radiation is indicated. The non-ionizing electromagnetic (EM) spectrum encompasses wavelengths from $3x10^8$ m to $3x10^{-2}$ nm (fig. 1).[12]

Figure 1.

NON-IONIZING ELECTROMAGNETIC RADIATION*



*Adapted from Air Force Manual AFM 161-8, 1969

As the frequency decreases, the EM energy of the emitted photons is insufficient, under normal circumstances, to dislodge orbital electrons, and produce ion pairs. The minimum photon energy capable of producing ionization in water and atomic oxygen, hydrogen, nitrogen, and carbon is between 12 and 15 electron volts (eV). Inasmuch as these atoms constitute the basic elements of living tissue, 12 eV may be considered the lower limit for ionization in biological systems. Since the energy value of 1 quantum of NIR is considerably less than 12 eV, the type of electronic excitation necessary for ionization is not possible no matter how many quanta are absorbed. NIR absorbed into the molecule either

affects the electronic energy levels of its atoms, or changes the rotational, vibrational, and transitional energies of the molecules. Changes are produced in biological systems through either photochemical (ultraviolet) and/or thermal modes (infrared, microwaves).[13]

A factor that has been a source of continuing concern has been the problem of measurement of energy absorbed by biological tissue. Knowledge of the incident energy is inadequate to explain what is happening within biological structures, and these occurrences must be correlated with absorbed energy. In some cases of NIR exposure we are incapable of describing the incident energy, not to speak of its absorption, as is the case in the near-field of a microwave source.

The phenomena of reflection, transmission, and energy absorption occur in biological tissues that are exposed to some NIR energies. In the case of microwaves, these phenomena occur not only at the initial entry point or exposed area, but also at deeper tissue interfaces such as the fat-fascia-muscle layers, and within tissues themselves. When microwaves are used, frequency specificity of interactions create complex problems. Considerable effort will have to be expended in this area of dosimetry before problems, controversies, and existing confusion can be resolved.

Ultraviolet Energy

For ultraviolet (u.v.) exposure, the critical organs are the skin and eyes, resulting in erythema of the skin and skin cancer, rapid skin aging, photosensitization, and keratoconjunctivitis.

Specific absorbed wavelengths of u.v. that can elicit a specific biologic response constitute the "action spectra" for that response. These action spectra define the relative effectiveness of different wavelengths in eliciting a specific response when absorbed.[6] Determination of exposure criteria for u.v. effects has become increasingly difficult with the proliferation of action spectra. A great number of uncertainites still exist in what constitutes a "threshold" effect.[13]

In the intact animal, incident u.v. does not penetrate through the skin. Below 290 nm absorption in humans is entirely in the epidermis. Between 290 and 320 nm, less than 10 percent reaches the dermis; above 400 nm, over 50 percent reaches the dermis. Whole-body exposure to u.v. is possible, however common articles of clothing are effectively opaque to ultraviolet.

In 1948, the Council on Physical Medicine of the American Medical Association issued criteria for safe exposure to radiant energy from u.v. germicidal lamps.[2] This group recommended that for the primarily used wavelength, 253.7 nm, exposures should not exceed 0.5 $\mu W/cm^2$ for periods <7 h, nor 0.1 $\mu W/cm^2$ in the case of continuous exposure.

The American Conference of Governmental Industrial Hygienists (ACGIH) has also proposed threshold-limit values (TLV) for u.v.[1] The TLV for occupational exposure to u.v. incident upon skin or eye where irradiance values are known and exposure time is controlled are as follows: 1) For the near u.v. spectral region (λ 320 to 400 nm) total irradiance incident upon the unprotected skin or eye should not exceed 1 mW/cm^2 for periods $>10^3$ s and for exposure times $<10^3$ s should not exceed 1 J/cm^2. 2) For the actinic u.v. spectral region (λ 200 to 315 nm), radiant exposure incident upon the unprotected skin or eye should not exceed 100 mJ/cm^2 for λ 200 nm to 1000 mJ/cm^2 for λ 315 nm within an 8 h period. However, the relative spectral ($S\lambda$) effectiveness of the radiation has to be taken into consideration, i.e. for 270 nm $S\lambda = 1.0$, for 254 nm $S\lambda = 0.5$. These are described as follows:[1,13]

	$\mu W/cm^2$	λ (nm)	$S\lambda$
Maximum Erythemal Dose	$2x10^4$ $3x10^3$ $1.26x10^4$	297 300 254	1.0 0.3 0.5
Maximum Keratitic Dose	10^4 $3x10^3$	288 270	1.0 1.0

A criteria document for a recommended standard for Occupational Exposure to Ultraviolet Radiation has been published by the U.S. Dept. of HEW, National Institute for Occupational Safety and Health (NIOSH). This document incorporates the ACGIH proposed TLV for Ultraviolet Exposure. These recommendations have as yet not been acted upon by OSHA.[13]

The above are related to exposures to the non-ionizing, non-coherent sources of u.v., where the mode of interaction with matter has been primarily a photochemical one. It is likely that exposure to low-power lasers emitting in the u.v. will also produce photochemical reactions. However, the highly collimated aspect of such generators may produce energy densities on the cornea sufficient to give rise to thermal effects. With higher output lasers, sufficient energy may penetrate the cornea of the eye and the epithelium of the skin to produce effects not known at present.[13]

Visible Energy (Light)

The hazards to man from visible light are relatively few and mostly come from artificial sources such as lasers and certain high intensity light sources which can produce absorbed energy levels greater than 50 $cal/cm^2/min$. Items which would probably fall in this category include high intensity reading lamps, movie and slide projector bulbs, spot lights, flood lights, etc.

The penetrating ability of visible light is slight except for transparent materials such as the lens and humors of the eye. Light entering the eye from a bright source is focused on the retina and therefore, the thermal irradiance is independent of the inverse square law for image sizes greater than the diffraction limit.[14] Because of its narrow depth of penetration, visible light in general does not manifest itself as a potential hazard. There are situations, however, in which it can become hazardous. For example, pulsating light at certain frequencies has been reported as a potential source for producing psychological effects. Epileptiform responses have been produced in animals and children exposed to pulsating light near the alpha rhythm frequency of the EEG.

Due to the optical properties of the eye the heat energy per unit area on a small part of the retina may be greater by a factor of 10^5 than on the cornea. For visible light a power density of 1 W/cm^2 will exceed the threshold for pain within 1 s; with a thermal time constant of 0.1 s, the threshold energy density per pulse will be 0.1 J/cm^2. These factors become exceedingly important in relation to coherent light sources (laser). The sensation of heat, however, serves as an effective warning system under those conditions where there is time to react.

Normally, intense and bright sunlight causes maximal constriction of the pupil thus reducing the energy density on the retina. Bright sunlight, furthermore, causes painful photophobia which will not permit prolonged direct and fixed observation of the sun. The lid reflex (approximately 150 ms) is another mechanism to protect the eye. The continuous action of these measures would be adequate under normal conditions to avoid burn injuries to the retina.[15]

There appear to be three predominant factors controlling potential hazard to the eye: 1) intensity, 2) pupil dilation (that is, the area of exposure), and 3) length of exposure. If these factors are controlled to keep the absorbed energy below the threshold of thermal burning (reported to be between 40 and 50 cal/cm^2 per min), no eye injury should be expected.

Infrared Energy

Infrared (i.r.) extends from beyond the red end of the visible portion of the EM spectrum (750 nm) to about 1x10^6 nm. The i.r. spectrum is frequently arbitrarily divided into three bands: the near i.r. (750-3000 nm), the middle i.r. (3x10^3 - 3x10^4 nm), and the far i.r. (3x10^4 to approximately 1x10^6 nm).

There is little evidence that photons in the i.r. (i.e., less than 1.5 eV) are capable of entering into photochemical reactions in biological systems, probably because they are too low in energy to affect the electron energy levels of these atoms. The interaction that does occur upon absorption involves an increase in the kinetic energy of the system, producing a degradation of the radiant energy to heat.[6]

Most biological materials are considered opaque to wavelengths shorter than 1500 nm because of the almost complete absorption of these energies by water. Radiant energies in the short wavelength region of the near i.r. can be transmitted into the deeper tissues of the dermis and the eye.

The most prominent direct effects of low wavelength i.r. on the skin include acute skin burn, increased vasodilation of the capillary beds, and an increased pigmentation which can persist for long periods of time. Under conditions of continuous exposure to high intensities of i.r., the erythematous appearance due to vasodilation may become permanent. Many factors mediate the ability to produce actual skin burn, and it is evident that for this immediate effect, the rate at which the temperature of the skin is permitted to increase is of prime importance.[6]

The threshold for warmth perception in the skin is reached at a warming of the skin at a rate of about .001-.002°C/s at a skin temperature of 32°C-37°C. Threshold and intensity of temperature sensation depend to a large extent on the size of the skin area changing temperature. Similarly, the minimal time of warming the skin before a temperature sensation is elicited depends on the size of the area affected and on the density of the specific temperature receptors in that area. Experimental evidence indicates that temperature sensation is little influenced by the absolute temperature of the skin and is governed by the rate of change of the skin temperature.[16] Results of Cook,[17] however, indicate that skin temperature is the vital factor in determining pain, though only in so far as this is a measure of the temperature of the thermal pain receptors below the skin surface.

The cornea of the eye is highly transparent to energies between 750 nm and 1300 nm and becomes opaque to radiant energy above 2000 nm. Thermal damage to the cornea is dependent upon the absorbed dose, and probably occurs in the thin epithelium rather than in the deeper stroma. A dose of 7.6 W s/cm^2 of λ 880-1100 nm was found to elicit minimum regressive corneal damage; whereas only 2.8 W s/cm^2 λ 1200-1700 nm produced this response.[18] These values are consistent with absorption characteristics. With excessive exposure to these critical wavelengths, there may be complete destruction of the protective epithelium, with opacification of the stroma due to coagulation of the protein. Obviously, such denaturization in an area over the pupil would seriously interfere with vision. The probability of incurring such an insult is low except where highly collimated sources can irradiate the eye without producing the sensation of pain in the surrounding skin tissue.[6]

Damage of the lens of the eye from i.r. has been the subject of considerable investigation over a period of many years. The term "Glass-Workers" cataract has become generic for lenticular opacities found in individuals exposed to processes hot enough to be luminous.[6] In 1907, Robinson[19] published the results of his investigations in England on the incidence of opacities on the posterior surface of the lens in the eyes of glass workers that were different than senile cataracts in appearance. It was upon his recommendation that the disease, radiation cataract, became scheduled in England as occupational in origin and by 1921 was copied into the U.S. Workman's Compensation Act. Although some serious dissent has arisen as to the validity of the data obtained by earlier investigators,[20] the weight of evidence as of now favors the concept that i.r. emitted from hot sources in industry is the etiological agent responsible for i.r. cataractogenesis.[21]

Protection guides for i.r. exposure are designed primarily for protection against ocular effects. The main difficulty, however, in devising protection standards against i.r.-induced cataract is to correlate the information on the radiation emitted during industrial processes with cataract formation. The intensities of i.r. which cause cataract are unknown. Only a small amount of experimentation on animals has been done but it has provided some knowledge of the way cataract is formed; the numerical data obtained cannot be used in devising standards, due to the relatively massive and frequent doses used in experiments, and possible physiological and anatomical differences in rabbit and human eyes.[22]

The tolerance limits of the human body for i.r. have been determined. An incident intensity of 0.04 cal/cm^2/s of short-wave i.r. can just be tolerated by epigastric and interscapular skin areas of 144 cm^2.[23] Approximately 25% of this energy flux would be reflected, so this corresponds to a tolerated transmitted intensity of 0.03 cal/cm^2/s. It can be estimated that the maximum incident intensity of long-wave i.r. that can be tolerated by a lumbar area, 12x12 cm, is also approximately 0.03 cal/cm^2/s.[24]

Transmission and absorption factors of the ocular media for the i.r. spectrum and threshold doses to elicit minimum damage have been determined:[6,18]

1) For corneal damage: 7.6 J/cm^2, 800-1100 nm; 2.8 J/cm^2, 1200-1700 nm.
2) Corneal exposure to produce damage in the iris: 10.8 J/cm^2, 800-1100 nm.
3) Corneal exposure for production of retinal burns: 1 J/cm^2 (this value determined with a 0.1 s exposure to 20-40 J/cm^2 causing a 1 mm burn).

Laser

The acronym LASER (light amplification by stimulated emission of radiation) is commonly applied to a device which produces an intense, coherent directional beam of light by stimulating electronic or molecular transitions to lower energy levels.[5] The characteristics of lasers which influence their effect upon biological systems include the duration of the pulse, the time interval between pulses, the specific wavelength emitted, and the energy density of the beam. The degree of damage produced depends upon the absorbing tissue, its absorption characteristics, the size of the absorbing area, and its vascularity.[6] It has become common practice to describe the output of pulsed lasers in terms of energy (joules), and that from continuous wave (CW) lasers in terms of power (watts). The J/cm^2 is used to express absorbed energy density, and the W/cm^2 to describe power density.[6]

Biologic effects can occur through three mechanisms of interaction: a) thermal effect; b) acoustic transients; or c) other phenom[illegible] The latter

two effects are only seen with high power density laser pulses. When laser light impinges on tissue, the absorbed energy produces heat. The resultant rapid rise in temperature can easily denature tissue protein. Since tissue is not homogeneous, light absorption is not homogeneous and the thermal stress is greatest around those portions of tissue that are the most efficient absorbers. Rapid and localized absorption produces high temperatures. Steam production, evident only at high exposure levels, can be quite dangerous if it occurs in an enclosed and completely filled volume such as the cranial cavity or the eye. A second interaction mechanism is an elastic or acoustic transient or pressure wave. As the light pulse impinges on tissue, a portion of the energy is transduced to a mechanical compression wave (acoustic energy), and a sonic transient wave is built up. This sonic wave can rip and tear tissue and if near the surface, can send out a plume of debris from the impact. Other phenomena such as free radical formation, are believed to exist during laser impact on biological systems, but this has not yet been conclusively demonstrated.[26]

The primary hazard from laser radiation is exposure of the eye. Exposure levels, if kept below those damaging to the eye, will not harm other tissues and organs of the body. Eye damage can range from mild retinal burns, with little or no loss of visual acuity, to severe lesions with loss of central vision, and total loss of the eye from gross over-exposure. Long-term exposure of the retina to wavelengths in the visible spectrum, at levels not far below the burn threshold, may cause irreversible effects.

Excessive i.r. (1.4-1000 μm) exposure causes a loss of transparency or produces a surface irregularity in the cornea. Damage results from heating of the cornea by absorption of the incident energy by tears and tissue fluid in the cornea. Although the critical temperature threshold is not known, it does not appear to be much above normal body temperature, and there are indications that it is a function of exposure time.[5]

Excessive u.v. (0.2-0.4 μm) exposure produces photophobia accompanied by redness, tearing, conjunctival discharge, surface exfoliation, and stromal haze. Damage to the corneal epithelium by absorption of u.v. probably results from photochemical denaturation of proteins or other molecules in the cells. Some of the most important molecules are the desoxyribonucleic acids (DNA) and ribonucleic acids (RNA). The absorption is probably by selective sensitive portions of single cells. Thus the action of the u.v. is photochemical rather than thermal, since the temperature rise calculated for experimental exposure is negligible.[5]

The type of damage inflicted on the eye by laser beams ranges, therefore, from a small and inconsequential retinal burn in the periphery of the fundus, to severe damage of the macular area, with consequent loss of visual acuity, up to massive hemorrhage and extrusion of tissue into the vitreous, with possible loss of the entire eye.[27]

The large skin surface makes this tissue readily available to accidental and repeated exposures to laser energies. The biological significance of exposure of the skin to lasers operating in the visible and i.r. regions is considerably less than exposure of the eye, as skin damage is usually repairable or reversible. Effects may vary from a mild reddening (erythema) to blisters, and charring. Depigmentation of the skin, ulceration and scarring and damage to underlying organs may occur from extremely high powered laser sources.

Latent and cumulative effects of laser exposure are not known at this time. Little or no data are available describing the reaction of skin exposed to lasers in the 0.2-0.4 μm spectral region, but chronic exposure to u.v. in this range can have a carcinogenic action on skin as well as eliciting an erythematous response.

On the basis of studies with non-coherent u.v., exposure to wavelengths in the 0.25-0.32 μm spectral region is most injurious to skin. Exposure to the shorter (0.20-0.25 μm) and longer (0.32-0.40 μm) u.v. is considered less harmful to normal human skin. The shorter wavelengths are absorbed in the outer dead layer of the epidermis (stratum corneum), and exposure to the longer wavelengths has merely a pigment-darkening effect. The sensitivity of skin, however, to the longer wavelengths may be increased by known or inadvertent usage of photosensitizers.[5]

One cannot discuss potential hazards from laser energies without mentioning operationally associated hazards such as: 1) compressed gases, 2) cryogenic liquids, 3) ionizing radiation that may emanate from laser power supplies and components, 4) toxic materials used in laser targets or laser system elements. Attention should also be paid to adequate ventilation to eliminate or reduce exposure to toxic materials to safe levels.[5]

ANSI, ACGIH, and BRH have or are in the process of developing laser standards. Selected maximum permissible exposure (MPE) laser levels for ocular effects are shown in Table II. These have to be understood, however, in the context and with consideration of the laser classification system that has been developed.[5] Because of the complexity of these standards, the ANSI standard[5] and the ACGIH, TLV[1] should be consulted.

TABLE II

Maximum Permissible Exposure (MPE) to Laser for Direct Ocular Intrabeam Viewing for Single Pulses or Exposures*

Spectral Region	Wave Length	Exposure Time, (t) Seconds	MPE
Ultraviolet	200 nm to 302 nm	10^{-2} to 3 X 10^{4}	3 mJ · cm^{-2}
	305 nm	" "	10 mJ · cm^{-2}
	310 nm	" "	100 mJ · cm^{-2}
	315 nm	" "	1.0 J · cm^{-2}
	315 nm to 400 nm	10^{3} to 10^{4}	1.0 mW · cm^{-2}
Visible and Near Infrared	400 nm to 1.4 μm	10^{-9} to 2 X 10^{-5}	5 X 10^{-7}J · cm^{-2}
	" "	10 to 10^{4}	1.0 mJ · cm^{-2}
	" "	10^{4} to 3 X 10^{4}	10^{-6} W · cm^{-2}
Far Infrared	1.4 μm to 10^{3} μm	10^{-9} to 10^{-7}	10^{-2} J · cm^{-2}
	" "	10 to 3 X 10^{4}	0.1 W · cm^{-2}

*Adapted from ANSI (5); ACGIH (1).

Microwaves/Radiofrequency

The radiofrequency (rf) portion of the EM spectrum is considered to extend from 0.03 MHz (very low frequency -VLF) to 300,000 MHz (extremely high frequency - EHF). On a functional or operational basis, frequencies in the region from 100 MHz to 300,000 MHz (300 GHz) are designated as microwaves.

Of the various NIR energies, the rf and microwave bands have elicited the greatest interest and concern as well as confusion in consideration of the real and substantiated effects vis a vis unsubstantiated or speculative effects. When considering the biological effects of rf energy, the wavelength of the energy and its relationship to the physical dimensions of exposed objects become important factors. Absorption of the energy depends upon the dielectric properties of the tissues and the relative absorption cross section of the exposed subject. Thus, the size of the object with relation to the wavelength of the incident field plays an important role.[28]

In biological systems absorbed rf is transformed into increased kinetic energy of the absorbing molecules, thereby producing a general heating of the tissue. Such heating results from both ionic conduction and vibration of the dipole molecules of water and proteins.[29] The absorption of rf is dependent upon the electrical properties of the absorbing medium, specifically, its dielectric constant and electrical conductivity. These properties change as the frequency of the applied electric field changes. Values of dielectric constant and electrical conductivity and depth of penetration have been determined for many tissues.[30] The absorption of rf energy is high and the depth of penetration low in tissues of high water content such as muscle, brain tissue, internal organs, and skin, while the absorption is lower in tissues of low water content such as fat and bone. Reflections between interfaces separating tissues of high and low water content can produce standing waves

accompanied by "hot spots" that can be maximum in either tissue, regardless of dielectric constant or conductivity.[29]

Extensive investigations into microwave bioeffects conclusively show that for frequencies between 200 MHz and 24,500 MHz, exposure to power density of >100 mW/cm^2 for 1 h or more could have pathophysiologic manifestations of a thermal nature. At power densities <100 mW/cm^2, however, evidence of pathologic change is non-existent or equivocal. According to the best evidence available, the most important, if not the only, effect of microwave absorption in the mammal is the conversion of the absorbed energy into heat. Whole-body exposure of various species of animals to microwaves at levels >10 mW/cm^2 is characterized by a temperature rise which is a function of the thermal regulatory process of the animal. The end result is either reversible or irreversible change depending on the conditions of the exposure and the physiologic state of the animal.[31] Smaller animals show a greater temperature response than do larger animals at equivalent exposures.[32]

Irradiance levels of 10,000 MHz and 3000 MHz microwaves required to produce a threshold sensation of warmth have been obtained.[33,34] These data indicate that when a 40 cm^2 area of the face is exposed to microwaves, thermal sensation can be elicited within 1 s at a power density of 21 mW/cm^2 for 10,000 MHz and 58.6 mW/cm^2 for 3000 MHz. Within 4 s the threshold is lowered by approximately 50%, i.e. 12.5 mW/cm^2 (10,000 MHz) and 33.5 mW/cm^2 (3000 MHz). On this basis, if the entire face were to be exposed, the threshold for thermal sensation to 10,000 MHz would be 4-6 mW/cm^2 within 5 s or approximately 10 mW/cm^2 for a 0.5 s exposure. Threshold for pain reaction to 3000 MHz exposure of a 9.5 cm^2 area of the forearm ranges from 830 mW/cm^2 for exposures longer than 3 min to 5.6 W/cm^2 for a 20 s exposure period. If a larger area (53 cm^2) is exposed, the pain threshold for a 3 min exposure is 560 mW/cm^2.[17] These data and other information on microwave sensation suggest that cutaneous perception of microwaves may provide a protection factor with sufficient margin of safety constituting a warning mechanism to prevent exposure to microwaves at levels that could be injurious.[3]

Microwaves have been shown to produce cataracts in some experimental animals, notably rabbits, and there are also reports of microwave-induced cataracts in man. In animal studies, the techniques used and interpretation of the results and conclusions, however, are quite often equivocal. Careful review of the reports on human cataractogenesis indicates that there has been insufficient quantitation and correlation of pathophysiology with the level of microwave exposure.

Carpenter and his associates[35] have reported that single or repeated exposures of rabbits' eyes with 2450 MHz pulsed or CW can cause opacity when the lens temperature increases 4°C. These authors have suggested a "cumulative" effect on the lens from repeated "sub-threshold" exposures of rabbits' eyes to microwaves.

In order not to confuse this suggested "cumulative" effect with that recognized for ionizing radiation, it is important to define the cumulative effect produced by ionizing radiation to put this point in its proper perspective. Cumulative injury from exposure to ionizing radiation is a manifestation of the irrepairability of a certain fraction of the injury which has been designated as Residual Radiation Injury. Such 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.[36] A cumulative effect is the accumulation of damage resulting from repeated exposures each of which is individually capable of producing some degree of damage. Careful analysis of the work of Carpenter *et al*[35], as well as Williams *et al*[37] and Birenbaum *et al*[38] reveals that whenever lens opacity is produced in animals, a threshold (>100 mW/cm^2; >1 h) becomes obvious. No one

has yet been able to produce cataracts even by repetitive exposures when the power density is really below threshold.

It is important to note that lens opacity has consistently been produced in only one species, namely the rabbit. One can question whether the rabbit is the most appropriate animal model. According to Cogan et al[39], with local microwave exposure the cataractogenic level for monkeys has been found to be higher than for rabbits.

Most epidemiological studies in the U.S. have involved the ocular lens. The few reports[40,41,42,43,44] that are available are highly questionable and have not been found acceptable by competent ophthalmologists.

The suggestion that microwaves may interact with the central nervous system (CNS) by some mechanism other than heating has been made by several investigators, mostly in East European countries, who stress that the CNS must be considered as being moderately or highly sensitive to rf or microwave energy absorption. The first report on the effects of microwaves on conditional response activity of experimental animals was made by Gordon et al.[45] In subsequent years, the study of the "nonthermal" effects of microwaves gradually occupied the central role in electrophysiological studies in the Soviet Union.[46]

Many investigators do not accept the possibility of nonthermal neural stimulation by microwaves and explain these effects entirely upon local heating.[47,48,49] They suggest that thermal stimulation of the peripheral nerves could produce the neurophysiological and behavioral changes that have been reported.

Eastern European investigators have contributed most of the reports of effects of rf and microwave energies in man.[45,46,50,51] The greatest emphasis is placed on effects produced at less than "thermogenic" power flux densities (<10 mW/cm^2). According to these investigators, the basic symptomatology and neuropathology underlying all of the reported syndromes is described as due to the functional disturbance created in the CNS by "non-thermal" mechanisms. These effects are reported to occur in occupational exposures at levels far below those required to produce a temperature rise. The symptoms are manifested by weakness, fatigue, vague feelings of discomfort, headache, drowsiness, palpitations, faintness, memory loss, and confusion. These syndromes are apparently completely reversible in most cases, with little or no time lost from work.[52] Much of these reports is based on subjective rather than objective findings, and measurement of field intensity in most cases is not comparable from worker to worker or factory to factory.[53]

Dodge,[54] in his review of the Soviet research in this area, has stated, "An often disappointing facet of the Soviet and East European literature on the subject of clinical manifestations of microwave exposure is the lack of pertinent data presented on the circumstances of irradiation...important environmental factors (heat, humidity, light, etc.) are often omitted from clinical and hygienic reports." A point that should be noted is that in the West the effects reported by East European investigators have not been observed, even at much higher exposure levels.

Microwave exposure standards for most of the Western world are based, with minor variations, on standards developed in the U.S. (table III). The original U.S. standard was tentatively adopted about 15 years ago on the basis of theoretical considerations by Schwan and his associates. This standard was based on the amount of exogenous heat which the body could tolerate and dissipate without any resulting rise in body temperature. This tolerance level was calculated to be 10 mW/cm^2 for continuous exposure. Intensive investigation into the biological effects of microwaves was subsequently carried out by the U.S. Department of Defense. None of these investigations was able to produce any evidence for a biological effect at levels even approaching the theoretical level of 10 mW/cm^2 and, indeed, no conclusive evidence was established for any effect below the level of 100 mW/cm^2.[31]

The ANSI standard of 10 mW/cm^2 for radiofrequency exposure recommended in 1966 and reaffirmed in 1973,[4] is roughly a factor of ten below thresholds of damage by thermal effects, assuming a long duration of exposure--i.e., one quarter h or more. The 10 mW/cm^2 level is based on thermal equilibrium conditions for whole-body exposure. For normal environmental conditions and for incident electromagnetic energy of frequencies from 10 MHz to 100 GHz, the radiation protection guide is 10 mW/cm^2 and the equivalent free-space electric and magnetic field strength: approximately 200 V/m RMS and 0.5 A/m RMS, respectively. For modulated fields, power density and the squares of the field intensities are averaged over any 0.1 hour period, i.e. none of the following levels should be exceeded in any 0.1 hour period: Electric Field Strength Squared - 40000 V^2/m^2; Magnetic Field Strength Squared - 0.25 A^2/m^2; Power Density - 10 mW/cm^2; Energy Density - 1 mWh/cm^2; this guide applies whether the radiation is CW or intermittent.[4]

Table III.

RECOMMENDED MAXIMUM PERMISSIBLE INTENSITIES FOR RADIO-FREQUENCY RADIATION

MAXIMUM PERMISSIBLE INTENSITY	FREQUENCY (MHz)	COUNTRY OR SOURCE	SPECIFICATIONS
	10-100,000	U.S.A.S.I. 1966; CANADA 1966	1 mWh/cm^2 FOR EACH 6 MIN.
	30-30,000	GREAT BRITAIN 1960	DAILY EXPOSURE
	1000-3000	SCHWAN AND LI 1956	WHOLE BODY
10 mW/cm^2	ALL	U.S. ARMY AND AIR FORCE 1965	10 mW/cm^2 CONT. EXP. 10-100 mW/cm^2, LIM. OCCUP. MIN. = $\frac{6000}{(X mW/cm^2)^2}$
		SWEDEN 1961	OCCAS. EXP. (OCCUPAT.)
		GERMAN FED. REPUBLIC 1962	--------
	ALL	SWEDEN 1961	GENERAL PUBLIC PROLONGED OCCUPAT. EXP.
1 mW/cm^2	> 300	USSR 1965; POLAND 1961	15-20 MIN/DAY
0.1 mW/cm^2	> 300	USSR 1965; POLAND 1961	2-3 HR/DAY
0.025 mW/cm^2	> 300	CZECHOSLOVAKIA 1965	CW 8 HR/DAY
0.01 mW/cm^2	> 300	USSR 1965 POLAND 1961 CZECHOSLOVAKIA 1965	6 HR/DAY ENTIRE DAY PULSED-8 HR/DAY
20 V/m	0.1-30	USSR 1965	
10 V/m	0.01-300	CZECHOSLOVAKIA 1965	PULSED-8 HR/DAY
5 V/m	30-300	USSR 1965	

There is no evidence in the scientific or medical literature of the Western world, that the present U.S. standards represent a hazardous exposure level. The ANSI standard[4] has been accepted by OSHA and with very little modification throughout the Western world. Microwave exposure standards for most of the Eastern European nations are based, with minor variations, on limits established by the USSR (table III). These limits, promulgated in 1959 by the USSR Ministry of Health are: a) 0.01 mW/cm^2 for an entire workday; b) 0.1 mW/cm^2 for 2 h; c) 1.0 mW/cm^2 up to 20 min. These standards are based on vague "asthenia" syndromes reported by individuals who work with microwave/rf energies. These effects have not been demonstrated by Western investigators.

The apparent discrepancy in maximum allowable exposures between Eastern European and Western countries may be due to differences in industrial hygiene philosophy. Magnuson et al[55] have noted that in the USSR, MPE is based on presence or absence of biological effects without regard to the feasibility of reaching such levels in practice. The MPE represents a desirable level for which to strive rather than an absolute value to be used in practice.

Conclusion

It is important to maintain a proper perspective, and assess realistically the biomedical effects of NIR so that the worker or general public will not be unduly exposed nor will research, development and beneficial utilization of these energies be hampered or restricted. There is a need for scientific competence and integrity. Although there is considerable agreement among scientists concerning the biologic effects and potential hazards of NIR, there are areas of disagreement, especially in relation to rf and microwaves. It is essential that research into the biologic effects of NIR be fostered and advanced to counteract the often voiced "what we don't know can hurt us" attitude with consequent overly restrictive and unrealistic standards. Free international exchange and closer personal contact between scientists would be invaluable in resolving discrepancies and divergence of opinion that exist in the understanding of some of the biologic and clinical implications of exposure to NIR and approaches to the setting of standards or protection guides.

Acknowledgement - The assistance of Mrs. Margaret Anderson Bush in organizing the material and typing this manuscript is acknowledged with appreciation.

References

[1] Threshold Limit Values for Chemical Substances and Physical Agents. Am. Conf. Govern. Industr. Hyg., Cincinnati, 1972, 94p. [2] Coun. Phys. Med. Amer. Med. Assoc., J.A.M.A. 137, 1600, 1948. [3] S.M. Michaelson. Proc. IEEE 60, 389, 1972. [4] U. S. Amer. Stand. Instit. USASI C95.1, New York, 1966. [5] Amer. Nat. Stand. Instit. Standard for the Safe Use of Lasers. ANSI Z/136.1, New York, 1972, 61p. [6] I. Matelsky. In: Industr. Hyg. Highlights. vol. 1, L.V. Cralley, G.D. Clayton (eds.), Pittsburgh, 1968, p. 140. [7] S.M. Michaelson. Amer. Indust. Hyg. Ass. J. 33, 156, 1972. [8] H.P. Schwan. IEEE Trans. Bio. Med. Eng. BME-19, 304, 1972. [9] F.H. Krusen et al (eds.). Handbook of Physical Medicine and Rehabilitation. W.B. Saunders, Philadelphia, 1971, 725p. [10] P.E. McGuff. Surgical Applications of Laser. Charles C. Thomas, Springfield, 1966, 200p. [11] S.M. Michaelson. In: Health Physics Society 7th Midyear Topical Symp. - Health Physics and the Healing Arts. San Juan, Puerto Rico, Dec. 1972, p. 44. [12] Laser Health Hazards Control. USAF, AFM-161-8, Wash., D.C., 1971, 45p. [13] I. Matelsky. The Non-Ionizing Ultraviolet Radiations. Amer. Industr. Hyg. Ass. Refresher Course, 1973. [14] K. Buettner, H.W. Rose. Sight Saving Rev. 23, 194, 1953. [15] W.J. Geeraets. Industr. Med. 39, 441, 1970. [16] E. Fischer, S. Solomon. In: S.H. Licht (ed.). Therapeutic Heat. E. Licht, New Haven, 1958, p.116. [17] H.F. Cook. J. Physiol. 118,1, 1952. [18] J.H. Jacobson et al. AMRL-TDR-62-96, 6570th Aerospace Med. Res. Lab., Wright-Patterson AFB, Ohio, 1962. [19] W. Robinson. Brit M.J. 2,381, 1907. [20] K.L. Dunn. Arch. Ind. Hyg. Occup. Med. 1,166, 1950. [21] D.G. Cogan et al. Arch. Ophthalmol. 47,55, 1952. [22] C.M. Edbrooke, C. Edwards. Arch. Occup. Hyg. 10,293, 1967. [23] D.L. Lloyd-Smith, K. Mendelssohn. Brit. M.J. #4559,975, 1948. [24] H.M. Whyte. Clin. Sci. 10,333, 1951. [25] L. Goldman. Biomedical Aspects of the Laser. Springer, New York, 1967. [26] W.F. Van Pelt et al. Laser Fundamentals and Experiments. U.S.D.HEW, PHS, BRH, Publ. No. BRH/SWRHL 70-1,1970. [27] T.L. Curtin, D.G. Boyden. Amer. J. Ophthal. 65,188, 1968. [28] A. Anne et al. In: M.F. Peyton (ed.). Biological Effects of Microwave Radiation . Plenum, New York, 1961, p.153. [29] C.C. Johnson, A.W. Guy. Proc. IEEE 60,692, 1972. [30] H.P. Schwan, K. Li. Proc. IRE 41,1735, 1953. [31] S.M. Michaelson. IEEE Trans. Microwave Theory and Techniques MTT-19,131, 1971. [32] S.M. Michaelson. Thermal effects of single and repeated exposures to microwaves - a review. International Symposium on Biologic Effects and Health Hazards of Microwave Radiation, Warsaw, 1973. [33] E. Hendler. In: J.D. Hardy (ed.). Thermal Problems in Aerospace Medicine. Unwin, Ltd., Surrey, 1968, p. 149. [34] E. Hendler et al . In: J.D. Hardy (ed.). Temperature Measurement and Control in Science and Industry. Pt. 3. Biology and Medicine. Rheinhold, New York, 1963, p.221. [35] R.L. Carpenter et al. Proc. 3rd Int. Conf. Med. Elect. (London) 3,401,1960. [36] H.A. Blair. Ann. N.Y. Acad. Sci. 114,150, 1964. [37] D.B. Williams et al. Arch. Ophthal. 54,863, 1955. [38] L. Birenbaum et al. IEEE Trans. Bio. Med. Eng. BME-16,7, 1969. [39] D.G. Cogan et al. Arch. Ind. Health 18,299, 1958. [40] M. Zaret. 39th Ann. Sci. Meeting Aerospace Med. Ass., Miami, 1968. [41] M. Zaret. 40th Ann. Sci. Meeting Aerospace Med. Ass., San Francisco, 1969. [42] F.G. Hirsch, J.T. Parker. Arch. Indust. Hyg. 6,512, 1952. [43] G.H. Kurz, R.B. Einaugler. Am. J. Ophthal. 66,866, 1968. [44] I.S. Shimkovich, V.G. Shilyayev. Vestn. Oftal. 72,12, 1959. [45] Z.V. Gordon. Biological Effect of Microwaves in Occupational Hygiene. Izdatel'stvo "Meditsina" Leningrad Otdelenie, 1966, 164p. [46] Yu.I. Novitsky et al. Radio Frequencies and Microwaves. Magnetic and Electrical Fields. NASA Tech. Trans. TT F-14,021, 1971. [47] L.R. Pinneo et al. The Neural Effects of Microwave Radiation. RADC-TDR-62-231, AD 722684, Arlington, 1962. [48] R.D. McAfee. IEEE Trans. Microwave Theory and Techniques MTT-19,251, 1971. [49] R.D. McAfee. Am. J. Physiol. 203,374, 1962. [50] I.R. Petrov (ed.). Influence of Microwave Radiation on the Organism of Man and Animals. Meditsine, Leningrad, 1970. [51] A.S. Presman. Electromagnetic Fields and Life. Izd-vo Nauka, Moscow, 1968. [52] Yu.A. Osipov. Occupational Hygiene and the Effect of Radio-Frequency Electromagnetic Fields on Workers. Izd. Meditsina, Leningrad, 1965. [53] J. Pazderova. Prac. Lek. 20,447, 1968. [54] C.H. Dodge. In: Symp. Proc. (PB 193-898). USDHEW, PHS, BRH, Rockville, 1970. [55] H.J. Magnuson et al. Amer. Indust. Hyg. Ass. J. 25,185, 1964.