

Table 4. Ocular changes with timolol pretreatment.

Power density avg. (mW/cm ²)	Specific absorption rate (W/kg)	Mean $\pm$ standard error of the mean	
		Endothelial damage ^a	Vascular permeability ^a
0	0	1.0 $\pm$ 0.00 (9)	1.0 $\pm$ 0.00 (9)
0.2	0.05	1.2 $\pm$ 0.17 (5)	1.0 $\pm$ 0.00 (5)
1.0	0.26	3.3 $\pm$ 0.25 (7) ^b	2.9 $\pm$ 0.31 (7) ^b

^aNumber of eyes examined appears in parentheses.^bProbability <0.001 when compared with sham-exposed subjects.

performed by Gerard Luty of the Wilmer Institute and D. Scott McLeod of APL, demonstrated significant photoreceptor damage and retinal detachment. A clear cause-effect relationship explaining this damage could not be established, because of the mixed exposure protocol of the primate subjects and the use of halothane gas anesthesia during microwave exposure. Researchers also questioned the possibility of pre-existing retinal pathology contributing to the effects observed. A new protocol was therefore established to address these concerns.

To eliminate possible pre-existing retinal pathology, ocular electrophysiologic testing was performed before exposing the primates to microwaves. All measurements and analyses were under the direction of Dr. Mary John-

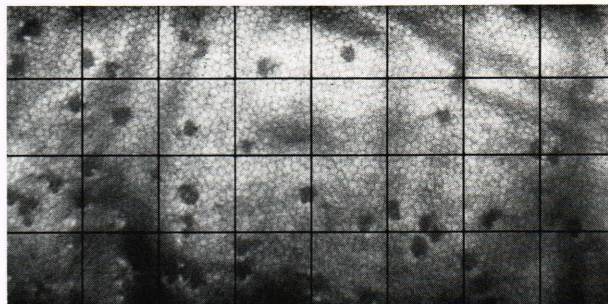
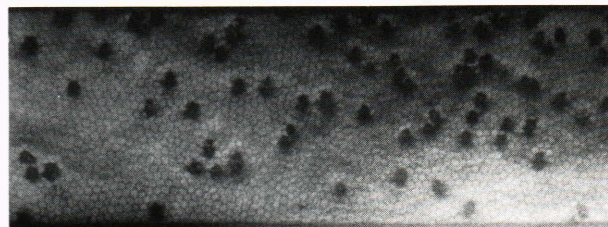
A**B**

Figure 9. Specular micrographs ($\times 120$) of the primate corneal endothelium. **A.** Primate corneal endothelium showing a minor number of lesions twenty-four hours postexposure to pulsed microwaves (10 mW/cm²) without drug pretreatment ($\times 120$). **B.** Primate corneal endothelium showing a moderate number of lesions twenty-four hours postexposure to pulsed microwaves (10 mW/cm²) after pretreatment with timolol maleate ($\times 120$).

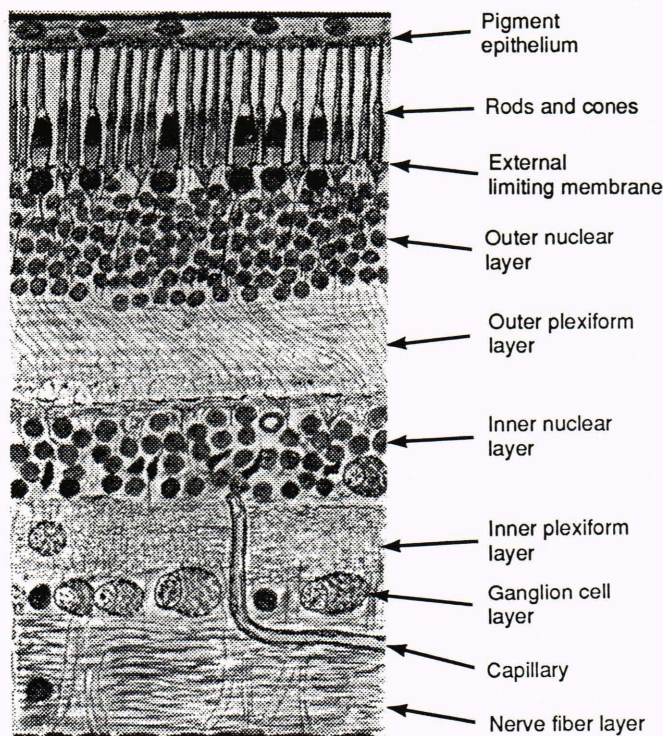
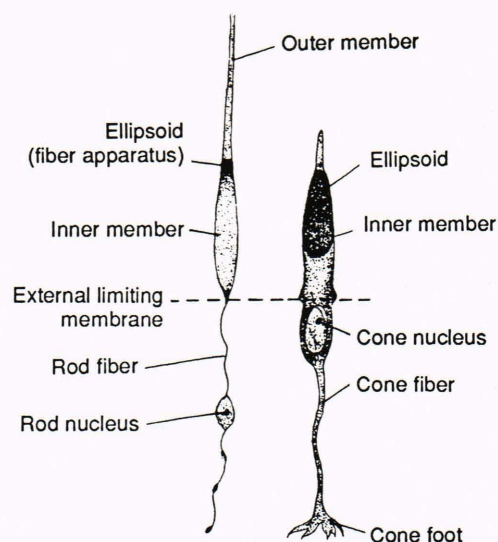
A**B**

Figure 10. Illustration of the retina of the eye. **A.** Layers of the retina. **B.** The retinal photoreceptors. (Figures reprinted, with permission, from Vaughn, D., and Asbury, T., *General Ophthalmology*, 8th Ed., © 1977 by Lange Medical Publications, Los Altos, Calif., p. 116 and 117, respectively.)

son of the Wilmer Institute. Each primate was again tested after microwave exposure to measure possible changes in retinal function. Electroretinography (ERG) was used to measure the transient action potentials initiated by a change in the light energy falling on the photoreceptors of the retina. It yields information regarding the earliest stages of the visual process. The basic clinical ERG protocol entails a scotopic test (rod photoreceptor response) and a 30-Hz flicker test (cone photoreceptor response).

The exposure protocol called for a four-hour exposure on three consecutive days for three consecutive weeks. Microwave exposure parameters were 1.25 GHz, pulsed ($0.5 \mu\text{s}$, 16 pps), 1-MW peak, producing an SAR of 4.0 W/kg. The primates were restrained but not anesthetized during microwave exposure (Fig. 11). Just before the first exposure and twenty-four hours after the ninth and last exposure, ERG was performed. Figure 12 shows "before and after" ERG's; a 60% reduction in the scotopic or rod response and a 90% reduction in the 30-Hz flicker cone response are seen.

Following the three-week exposure protocol and ERG testing, the primates were euthanized and the eyes prepared for light microscopy. Figure 13 shows the histopathology observed in an exposed primate. The effects include photoreceptors with degenerative changes both in the nuclei and in the outer and inner segments. These changes correlate with the ERG findings demonstrated in Figure 12.

SUMMARY

The experiments reported herein demonstrate that exposure to relatively low levels of microwave radiation can result in significant ocular changes in the nonhuman primate. These changes range from cellular disruption to altered visual function. At 10 mW/cm^2 , we have observed corneal endothelial lesions, increased iris vascular permeability, and degenerative changes in the cells of the iris and the retina. When the primates were pretreated with timolol maleate, a ten-fold reduction in the threshold microwave power density that produces these effects was observed. At an exposure level of 10 mW/cm^2 , ERG has demonstrated a significant decrease in visual function. Measurements of the SAR of these low-level exposures to microwaves indicate strongly that significant tissue heating is not the mechanism responsible for the exhibited changes.

In the United States, several permissible exposure standards for RF radiation have been promulgated and revised over the years. The current frequency-dependent American National Standards Institute guideline¹³ for occupational and general population exposure permits unlimited exposure to 1 mW/cm^2 at frequencies from 30 to 1500 MHz, and exposure to 5 mW/cm^2 at frequencies above 1500 MHz. The most recent standard, Institute of Electrical and Electronic Engineers C95.1,¹⁴ permits unlimited exposure to 10 mW/cm^2 in controlled environments for frequencies above 1.5 GHz. The American Conference of Governmental and Industrial Hygienists¹⁵ recommendation permits occupational exposures of 10 mW/cm^2 , whereas the National Council on Radiation Protection and Measurements¹⁶ recommends that occu-

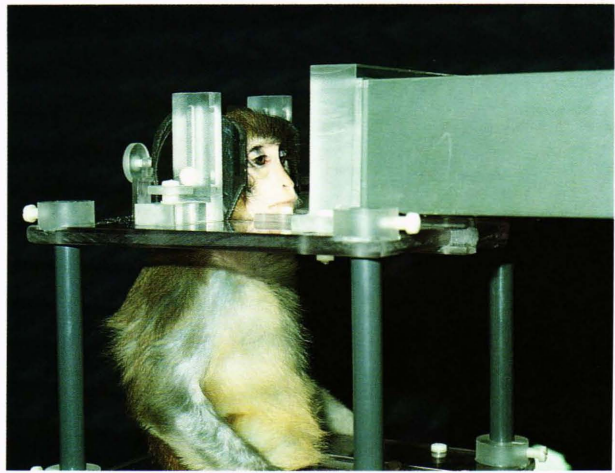


Figure 11. Restrained, nonanesthetized primate positioned for ocular irradiation.

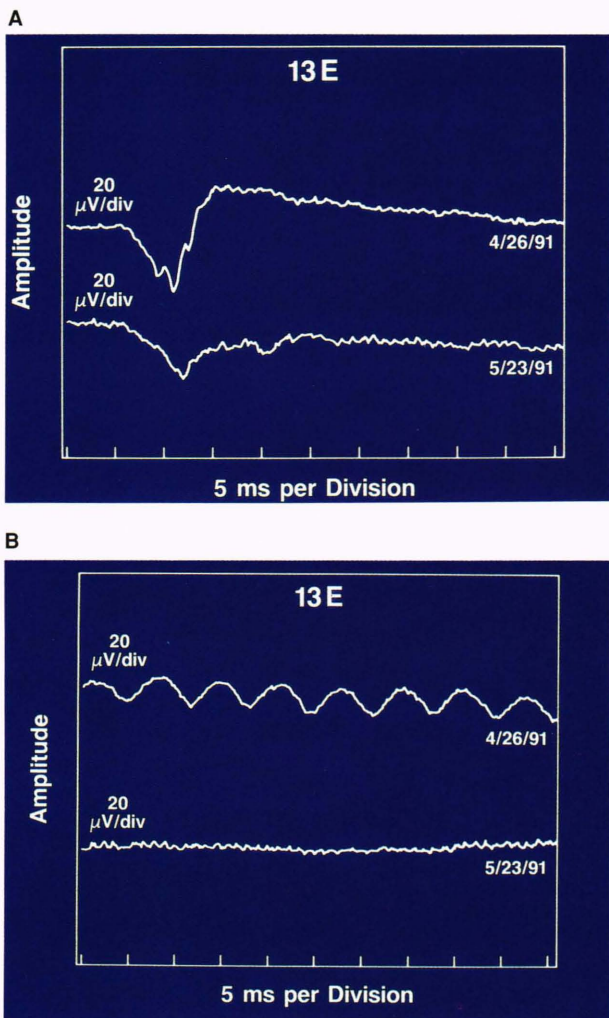


Figure 12. Electrophysiologic measurement of the primate retina (from primate subject 13E) before and after pulsed microwave exposure. **A.** Scotopic electroretinogram demonstrating rod response before and after microwave exposure (60% reduction). **B.** 30-Hz flicker electroretinogram demonstrating cone response before and after microwave exposure (90% reduction). Signal amplitude is the signal produced by the retina before (upper curve) and after (lower curve) microwave exposure.

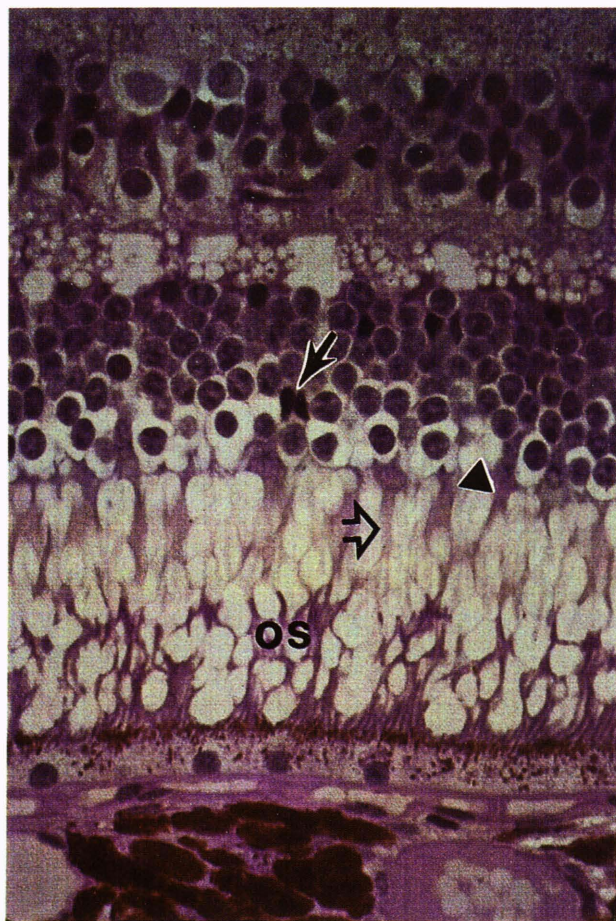


Figure 13. Macular region of the retina from a microwave-exposed primate. Degeneration of the photoreceptor nuclei (arrow) and disappearance of nuclei (arrowhead); degeneration of outer segments (OS) and inner segments (open arrow) is evident.

pational exposure not exceed 1 mW/cm^2 . The data presented in our study suggest that ocular effects may actually occur at exposure levels close to or below those recommended by these safety guidelines, particularly when medications such as timolol are being used. At the very least, our observations indicate that the current safety guidelines lack an inherent safety factor, which heretofore has been considered to be an important aspect of any safety guideline.

On the basis of some of our early histological findings, it seemed likely that cellular change could occur at exposure levels below 10 mW/cm^2 in either sensitive individuals or under prolonged exposure conditions. In recognition of this possibility, and to provide a sufficient safety factor between the level where biological effects occur and levels recommended for human exposure, APL adopted its own safety standard¹⁷ for microwave exposure—an exposure limit of 0.1 mW/cm^2 for all employees, regardless of whether they work with microwave-generating equipment or not.

The results observed in our studies were obtained under specific laboratory conditions, including prolonged

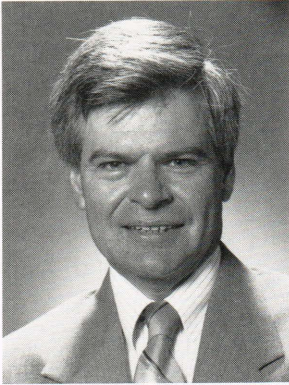
microwave exposure and the use of anesthesia. These exposure sessions do not necessarily duplicate conditions found in the environment. The ocular effects obtained, however, were demonstrated in a nonhuman primate model, which is anatomically and physiologically similar to the human eye. The data suggest strongly that individuals could be subject to ocular effects from previously unsuspected low-level microwave exposure with low average absorbed dose rates. Attempts to extrapolate the data beyond the actual conditions of the experimental paradigm should be pursued with appropriate caution.

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