

DO MICROWAVES ALTER NERVOUS SYSTEM STRUCTURE?*

Ernest N. Albert and Mark DeSantis

*Departments of Anatomy
The George Washington University School of Medicine and Health Sciences
and Georgetown University School of Medicine and Dentistry
Washington, D.C. 20037*

Electromagnetic radiation in the superhigh frequency or microwave range has been reported to alter central and peripheral nervous system structure. Structural modifications in both neurons and glia have been described.^{1-3,5} Vascular changes also noted include hemorrhage, congestion (hyperemia), and perivascular edema.^{1,5} Others, however, have reported an absence of histologic changes after microwave irradiation.⁴

Short-duration (less than 1 hr) single exposures to continuous wave electromagnetic radiation at power densities from 20 to 100 mW/cm² and wavelengths of about 10 cm (3-12.2 cm) alter neuronal structure. Changes reported include tigrolysis and cytoplasmic vacuolization in hypothalamic neurons,¹ pyknosis of cerebral, hippocampal, deep cerebellar, and brainstem neurons,⁵ and tigrolysis, nuclear eccentricity, and karyocytolysis in spinal ganglion cells.¹ Oligodendroglia have been described as edematous after exposure under similar conditions.¹ Fragmentation and increased argyrophilia of cutaneous sensory nerve fibers have been detected after exposure to microwaves of millimeter wavelength.¹ In general, pulsed waves are apparently more effective in inducing central nervous system alterations than continuous waves, and longer wavelengths of radiation are more effective than shorter wavelengths.³

Long-term (minimum of 1 month) repeated exposure to continuous wave electromagnetic radiation at a power density of 10 mW/cm² and a wavelength of about 10 cm (3-12.2 cm) has also been reported to alter neuronal structure.¹ These values are thought to be below the threshold level for thermal effects on animal tissues.²

Electrophysiologic, behavioral, and clinical evidence suggests that microwave exposure may be deleterious and result in functional pathologic states.^{3,4,10} Further documentation of these morphologic changes is necessary, as is a better estimate of the anatomic extent and factors responsible for the alterations reported.

MATERIALS AND METHODS

Examination of the central nervous system of 60 adult male and female Chinese hamsters form the basis of this communication. Irradiations were performed in an anechoic chamber, 16 ft wide by 16 ft high by 32 ft long, by an HP 8 690B oscillator with an HP 8699B plug-in head.[†] Power densities were determined in free space by means of the identical horn technique. These measurements were made 10 ft from

*Supported by Department of Health, Education and Welfare Grant ROIFD 00649, the Federal Drug Administration and by the Bureau of Medicine and Surgery, Department of the Navy.

[†]The anechoic chamber was located at The Walter Reed Army Research Facilities at Forest Glen, Md.

the horn, and intensity fluctuations of $\pm 1.5 \text{ mW/cm}^2$ were calculated over a radius of 9 in. from the axis. Amply ventilated Plexiglas® chambers that contained one Chinese hamster each were placed in the probed area. All animals were exposed to 2450 MHz electromagnetic radiation at either 50 or 25 mW/cm² power density. Exposure time varied from 30 min to 24 hr for the greater intensity and was 22 days (14 hr/day) for the lesser intensity. During the 14 hr/day long-term exposures, control and experimental animals were given one pellet of chow and 5 ml of water each day. After exposure, both groups were provided with ample food and water until the next exposure time. Animals were sacrificed immediately after either the prescribed exposure period or a recovery period that ranged from 1 to 2 weeks following the last treatment. Appropriate controls were matched with experimental animals, and both groups were treated identically, except for exposure to microwaves.

All animals were anesthetized with pentobarbital and perfused with fixatives via the ascending aorta. Animals processed for light microscopy were perfused with 10% buffered formalin; those processed for electron microscopy were fixed with glutaraldehyde-paraformaldehyde solutions, according to the method of Karnovsky.⁶ Tissues prepared for electron microscopy were postfixed in 2% buffered osmium tetroxide, dehydrated through an ascending series of ethanol, and embedded in English Araldite® or Epon® 812.⁷ Tissues prepared for light microscopy were either embedded in paraffin or frozen sections were prepared. Sections from paraffin-embedded tissue were cut at 10 μm and stained with cresyl violet, thionin, or hematoxylin and eosin. Two animals were fixed with a potassium dichromate solution and stained by the Marchi technique for degenerating myelin.⁹ In every instance where light microscopy was employed, tissue sections from an experimental and a control animal were mounted on the same slide for the staining procedures. Frozen sections were stained by the Fink-Heimer⁸ technique for degenerating axons. A special control was used for correctly applying and interpreting this technique. One eye was surgically removed from a hamster; after a 9-day survival period, the animal was sacrificed and routinely processed. The known lesion in this animal served as a positive control for tissues from irradiated animals stained by the Fink-Heimer technique.

OBSERVATIONS

Animals exposed for 30 min or longer (up to 24 hr) to microwaves at a frequency of 2450 MHz and a 50 mW/cm² power density exhibited morphologic changes in the cytoplasm of certain neuron cell bodies. At the light microscopic level, the cytoplasm of hypothalamic neurons was markedly pale and displayed vacuolization and chromatolysis (FIGURE 1). The cytoplasm often appeared frothy, whereas the nuclear structure remained unaltered. Vacuoles could be seen preferentially distributed in the cytoplasm (FIGURE 1). In rare instances, vacuoles were detected within nuclei. Vacuoles were not present in the cytoplasm or nuclei of glial cells. The preferential appearance of vacuoles in the cytoplasm is vividly seen in Epon-embedded sections (FIGURE 2) in hypothalamic neurons of animals exposed to 25 mW/cm² power density for 22 days.

In comparing the morphologic aspects of hypothalamic neurons from animals sacrificed immediately after exposure, at a 50 mW/cm² power density, with that of those sacrificed 6–10 days after treatment, it appeared that cytopathologic phenomena were more numerous in the former (cf. FIGURES 1 and 3). The neurons in FIGURE 3 display vacuolization and chromatolysis but not to as great an extent as that seen in FIGURE 1.

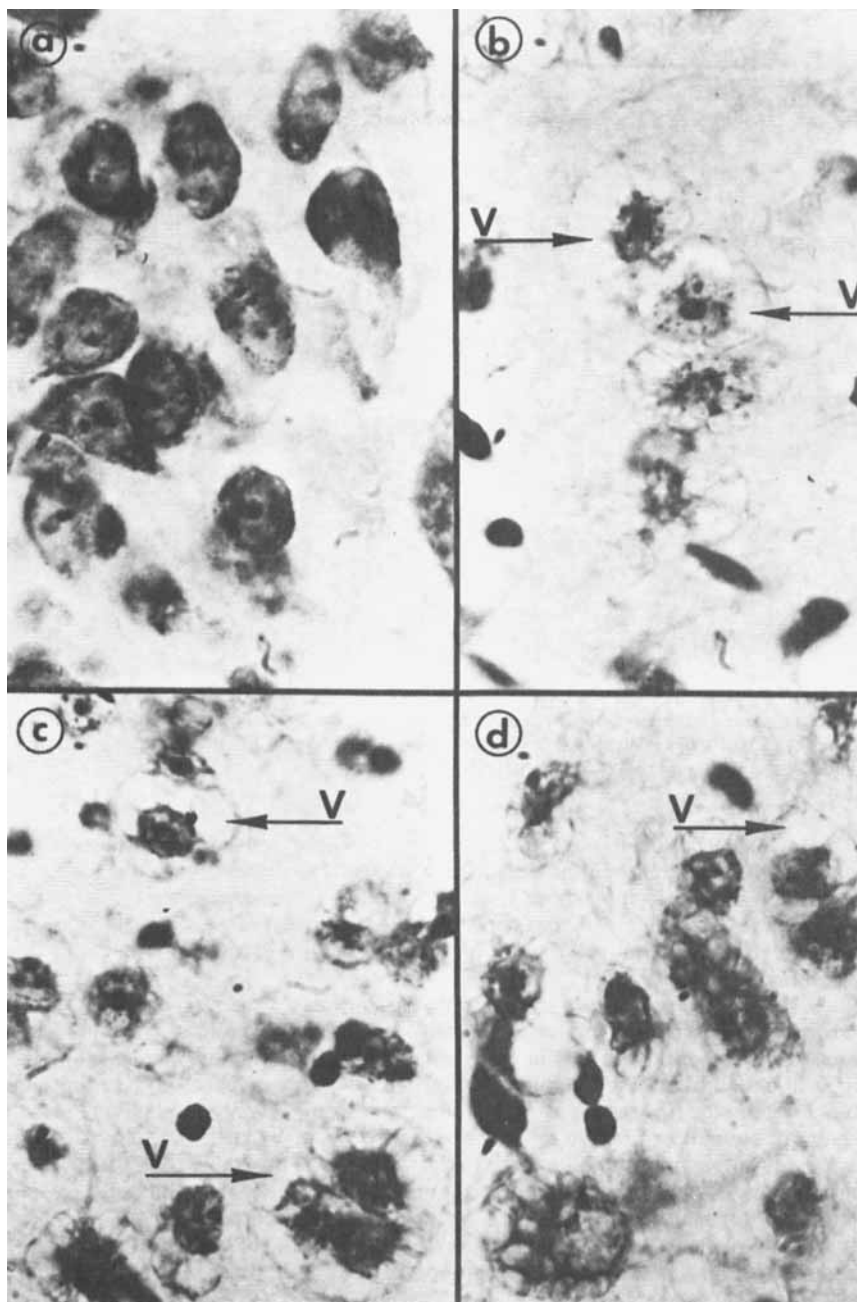


FIGURE 1. Hypothalamic neurons of control (a) and experimental (b,c,d) animals. Experimentals were exposed to a power density of 50 mW/cm^2 for 24 hr and sacrificed immediately after exposure. Note the basophilic-staining cytoplasm of control (a). Cytoplasm of experimental (b,c,d) appears pale and contains large and small vacuoles (V). Embedded in paraffin and stained with thionin.

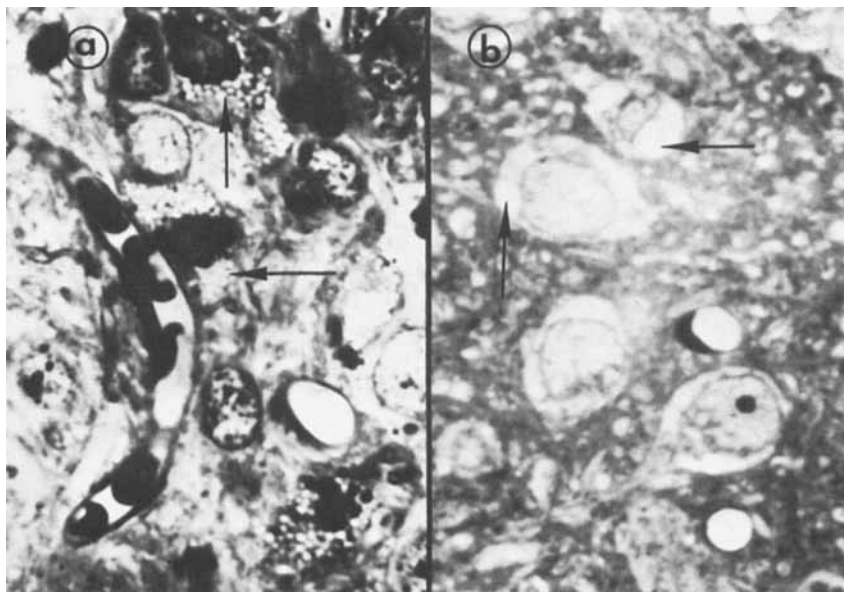


FIGURE 2. Hypothalamic neurons of experimental Chinese hamsters exposed to a 25 mW/cm² intensity for 22 days. Note the large vacuoles in the cytoplasm (arrows in b) and the appearance of multiple smaller vacuoles (arrows in a). Embedded in Epon and stained with toluidine blue.

Electron microscopic observations of hypothalamic neurons from animals exposed to a 25 mW/cm² power density also provided evidence of vacuolization and chromatolysis. The cytoplasm of these neurons appeared to have very little rough endoplasmic reticulum and few polyribosomes (FIGURES 4-6). Pockets of fragmented cytoplasm (FIGURES 5 & 6) may be a correlate of vacuoles seen with the light microscope.

At the specific microwave irradiation conditions described above, vacuolization and frothy-appearing cytoplasm of neurons were also observed in the subthalamus (FIGURE 7). However, neurons in areas adjacent to the thalamus, namely, the ventrobasal complex and the lateral geniculate nucleus, appeared the same as those of control animals (FIGURE 8). Similarly, neurons in other central nervous system regions of animals exposed to microwaves at 50 or 25 mW/cm² power densities, for various durations, appeared unaltered relative to the comparable tissue from control animals. No changes were detected in cell bodies of Purkinje cells of the cerebellum, motor neurons in the ventral horn of the spinal cord (FIGURE 9), the mesencephalic nucleus of the trigeminal nerve (FIGURE 10), or the hippocampal pyramidal neurons. Neurons of these groups have not yet been examined with the electron microscope.

Electron microscopic examination of the hypothalamic neuropil from animals irradiated at 50 mW/cm² for 24 hr revealed some structural peculiarities in dendritic processes. The most pronounced changes in dendrites of experimental animals were the appearance of myelin figures, swollen smooth membrane structures, and loss of neurotubules (cf. FIGURES 11-13). The dendritic processes generally ap-

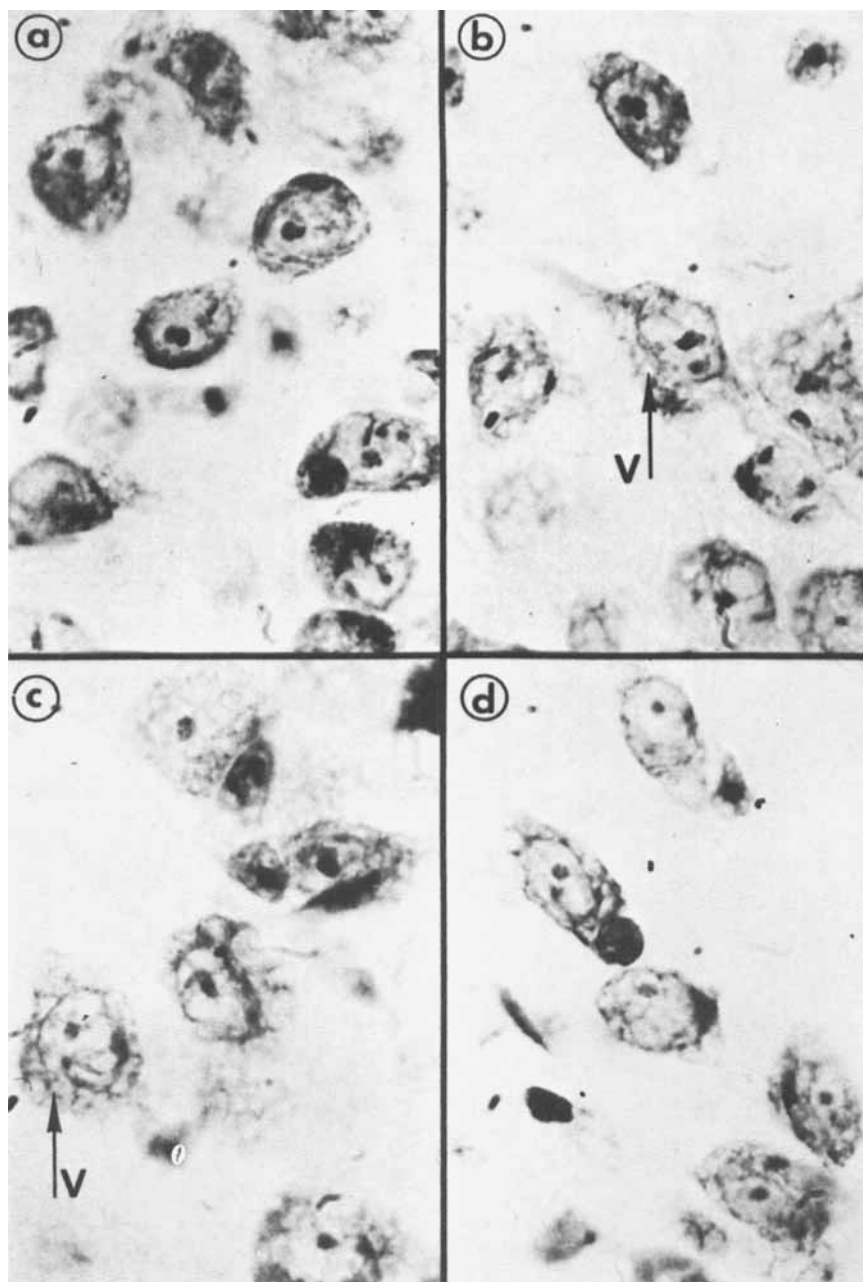


FIGURE 3. Hypothalamic neurons from control (a) and experimental animals (b,c,d). Experimentals were exposed to a 50 mW/cm^2 power density for 24 hr and allowed to recover for 6 days prior to sacrifice. Note the vacuolization (V) in experimentals (b,c,d); compare with control (a). Embedded in paraffin and stained with thionin.

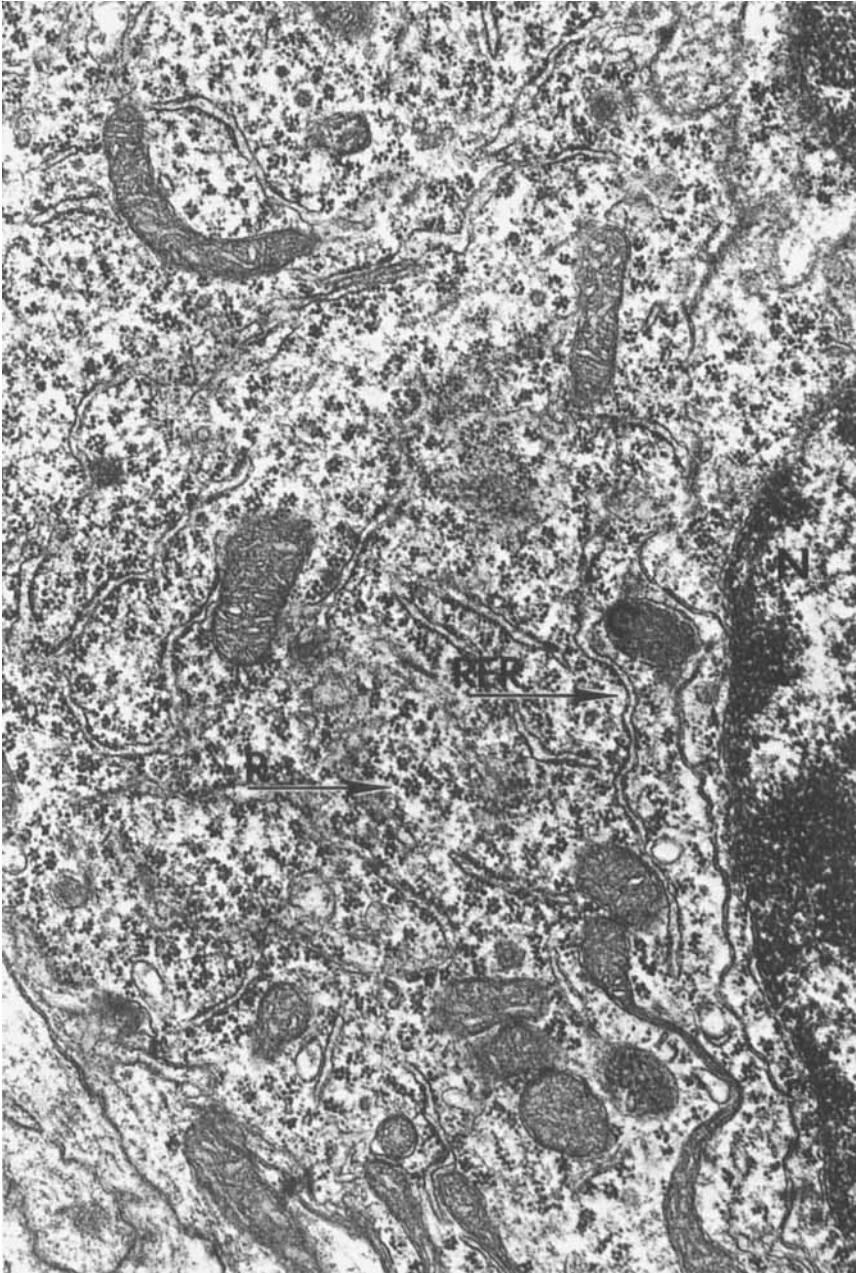


FIGURE 4. Electron micrograph of a hypothalamic neuron from a control animal. Note the abundance of free ribosomes (R) and rough endoplasmic reticulum (RER) in the cytoplasm (N, nucleus). Stained with uranyl acetate and lead citrate.

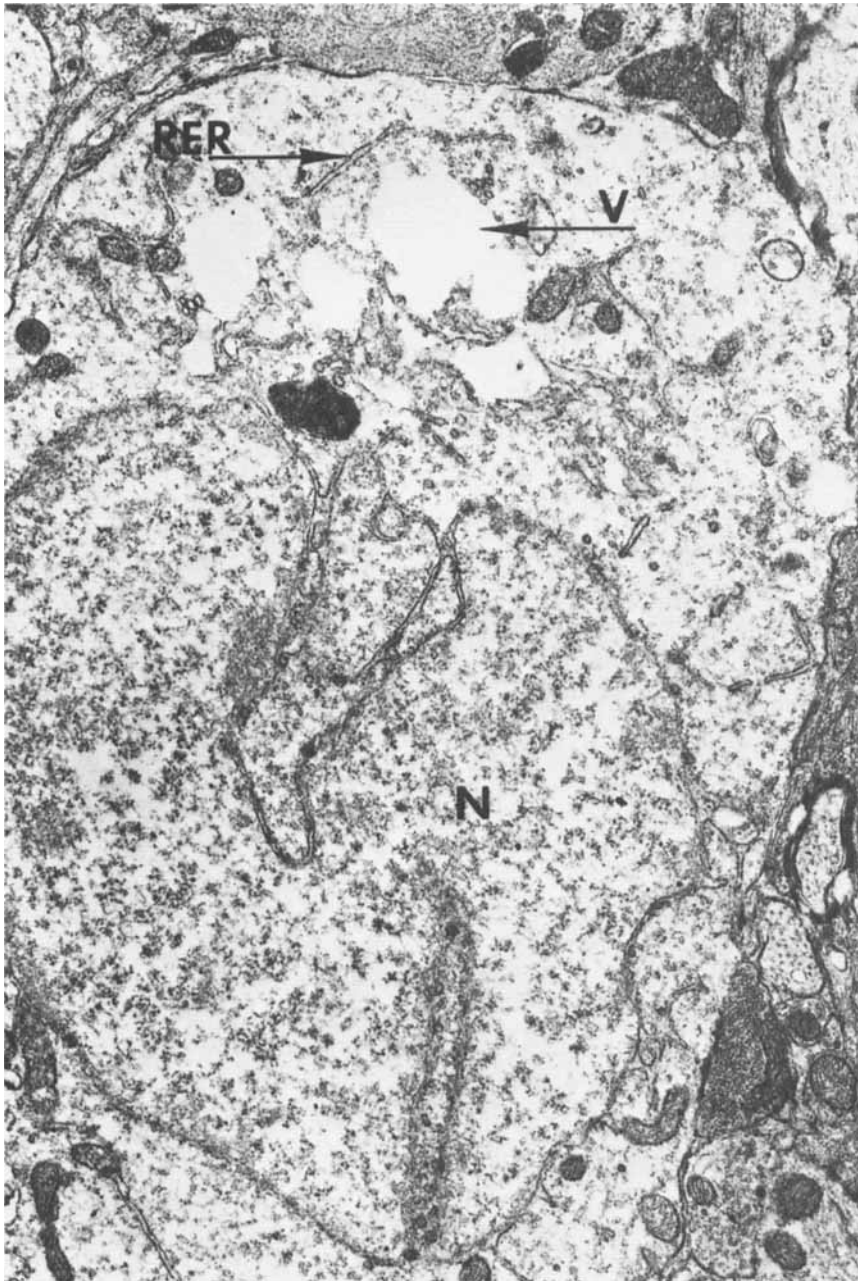


FIGURE 5. Portions of neurons from the hypothalamus of Chinese hamsters exposed to a 25 mW/cm² power density for 22 days. Neurons are similar to those in FIGURE 2. Note the presence of vacuoles (V) confined to the cytoplasm and the paucity of free ribosomes and rough endoplasmic reticulum (RER). Compare with FIGURE 4. M, mitochondrion; N, nucleus. Stained with uranyl acetate and lead citrate.

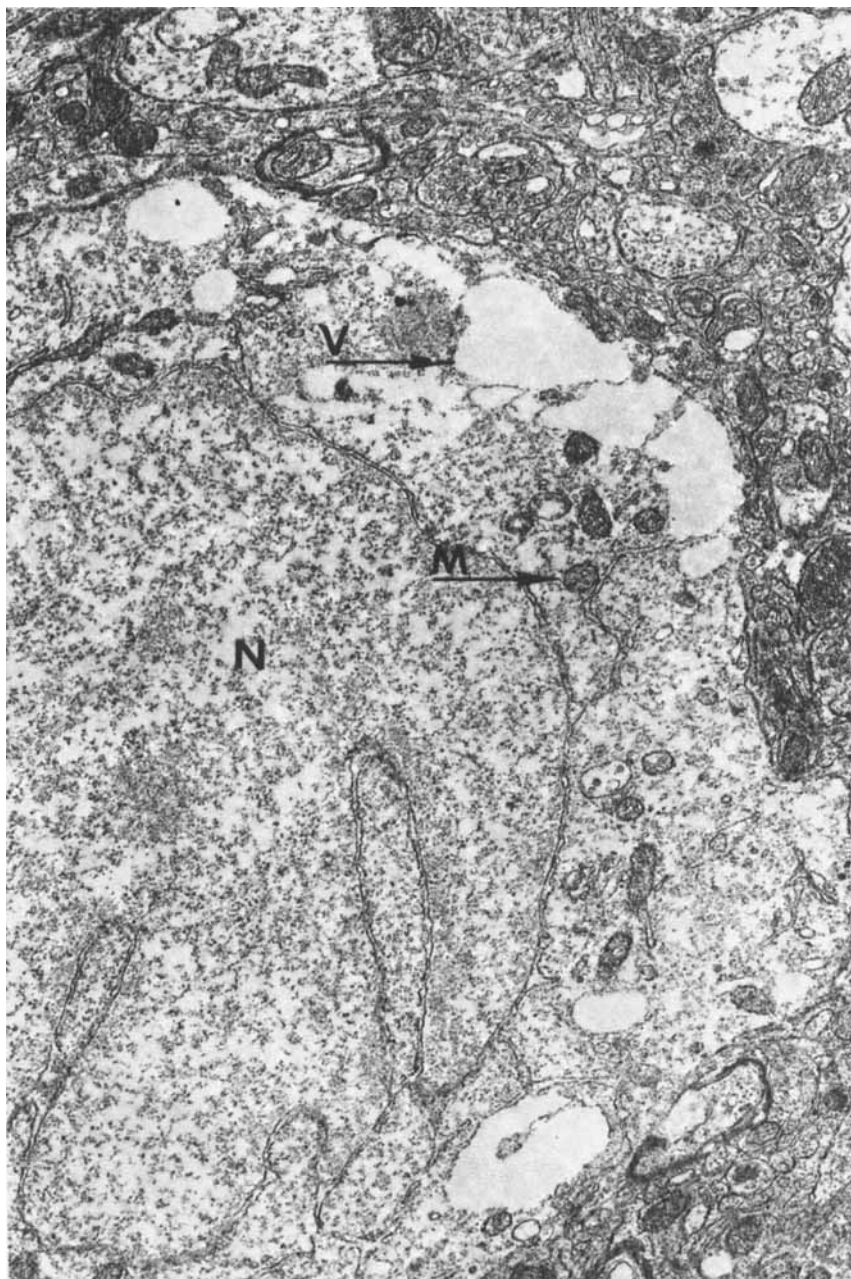


FIGURE 6. See FIGURE 5 legend for explanation.

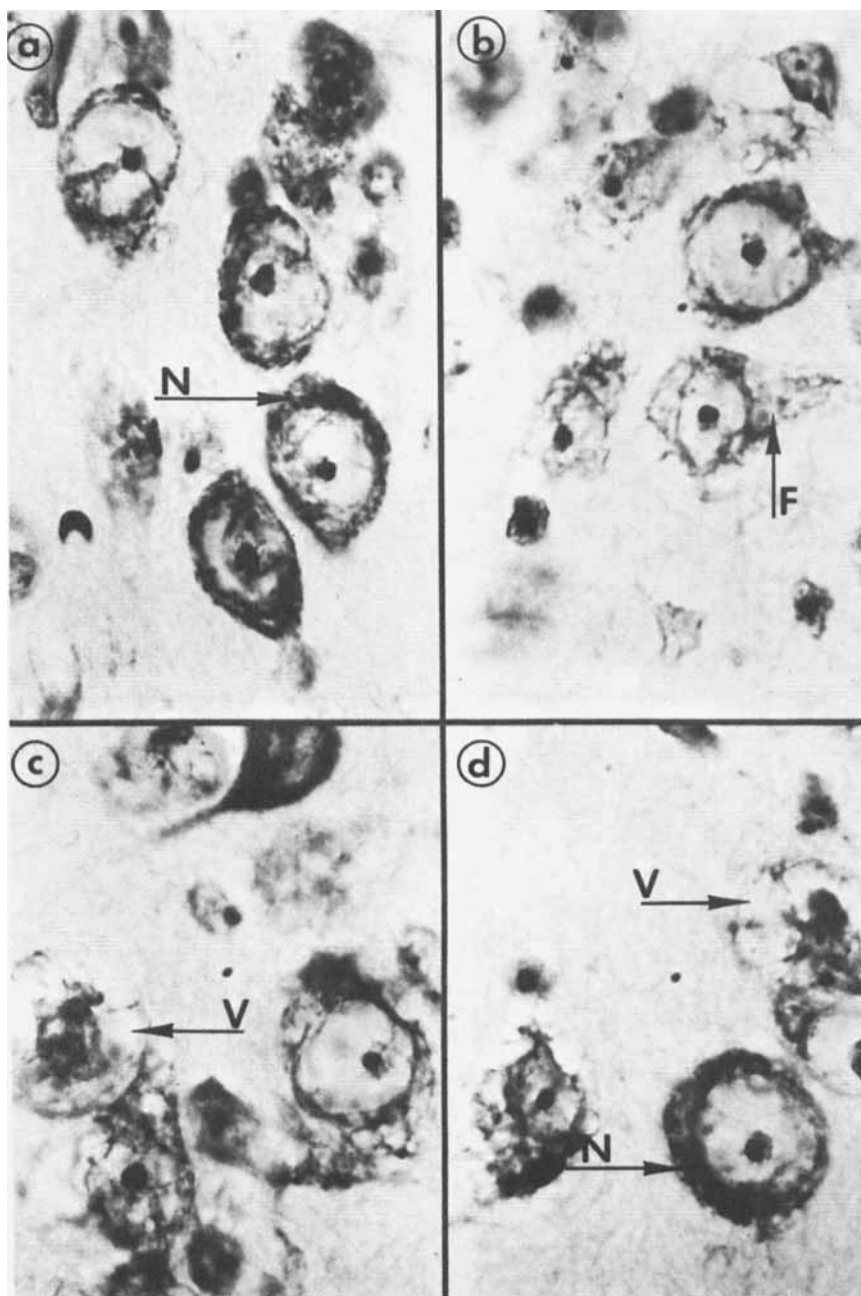


FIGURE 7. Neurons from the subthalamus of control (a) and experimental (b,c,d) animals irradiated with a 50 mW/cm^2 power density for 24 hr. Note the frothy appearance (F) of cytoplasm and vacuoles (V). Also note the presence of a normal appearing neuron (N) in d.

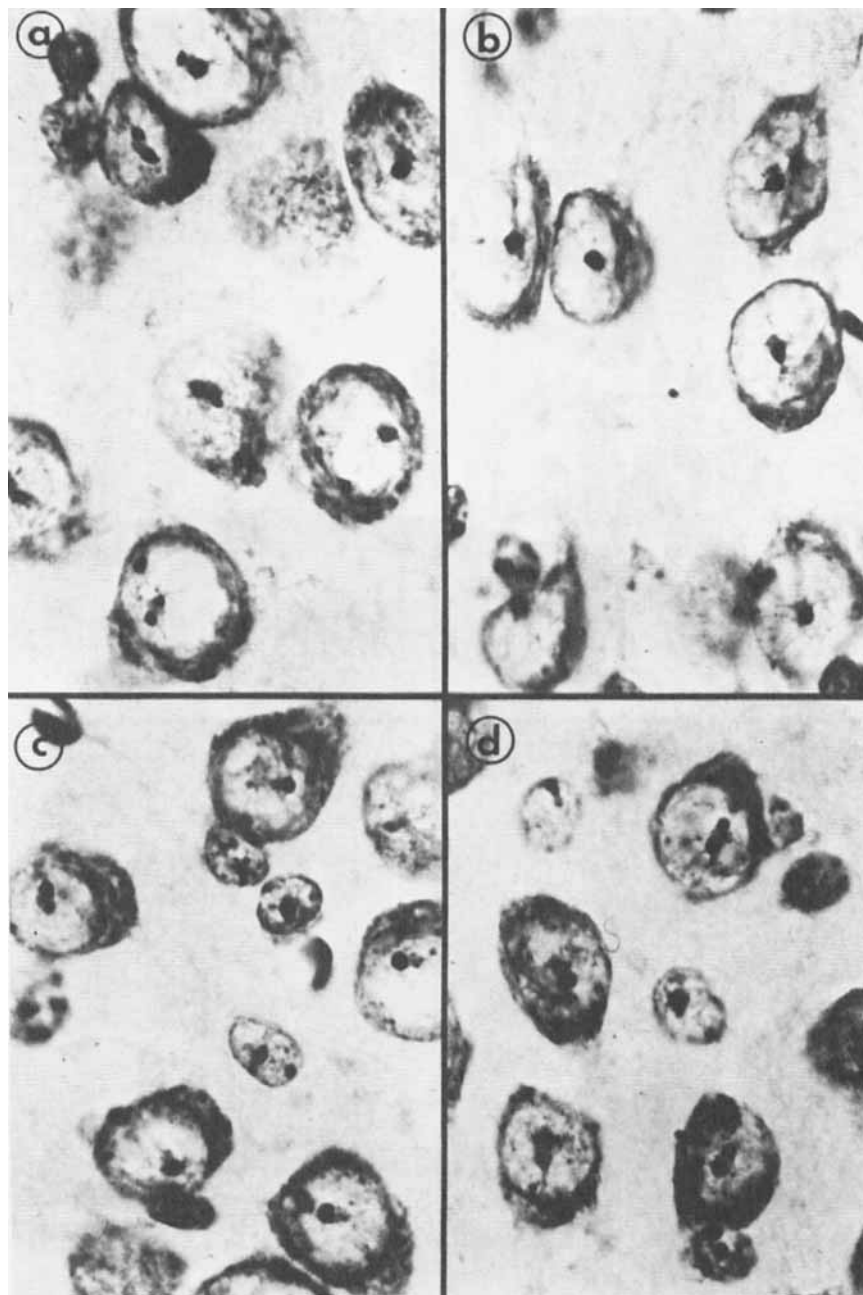


FIGURE 8. Neurons from the ventrobasal complex (a,b) and lateral geniculate (c,d) nucleus of the thalamus from control (a,c) and experimental (b,d) animals. Experimentals are same as in FIGURE 7. Unlike the subthalamic neurons, these thalamic neurons appear unaffected by microwave irradiation.

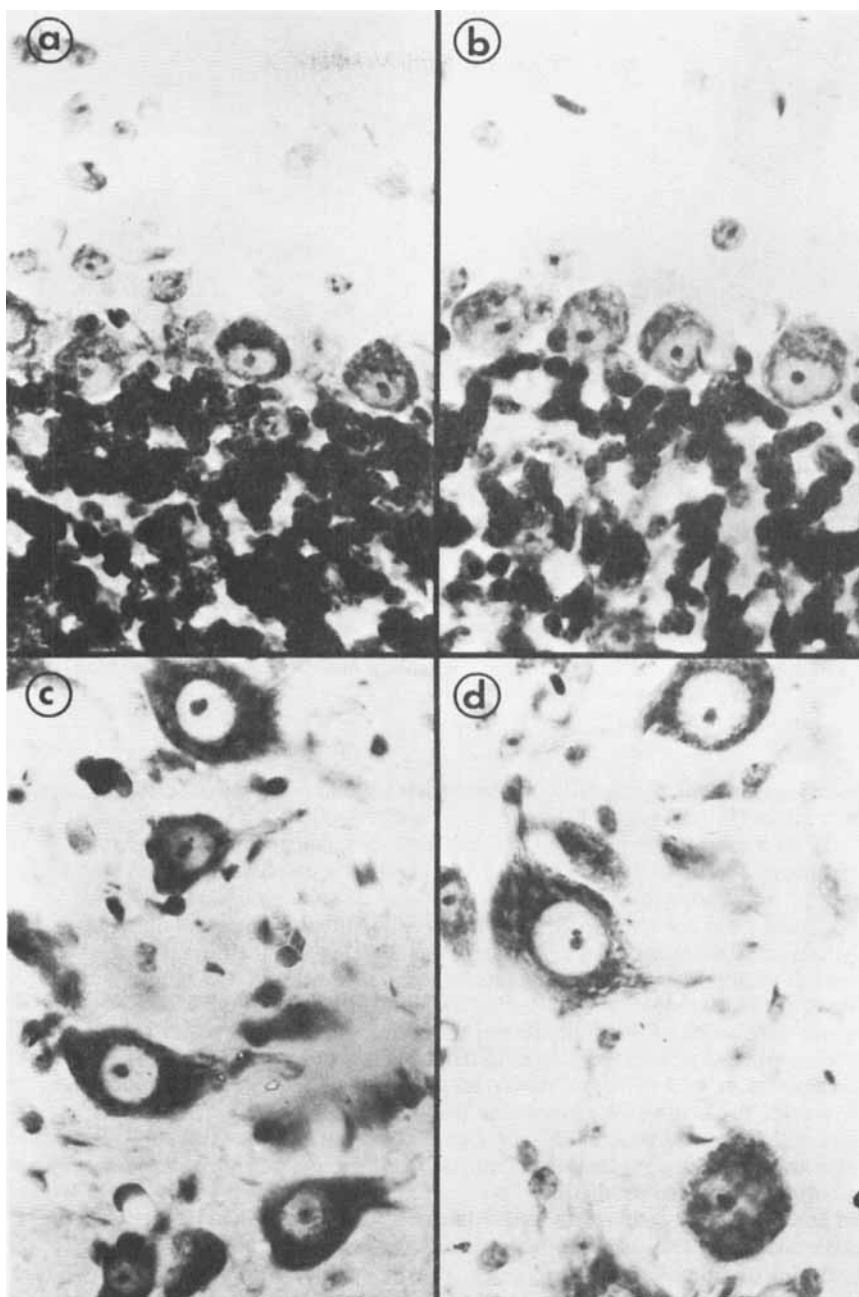


FIGURE 9. Cerebellar Purkinje cells (a,b) and ventral horn motor neurons (c,d) from control (a,c) and experimental (b,d) animals exposed to a 50 mW/cm² intensity for 24 hr. No morphologic differences were apparent between controls and experimental.

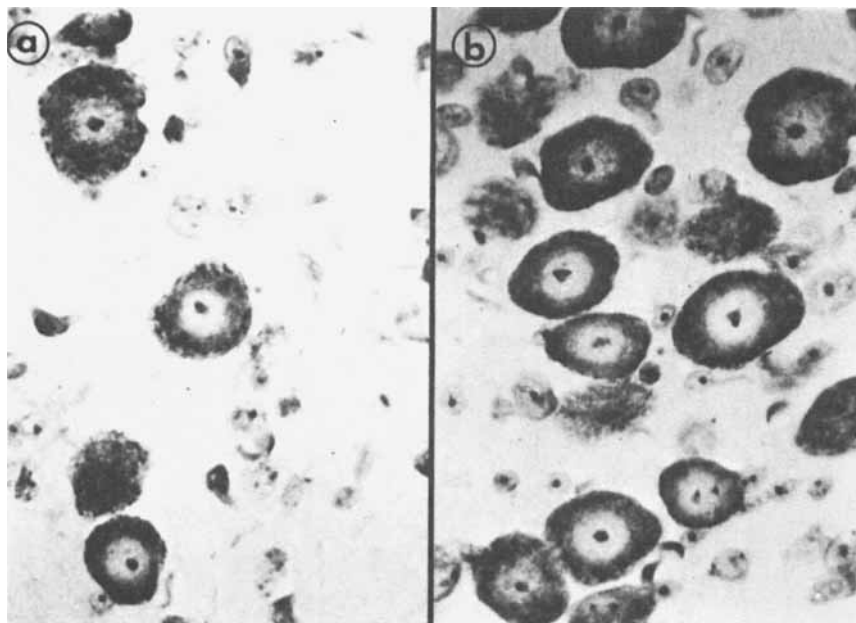


FIGURE 10. Neurons of the trigeminal mesencephalic nucleus of control (a) appeared similar to those of an animal exposed to microwaves (b).

peared washed out. Conversely, myelinated axons and presynaptic terminals seemed to be intact (FIGURES 12 & 13).

In an attempt to explore the possibility of axonal degeneration after microwave treatment, the Fink-Heimer staining technique was employed. Frozen sections of brains from irradiated and control animals were stained simultaneously.

Tissue from the enucleated control animal exhibited degenerating axons in the optic tract and degenerated axons and their terminals in the lateral geniculate nucleus (FIGURE 14). Degenerating axons were also seen in the hypothalamus bilaterally (FIGURE 15). In contrast to the control observations used to check the proper application of the Fink-Heimer technique, degenerating axons were observed in the pontine tegmentum and hypothalamus (FIGURE 15) of animals irradiated with microwaves at a 50 mW/cm² power density for 2 hr and allowed to recover for 10 days prior to sacrifice. We are aware that the Fink-Heimer technique was not applied in the classical manner and that vagaries may occur when this stain is used, yet the above evidence does tentatively indicate that some axonal degeneration may occur after microwave irradiation. The degenerated axons were widely scattered and did not seem to be confined to well-delineated pathways or distinct nuclear groups, as is customarily the case in animals with known lesions.

Brains of animals exposed to power densities of 25 or 50 mW/cm² for 22 days and 24 hours, respectively, were stained with the Marchi technique for myelin degeneration. It appeared that microwaves at the treatment conditions described above do not affect myelin, as determined by this method.

There was no evidence of gliosis, hemorrhage, or perivascular edema in any of

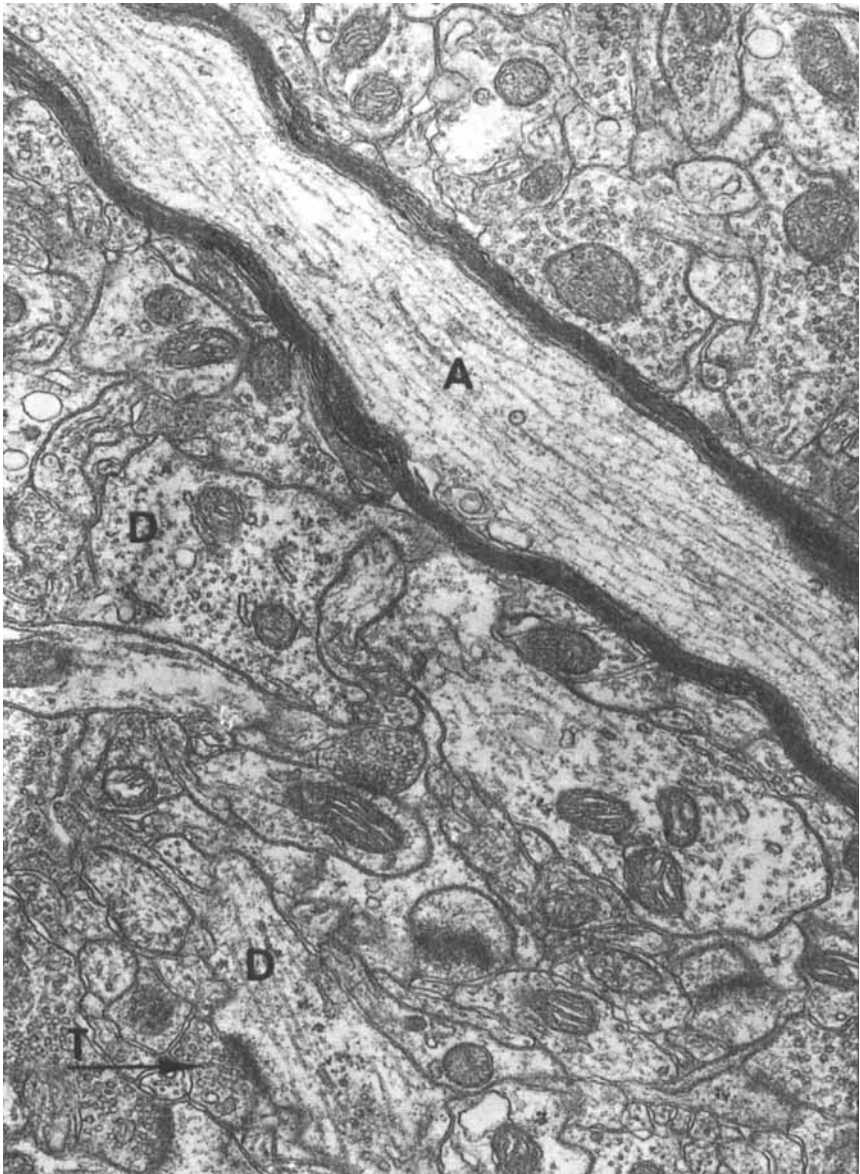


FIGURE 11. Electron micrograph of hypothalamic neuropil from a control animal. Note the morphologic appearance of the axon (A), dendrites (D), and synaptic terminal (T). The neurotubules, neurofilaments, mitochondria, and other structures appear normal. Stained with uranyl acetate and lead citrate.

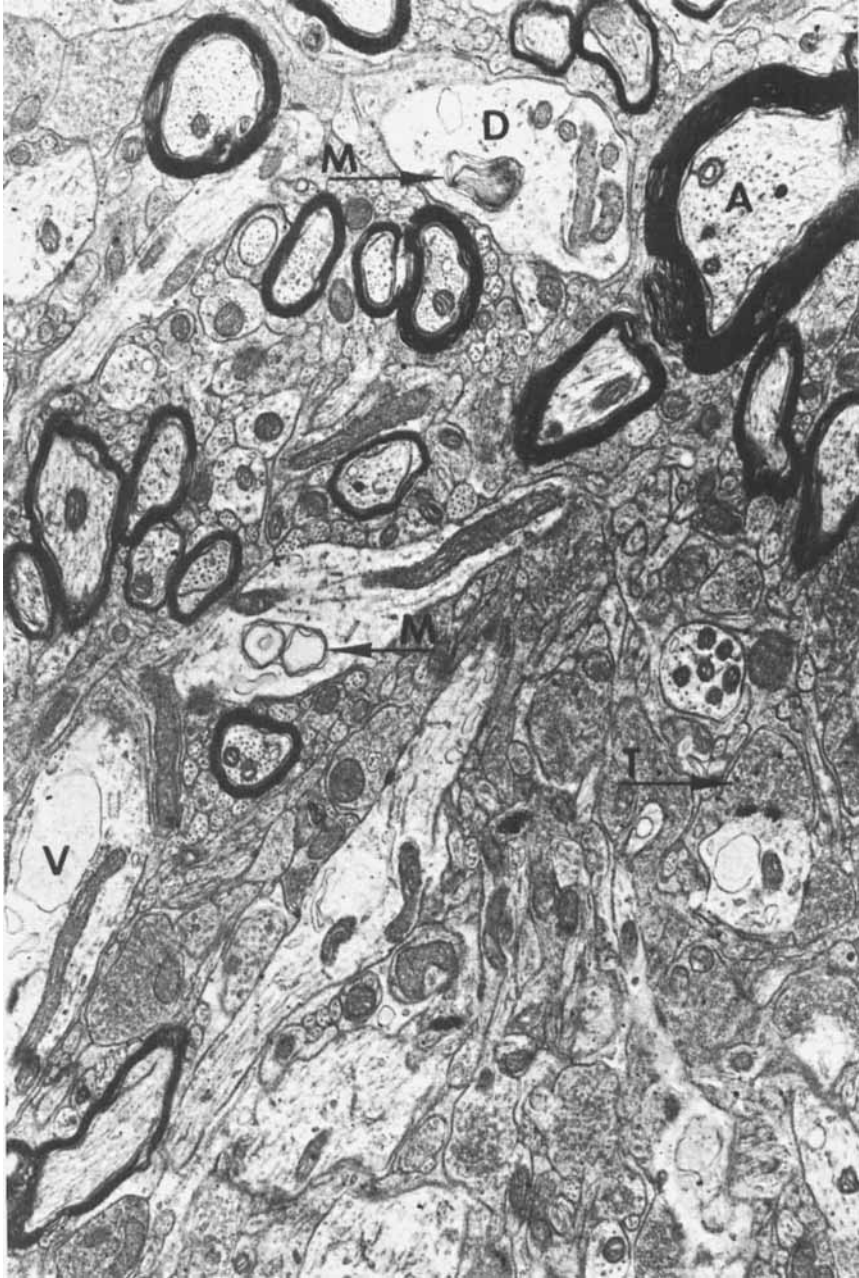


FIGURE 12. Electron micrographs from hypothalamus of experimental animals exposed to a 50 mW/cm² power density for 24 hr. Note the presence of myelin figures (M) in dendrites (D). There is an increase in number and size of smooth membranous structures within dendrites (V). Axons appear to be unaltered. Stained with uranyl acetate and lead citrate.

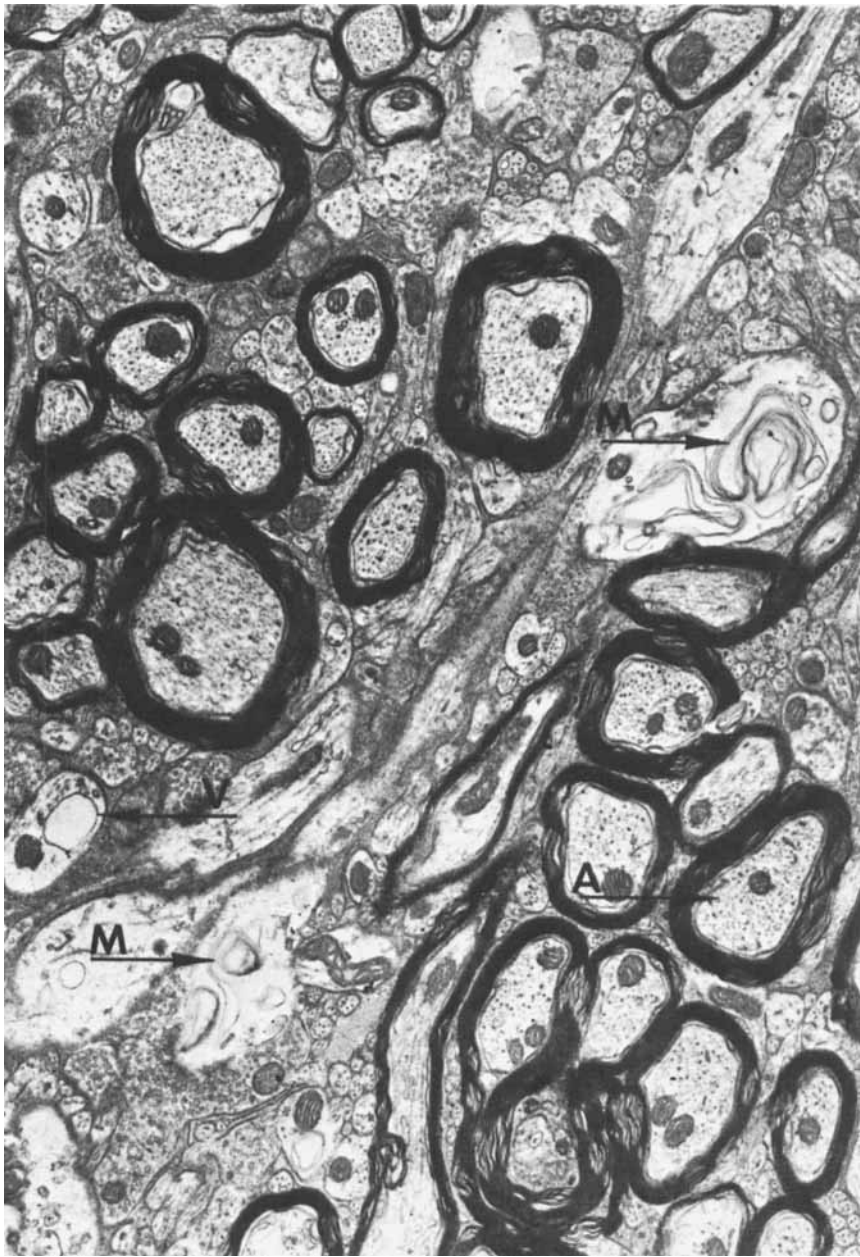


FIGURE 13. See FIGURE 12 legend for explanation.

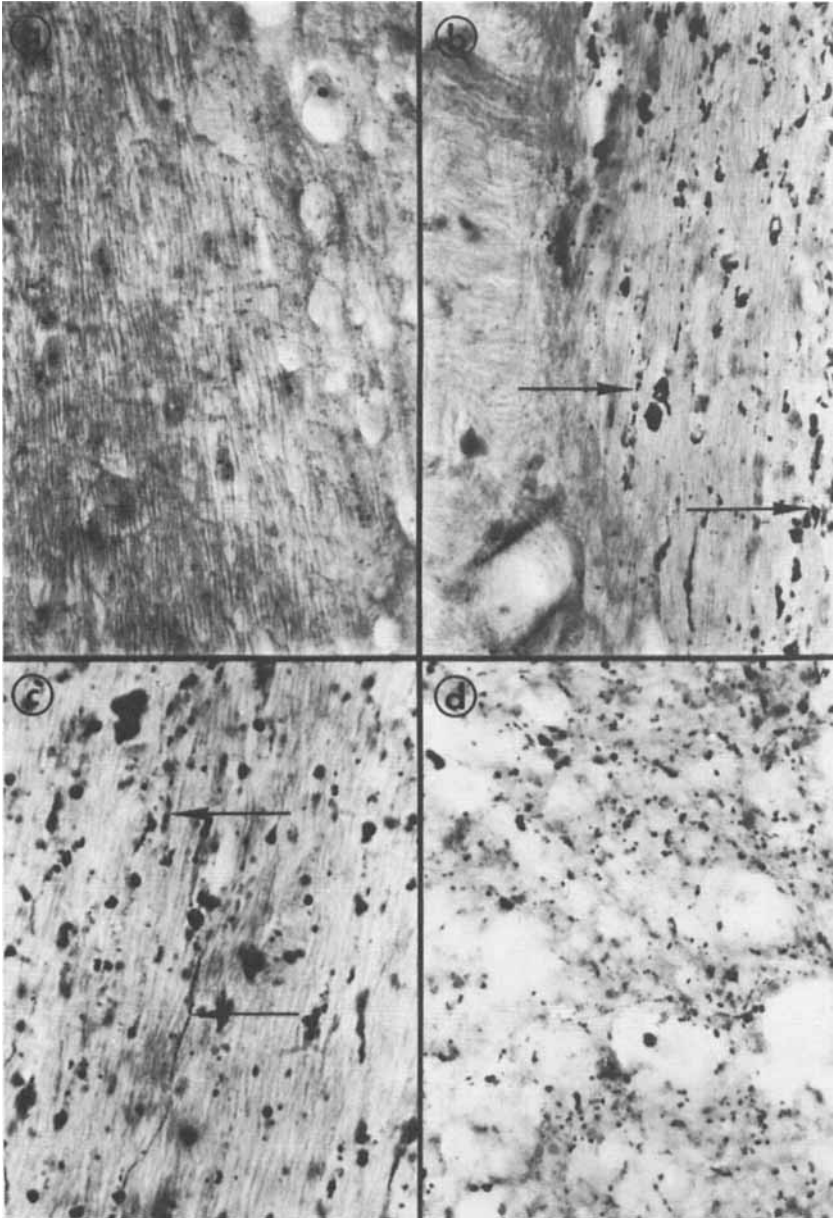


FIGURE 14. Optic tracts from a control animal stained by the Fink-Heimer technique (a) and from a positive control (one eye was enucleated) (b,c). Note the appearance of degenerated fibers after Fink-Heimer staining. Lateral geniculate nucleus of same animal as in b and c (d). Note the degenerating axons and their terminals.

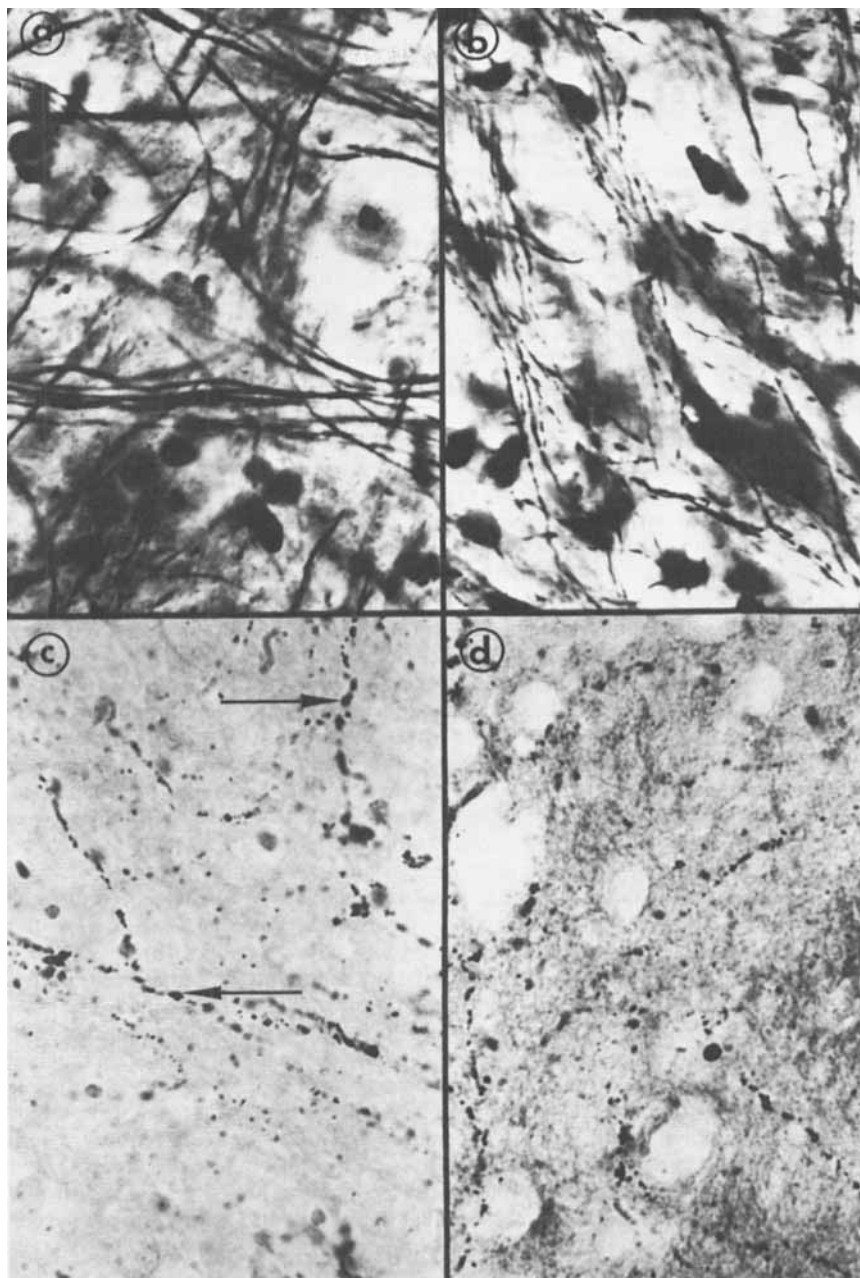


FIGURE 15. Pontine tegmentum of control (a) and microwave exposed (b) animals. Note the beaded appearance of fibers in the experimental (b) tissue after Fink-Heimer staining. Dorso-medial area of hypothalamus from enucleated (c) and microwave-exposed (d) animals. Both show some degenerated fibers with Fink-Heimer staining.

the irradiated animals. However, vascular stasis, or congestion, was observed in some animals.

DISCUSSION

Microwave exposure can cause severe structural alterations in neuronal cytoplasm. At the light microscopic level, the result is a decreased, patchy basophilic staining and vacuolization of the cytoplasm.

At the electron microscopic level, neurons of experimental animals are devoid of polyribosomes and rough endoplasmic reticulum. Other cytoplasmic abnormalities are noticeable, including cytoplasmic vacuoles and milder dilation of smooth membrane cytoplasmic organelles. The dendrites of neurons in exposed animals display vacuoles, myelin figures, and a paucity of neurotubules. In contrast, little structural perturbation occurs in nuclei of affected neurons. It is not clear why structural disturbances appear to be chiefly cytoplasmic. Other investigators have reported alterations in the cytoplasmic processes of neurons, including loss of dendritic spines on cortical neurons and disruption of sensory peripheral nerve fibers.² We have cautiously begun to explore the possibility of axonal degenerative phenomena in the central nervous system with the Fink-Heimer technique.

Some discrepancies exist between the present report and others as to which neurons are affected. In our experimental animals, only hypothalamic and subthalamic neurons appear to be structurally altered. As best as we could determine, neurons of the thalamic relay nuclei, cerebellum, pons, and spinal cord were unaffected. Though we have not explored the cerebral cortex in detail, hippocampal neurons appeared unaltered. Austin and Horvath,⁵ who reported only slight microscopic changes in dogs after brief single exposures to microwaves at 2450 MHz, described pyknosis in certain cerebral cortical neurons, including the pyramidal layer of the hippocampus and deep cerebellar nuclei and unnamed parts of the brainstem. Tolgskaya and Gordon¹ have demonstrated alterations in hypothalamic, cerebral cortical, and spinal ganglion neurons in rats subjected to microwave treatment under conditions comparable to those used for this report. In their study, long-duration low-intensity microwave irradiations also affected thalamic neurons. In our experimental animals, thalamic relay neurons showed no changes from those of control animals. The discrepancy may be explained on the basis of exposure parameter differences (duration of exposure and power densities) or it may be a problem of semantics in that the area we call the subthalamus is sometimes referred to as the ventral thalamus.¹¹ It is less easy to discount the difference between first-order sensory neurons. Though we did not look at spinal ganglion cells, we did systematically examine cells of the mesencephalic nucleus of the trigeminal nerve. These first-order sensory neurons in carnivores and primates innervate muscle receptors and certain receptors associated with teeth.^{12,13} In no case did we see signs of retrograde pathologic changes in the neuronal somas. The radiation wavelength was long enough to penetrate the skin and presumably affect these deep receptors.^{2,3} Similarly, if microwave exposure disrupts the peripheral fibers of both motor and sensory neurons in peripheral tissues, we might have expected to observe retrograde alterations of motor neurons. In fact, none could be found. This question deserves more experimental scrutiny.

It is not yet clear how structural alterations of neurons are incurred after microwave exposure. One question yet to be resolved is the energy form responsible for the observed changes. It is not clear whether the electromagnetic radiation itself or tissue heating, due to radiation energy absorption, is responsible for the effects ob-

served. The power density of 50 mW/cm² used in this study is capable of producing heating effects.² In fact, both the small animal and the frequency value employed here would lead one to expect that a maximal heating effect would occur in the diencephalic region.⁴ However, our animals were freely able to change their positions, receiving total body exposure, rather than one directed to a certain area of the head. Nor is it clear that the heating effect would be so confined to an area less than 3 mm³, which would seem necessary to account for the lack of involvement of thalamic relay nuclei. And, at a power density of 25 mW/cm² and a frequency of 2450 MHz, similar pathologic alterations were noted; under such treatment conditions, the heating effect begins to approach a 2.5 times threshold value.³ Even lower power densities need to be explored to determine whether they elicit similar selective changes. Animals should also be heated until near death to see if there are structural alterations similar to those reported to occur with microwave exposure.

Another major problem with respect to structural changes in neurons effected by microwave exposure involves the biologic mechanism rather than the responsible energy form. The energy form may affect the neurons themselves; the other broad possibility is that the effect is indirect. The latter possibility gains some support from reports that the cerebral vasculature exhibits signs of hemorrhage, congestion, or perivascular edema.^{1,5} We have not found definitive evidence of hemorrhage or perivascular edema either by routine histologic examination or by injection of trypan blue to determine if the dye, normally excluded from the brain parenchyma by the so-called blood brain barrier, leaked into the brain tissue in animals irradiated with microwaves.¹⁴ Austin and Horvath⁵ also could not find gross or microscopic evidence of leakage into brain tissue in dogs injected with trypan blue and exposed to microwaves. We have noted hyperemia in animals treated with microwaves. It is possible that at least some structural modification is produced by the fixation technique. To account for the differences detected between control and experimental animals, however, it is necessary to postulate differential fixation qualities. Such phenomena could be due to vascular congestion and, in turn, poor perfusion or to changes in the local microenvironments, which might yield differential effects of the same fixative.

CONCLUSIONS

Thus, in answer to the question posed by the title of this paper, we can say that microwave irradiation at a frequency of 2450 MHz and at power densities of 25 or 50 mW/cm² for 30 min to 22 days consistently alters some areas of the central nervous system, whereas other regions appear unaltered. Our major observations are enumerated below:

The hypothalamus and subthalamus are consistently affected. At the light microscopic level, neuronal somas in the hypothalamus and subthalamus display vacuolization and chromatolysis. Observations of the central nervous system with the electron microscope revealed a decrease in the protein synthesis apparatus. There was a distinct scarcity of rough endoplasmic reticulum and polyribosomes. In addition, myelin figures and swollen membranous structures were frequently detected in hypothalamic neuropil dendrites.

No changes were evident in cell bodies of hippocampal pyramidal neurons, Purkinje cells of the cerebellum, mesencephalic nucleus of the trigeminal nerve, motor neurons of the spinal cord ventral horn, or the cell bodies of thalamic relay nuclei.

The Fink-Heimer technique has provided some tentative evidence that microwave irradiation may produce axonal degeneration.

ACKNOWLEDGMENTS

The authors thank Ms. Grace Lau and Mr. Ralph Hill for their technical assistance. We are indebted to the Department of Microwave Research, Walter Reed Army Institute of Research for making available their facilities for exposure of animals and their engineering assistance for accurate dosimetry.

REFERENCES

1. TOLGSKAYA, M. S. & Z. V. GORDON. 1973. *Pathological Effects of Radio Waves*. B. Haigh, Transl. Consultants Bureau. New York, N.Y.
2. PRESMAN, A. S. 1970. *Electromagnetic Fields and Life*. F. R. Sinclair, Transl. Plenum Publishing Corporation. New York, N.Y.
3. GORDON, Z. V. 1970. *Biological Effects of Microwaves in Occupational Hygiene*. N. Kaner, Transl. NASA TTF-633. The Israel Program for Scientific Translations. Jerusalem, Israel.
4. LIN, J. C., A. W. GUY & G. H. KRAFT. 1973. Microwave selective brain heating. *J. Microwave Power* 8: 275-286.
5. AUSTIN, G. N. & S. M. HORVATH. 1954. Production of convulsions in rats by high frequency electrical currents. *Amer. J. Phys. Med.* 33: 141-149.
6. KARNOVSKY, M. J. 1965. A formaldehyde-glutaraldehyde fixative of high osmolarity for use in electron microscopy. *J. Cell Biol.* 27: 137A, 138A.
7. LUFT, J. H. 1961. Improvements in epoxy resin embedding methods. *J. Biophys. Biochem. Cytol.* 9: 409-414.
8. FINK, R. P. & L. HEIMER. 1967. Two methods for selective silver impregnation of degenerating axons and their synaptic endings in the central nervous system. *Brain Res.* 4: 369-374.
9. DAVENPORT, H. A. 1960. *Histological and Histochemical Technics*. 196-198. W. B. Saunders Company. Philadelphia, Pa.
10. LAMBERT, P. D., R. C. NEALEIGH & M. WILSON. 1972. Effects of microwave exposure on the central nervous system of beagles. *J. Microwave Power* 7: 367-386.
11. CROSBY, E. C., T. HUMPHREY & E. W. LAUER. 1962. *Correlative Anatomy of the Nervous System*: 307-309. The Macmillan Company. New York, N.Y.
12. JERGE, C. R. 1962. Organization and function of the trigeminal mesencephalic nucleus. *J. Neurophysiol.* 26: 379-392.
13. CORBIN, K. B. 1940. Observations on the peripheral distribution of fibers arising in the mesencephalic nucleus of the fifth cranial nerve. *J. Comp. Neurol.* 73: 153-177.
14. ALBERT, E. N. & M. DESANTIS. 1973. Unpublished observations.

DISCUSSION

DR. S. M. MICHAELSON: You find that selective damage occurs to the hypothalamic and subthalamic areas and that the rest of the brain exhibits only minor injury. Also, you indicate that tremendous activity exists in the hypothalamus. You may be observing an exhaustion phenomenon in the hypothalamus. Without critical temperature measurements in that brain region, it's impossible to distinguish specific microwave effects on the central nervous system or hypothalamus from effects of a high fever or those caused by any stimulus to the hypothalamus.

DR. A. W. GUY: For the size of animals that you're using, we've measured

absorbed energy as high as 4 W/kg per incident milliwatt. So, considering your range of incident powers, from 10 to 50 mW/cm², you could be inducing peak absorbed power densities in the animal of 40 to 200 W/kg. This range far exceeds what is normally used for diathermy treatments in 20-min exposures of patients. This could appear in localized areas of the body, such as the brain, especially where you have small cross sections. And, a rectal temperature measurement won't necessarily reflect this high power absorption in localized areas.

DR. ALBERT: We were not able to measure hypothalamic temperature.

DR. H. WACHTEL: Did you examine synaptic membranes? If so, were any changes evident that you think might lead to an altered function at synapses?

DR. ALBERT: We took a cursory view of the synaptic junction, in both pre- and postsynaptic areas. We've stained with PTA to delineate these zones, which become very intensely colored. Cursory observations detected no changes, but we are continuing to investigate at higher magnifications and with electron histochemical stains.

DR. WACHTEL: Were any of the dendritic alterations possibly functionally significant in terms of synaptic transmission?

DR. ALBERT: I think that the changes were early indications of degeneration. The presynaptic terminals in all cases appeared quite normal. But, until the specialized zones of the post synaptic membrane are totally degenerated, transmission may be possible if the presynaptic membranes are still intact.

DR. J. WEST (*Armed Forces Radiobiology Research Institute, Bethesda, Md.*): Have any behavioral or performance decrement studies been conducted to relate altered behavior to the types of morphologic changes that you have described?

DR. ALBERT: We have not done any functional correlates.

DR. WEST: In view of the possible vascular mediation of the damage you have described, if you kept the animals and serially sacrificed them over a more lengthy time period, might other changes be manifested later?

DR. ALBERT: That was our initial thought. Two observations might reflect on your question. First, the animals sacrificed after a recovery period actually showed fewer cytoplasmic changes. I don't know whether this was a recovery phenomenon or whether it was delayed neuronal reaction. We haven't gone far enough in our studies to solve this problem. Second, the optimum time for studying retrograde and anterograde degeneration is in the vicinity of 7–10 days. We wanted to utilize this information for our experiments.

DR. J. W. FRAZER: With 15 mW/g absorbed power, there's quite a loss of divalent metals in the ribosomal fraction of liver extracts. Observations of RNA changes are by no means limited to the brain. This loss may be a fairly general thermal tissue phenomenon. We've irradiated rat brains, with a microwave source, and thermal scans have shown that along the basicranium, in the vicinity of the circle of Willis, a hot spot is present.

DR. ALBERT: We have looked at other tissues and have found generalized changes, particularly in certain liver and heart mitochondria. Even though the ability of these organelles to carry on oxidative phosphorylation is not affected, we can see morphologic changes.

DR. FRAZER: How do your histologic findings compare to those seen in animals that have experienced fevers of 4–5°C?

DR. ALBERT: Vacuolation and chromatolysis in the hypothalamus are typical responses of heated neurons.

DR. S. SZMIGIELSKI: Were your power density measurements made inside the exposure cages or outside of them in air?

DR. ALBERT: All measurements were made with the exposure chamber removed.

DR. SZMIGIELSKI: The walls of the Plexiglas® cages could slightly influence the exposure through reflection, attenuation, and other factors, so your actual incident energy of exposure was, I think, much lower than that which you have indicated. I feel that there are at least three critical points about such a study. One is dosimetry; the second is the species of animal. You could obtain quite different changes in rabbits, rats, or hamsters. My experience also suggests the influence of a third factor, hypoxic hypoxia. The histologic results additionally depend on how long after exposure the animals are sacrificed. Findings will be quite different when the subjects are sacrificed immediately, because the most significant changes appear after 24–48 hr. Thereafter, there may be a period of recovery for a few days. I think that it is very important to compare changes at different periods after treatment has been discontinued.