Ohio State Highway Patrol
Planning and Analysis
March 1991

Staff Study

Nonionizing Radiation And Speed Monitoring Devices

Research and Development

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Patrol

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Department of Highway Safety

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Problem

Are the Officers of the Ohio State Highway Patrol
Exposed to any Known Health Hazard
By Using Traffic Speed Monitoring Radar Devices?

Executive Summary

Thousands of articles have been written addressing the question of nonionizing radiation safety. Scholars, scientists and experts are decisively torn over the impact this radiation is having on humans. Is it a mentor or murderer? Both extreme views have impressive advocates.

The radar units currently used by the Patrol conform to the stringent standards of the American National Standards Institute (ANSI). Proof that Patrol radar meets ANSI standards was produced when units were tested by Battelle Memorial Institute and reviewed by the Bureau of Workers Compensation, Department of Safety and Hygiene. Both Battelle and Safety and Hygiene reported that the radar units used by the officers of the Patrol operate within all applicable standards.

Currently, there is no reason for the Patrol to stop using police radar speed monitoring devices. It is imperative, given todays growing knowledge bank, that the Patrol keep abreast with any developments in the field of nonionizing radiation.

Relevant Facts

1. The safety and well-being of officers of the Patrol using radar devices is of primary concern.

2. Current U.S. standard for exposure to radio frequency electromagnetic field 10.525GHz (X-band radar) is $5mw/cm^2$ (Appendix A).

3. Nonionizing radiation is absorbed at different levels based on the frequency used.

4. Considerable research has been performed on the potential health hazards of low level nonionizing radiation with dichotomous results.

5. Nonionizing radiation at intensity levels high enough to cause thermal increase in human tissue is not safe.

6. Radar equipment is in use most of the time an officer is on patrol.

7. An officer will be on patrol approximately 57% of their usual and normal annual work cycle. (1,071,858 patrol hrs. by 1,872,926 work hrs.)

8. No federal, state or local agency is willing to establish a universal maximum safe exposure limit at this time.

Assumptions

1. Continued research and ensuing federal regulation will resolve questions concerning any perceived safety hazard.

2. Patrol equipment will continue to be tested on a semiannual basis to ensure its operation remains within manufacturers specifications and acceptable safety standards.

3. Literature reviewed was in regard to police traffic radar irradiation alone. The combination of radar equipment and communications equipment may have differing results.

4. Police traffic radar could be replaced by another enforcement system should probable cause show the need to do so.

Discussion

Survey of Literature

There is much concern regarding the effects of devices emitting microwave radiation and their potential for harm to individuals using them. First, it should be understood that the radiation emitted from radar, television and radio transmitters, as well as microwave ovens, is nonionizing. Nonionizing radiation is at the low end of the total radiation spectrum. Examples of nonionizing radiation include: radio waves, microwaves and visible light (radiation that we see: Appendix B). On the opposite end of the spectrum is ionizing radiation. This type of radiation is emitted by some irradiating medical equipment and atomic reactions. X-rays, cosmic rays and gamma rays are examples of ionizing radiation. The potential for harm by these rays is well known. The key that divides ionizing and nonionizing radiation is the ability of the ray (or wave) to permanently change the physical makeup of an atom. Nonionizing radiation does not have the ability to alter atomic makeup as witnessed in nuclear radiation effects.

Though nonionizing radiation cannot cause permanent changes in atomic makeup, it can still be dangerous. In high powered forms it can burn, as can be observed by cooking in microwave

ovens. There is no question in anyone's mind that microwave radiation at levels that can raise the temperature of it's target is harmful. The controversy begins when radiation intensities drop below the levels that cause thermal changes in it's target. Experts are divided on what level of radiation constitutes a threat to human life once below the thermal break off.

Microwave technology was developed during World War II in the form of radar. Accounts of men warming themselves in front of the radar antenna during this development have been recorded. In the 1940's and 50's initial studies were performed to determine if radar exposure could have any harmful side effects. These, and many other studies, have over time culminated in the American National Standards Institute standard C95 (ANSI C95) initially published in 1966. It is important to note that ANSI is not a governmental agency or in any way responsible to any level of government.

Since the development of ANSI C95 to today, there has been considerable research concerning the relationship wavelength, or frequency, may have on absorption. However, the only real change in the standard was an adjustment reducing exposure to frequencies lower than police radar. It is imperative that any concept of an exact safety limit be purged. Currently, no consensus of what exposure is safe has been reached. Considering radar, ANSI has somewhat arbitrarily decided that an exposure of one tenth of that required to cause a thermal increase in it's target would be

considered safe. It would take a beam ten times above the maximum standard allowed to cause a temperature change. The current standard, ANSI 95.1-1982 indicated that maximum safe exposure for X band traffic radar, 10.525GHz, is 5mW/cm^2 or 5 milliwatts of energy per one square centimeter of tissue (skin) over a six minute time duration[I]. The maximum total body exposure is 0.4 W/Kg or .4 watts of absorption for each kilogram of tissue[II].

Twenty traffic radar units tested by Law Enforcement Standards Laboratory, Center for Consumer Product Technology, U.S. Department of Commerce, National Bureau of Standards, produced aperture power densities or beam intensities from .25 to 2.82 mW/cm^2. This range represents the intensity of the beam produced by the antenna without any shielding. Because of the shielding that is used in radar units, the power emitted from the rear and sides of the units range from 0.02 to <0.001 mW/cm^2 in most situations tested. There was one specific exception when the radar unit was positioned on the passenger side rear seat facing forward. In this situation direct exposure for the passenger side front seat was increased. Yet only two units showed a significant increase to 0.36 and 0.137 mW/cm^2 which is still far below standard acceptable exposure (Appendix C).

I Appendix A, ANSI Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 300kHz to 100GHz: Table 1; column 4; 1500-100,000 MHz, pg 10.

II I.B.I.D., Text under Explanation

Opponents to the current standards have conducted considerable research attempting to prove that levels much lower than those currently acceptable are actually dangerous. A large portion of the research has been in the area of nonionizing radiation produced by electromagnetic fields.

A study of interest, "Brain Tumor Mortality Risk Among Men With Electrical and Electronics Jobs: A Case-Control Study" by Terry L. Thomas PH.D and associates, developed interesting conclusions based on occupational exposure to both microwave and electromagnetic radiation. In this study subjects were separated based on two criteria: exposure to microwave/radio frequency (MW/RF) radiation and exposure to electromagnetic fields. The results indicated that exposure to electromagnetic fields alone or in concert with MW/RF radiation resulted in a statistically significant increase in brain cancer formation. However, exposure to MW/RF radiation alone did not show a statistically significant increase in cancer rates. The subjects falling into the last category included welders and radio broadcasters. These occupations are classified as method 1, category 2 in Appendix D. Police radar equipment would be classified as MW/RF radiation alone by this study. Also, the radiation frequencies used in this study were much lower than those used by police radar.

Several studies dealing strictly with exposure to microwave energy have had varied results. The first study to examine is, "Suppression of T-Lymphocyte Cytotoxicity Following Exposure to

Sinusoidally Amplitude-Modulated Fields", by Lyle, Schenchter, Adey and Lundak. Cells used for this experiment were from Sprague-Dowley rat spleens. This study suggests that exposure to varying (modulating) microwave energy reduces white blood cells (lymphocyte) ability to attack foreign entities in the body (cytotoxicity). Their results showed that the body's ability to attack disease is decreased by exposure to low level energy over a fairly short period of time (2 to 4 hours). However, the base wave frequency used in this experiment was 450 MHz, which is below the frequency used by police radar (Appendix E).

Another author, Milton M. Zaret, expounded on his observations with radio frequency irradiation. In his study, "Radio Frequency Irradiation as a Factor in Human Tumors", Zaret presents a case report of a patient that contracted cancer of the pancreas and a cataract in his left eye. These occurrences are a supposed result of long term repeat exposure to irradiation at 115 MHz and 470 MHz. The patient's occupation was in radio frequency equipment repair. In addition, he used a portable radio which he held in his left hand or attached to his belt. A correlation is implied between left hand radio use and his left eye cataract. Also, abdominal radio exposure and pancreatic cancer is implied (Appendix F). As discussed earlier by Dr. Thomas in his research, the patient's professional exposure to electromagnetic radiation as a radio equipment repairman could have influenced Dr. Zaret's observations. In addition, police radar equipment operates on frequencies much higher than those used in this experiment.

To further confound the issue, "Alterations in Protein Kinase Activity Following Exposure of Cultured Human Lymphocytes to Modulated Microwave Fields", by Byus, Lundak, Fletcher and Adey takes this murky issue and stirs it up. In this study human white blood cells were cultured and then exposed to low level 450MHz modulated radiation. The objective was to show the effects of radiation on the process that uses proteins to create enzymes (kinase). All human cells are completely dependant on enzyme activity to function. Their findings showed that in the short run, between 15 and 30 minutes of exposure, there was a marked reduction in protein kinase. But, by 45 minutes of exposure, kinase was back to pre-exposure levels and remained there for the duration of the experiment. There is no doubt that exposure has an effect. However, there is no proof that in this case it has any long-term harmful effects (Appendix G).

Reversing direction, advocates of the safe use of microwave energy are just as confident in their position. In "The Microwave Problem", authors Arthur Guy and Kenneth Foster present their views. In this article the authors suggest that many of the experiments that found microwave energy harmful either could not be reproduced or simply by continued experimentation were found in error. Two issues in this article are of significance to radar emissions: the relaxation of Russian standards from microwave energy exposure and an experiment that comes very close to police radar exposure. First, it has been a long time thorn in the side

of ANSI that Europe, the Soviet Union and Canada have standard exposures to microwave energy much lower than that of the U.S.. Since 1986 the Soviet Union and the Warsaw Pact countries have changed their limits to .4 watts per kilogram for one hour, which is comparable to the ANSI standard of .4 watt per kilogram averaged over six minutes.

The second issue of interest in Foster and Guy's article is an experiment in long term low dose irradiation effects very similar to radar exposure. The experiment consisted of rats exposed to low dose energy of .2 to .4 W/Kg. The study was conducted for fifty-nine weeks with the rats irradiated twenty-one hours a day. The only significant difference was that eighteen exposed vs. five control rats developed primary malignant tumors. This would be alarming if it wasn't known that for this particular strain of animal the expected rate of cancer was about that of the exposed population. Simply, the control group did not conform to the cancer rate expected for its specie. When the results are illuminated with this information they are far less, if at all, conclusive.

Perhaps of more direct concern are radar units that do not conform to their own specifications. Testing of twenty-four radar units on the market in 1984 by the International Association of Chiefs of Police (IACP) to determine if these radar units operated exactly as the manufacturers claimed. The results were interesting. The most common criteria that radar units failed to meet

during initial tests was processing channel sensitivity requirements. Each manufacturer was then given an opportunity to make any adjustments needed to conform to their specifications before testing resumed. Once adjustments were made there were no units that failed outright. Various units failed on one or two minor points. However, none varied so far as to cause any known health hazard. As a result of this testing the IACP, with the National Highway Transportation Safety Administration (NHTSA), has created a Consumer Products List of units that meet or exceed model specifications. Agencies should ensure units under consideration have NHTSA/IACP certification. Manufacturers may produce, under one model number, units that conform and do not conform to NHTSA/IACP model specifications. The publication will indicate which units have their approval and how to identify them.

Similar to radar exposure in negative publicity is amateur radio. Studies have produced concern for the same basic hazards and negative conclusions. In "Is Amateur Radio Hazardous to our Health?", Ivan Shulman M.D. covers much of the ground we have already. The major difference between the two is that amateur radio utilizes a much greater power supply running close to the user along with powerful transmitting equipment. At the end of the article there are measures suggested to reduce exposure. Several would be well heeded by radar operators such as: do not operate the equipment when cover panels and protective shields are

out of place, and use only as much power as necessary to do the job (Appendix G). It is interesting to note that Arthur Guy, co-author of "The Microwave Problem", is also an amateur radio operator.

Taking a step back from clinical analysis, the question of risk assessment arises. What risks are we willing and conversely not willing to accept. In "Ranking Possible Carcinogenic Hazards" by Ames, Magaw and Gold, numerous carcinogens and their dose per exposure are presented. As an example, one very potent carcinogen is aflatoxin. Not only is aflatoxin a carcinogen it is also a mutagen, which means part of how it causes disease is by subverting the genetic material (DNA) in living cells. By being able to change DNA it is playing with the very material that decides everything from eye color to whether a person will develop Alzheimer's disease. Yet every time people consume nuts, wheat, corn and to a lesser extent drink milk we are exposing ourselves to low levels of aflatoxin. One peanut butter sandwich, thirty-two grams or just over one ounce, contains one tenth of the FDA limit of aflatoxin.

In the same article, the authors present data showing that what is a carcinogen to one test subject may be benign to another. To quote statistics, out of three hundred ninety two (392) different chemicals in the database used for the study, ninety six (96) were positive in mice but not in rats. Conversely, fifty six (56) chemicals were positive in rats that were not positive in mice.

If two mammals very closely related are not consistent in there reactions to chemicals, can we be certain any relationship exists linking these animals to man? These are all good questions with very few answers.

Battelle Study - Nonionizing Energy

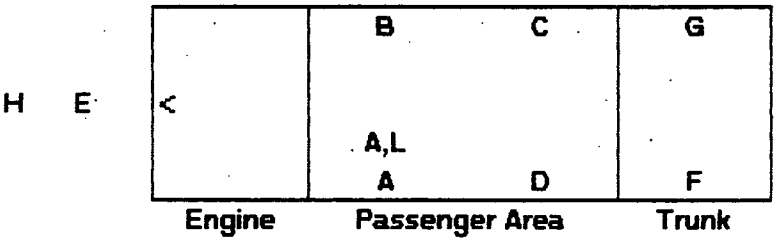
The state of Ohio contracted the internationally recognized scientific research organization Battelle Memorial Institute, Columbus operation, to measure the power densities that officers are exposed to operating radar, and communication devices. The tests were conducted December, 13 1990. Engineers used the Narda Model 8611 Radiation Probe measuring device.

In every case the MPH K-55 radar unit, currently used by the Patrol, was well within ANSI standards. Looking at the diagram on the following page, the radar unit was centered on the front dash board of the patrol car. Readings were taken with the unit facing directly forward, rearward, and at a forty five degree angle. These tests accurately reproduce actual field operations. During the MPH K-55 testing, readings were taken for each of four passenger positions in the car. In every case, the signals were too weak for the probe to register. The probe was moved about the general area of each passenger position until the highest reading could be determined. Additional samples taken were: within one foot or less of the radar unit itself; ahead of the car at the front bumper and fifteen feet forward of the bumper with the unit aimed forward. The highest reading for the K-55 was 1.0 mW/cm^2. This reading was obtained with the unit facing rearward and the probe twelve inches from the face of the unit.

Battelle Test Results

Probe Testing Sights:

10 A.M. - 3 P.M.
December 13, 1990



1987
CHEVROLET

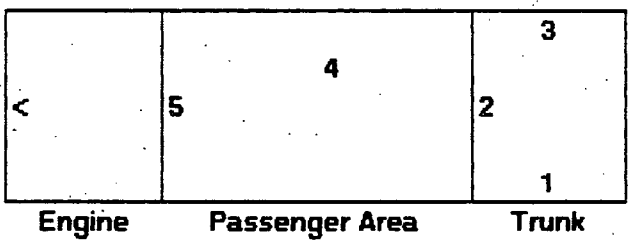
1989,1990
FORD

A - Drivers Position
A,L - Drivers Right Leg
B - Front Seat Passenger
C - Passenger Side Rear
D - Driver Side Rear Passenger
E - Front Bumper
F - Low Band Antenna
G - High Band Antenna
H - 15' Forward of Bumper

Antenna Sights:

Test Device:

Narda
Radiation
Probe:
#1 .3 - 100 MHz
#2 300 Mhz to
40 GHz



1 - Low Band, 45 MHz, 100W
Patrol Radio
2 - CB, 27.065 MGz, 3.5W
3 - High Band, 155 MHz, 100W
LEERN Radio
4 - VHF Repeater, 465 MHz, 500 mw,
on Lightbar
5 - Radar, 10.525 GHz, 25 mw

The MR-7 radar, manufactured by Kustom Electronics Inc., proved to be well in excess of the ANSI standards when used directly behind the officer. For testing, the unit was placed on both the inside and outside window positions. The power densities produced inside were several times those acceptable. It is important to note that the MR-7 was intended for exclusive outside mounting. By failing to follow this requirement of operation officers could expose themselves to excess irradiation.

Strictly from a perspective of the radar units currently used by the Patrol, there is practically no measurable exposure involved in their use.

The same is not true of the communications equipment. First, radio equipment is only producing radiation when transmission is taking place. During the vast majority of a shift an officer will be listening to other transmissions. On several occasions when the 100 watt transmitters were used the rear seat passengers were exposed to short term power densities well beyond the ANSI standards during transmission. Additionally, excessive readings were registered when the probe was placed within inches of the antenna broadcasting. However, recall that the ANSI standard requires exposure to be measured over six continuous minutes. When measurements are averaged over the required six minutes, the overall levels fall within, or close, to the ANSI standard. Considering that the average transmission lasts 5 to 15 seconds with a break

of 3 to 5 times that, the average exposure for six minutes drops significantly.

Until now only the ANSI standards have been discussed, however, there are other standards that can come into play. The Occupational Safety and Health Administration (OSHA) has it's own standards for acceptable industrial exposure. Basically, the measurements and time duration are the same, but the maximum exposure for all communications equipment is 10 mW/cm^2 rather than the 1 to 5 limits used by ANSI. Under these conditions, the equipment used by the Patrol is well within OSHA standards. Detailed speculation of the combined effects of both radar and communications devices is beyond the scope of the Battelle findings.

Bureau of Workers Compensation

Division of Safety & Hygiene

The Division of Safety & Hygiene (S&H), at the request of the Patrol, reviewed the Battelle study discussed in the previous chapter. The purpose was for S&H to assess the health effects of using the communications and radar equipment.

S&H came to five conclusions concerning the Battelle study. First, the vehicular repeater, citizens band radio, and cellular telephone, "...were of sufficiently low power to be of little concern."[III] They also confirmed that the K-55 radar units never exceeded the ANSI standards.

Second, S&H concluded that the hand-held radios were not a concern, even though one measurement was above ANSI standard. The reason being, the ANSI standard has exclusions for hand-held, mobile, and marine radios. The standard is designed to measure absorption over the whole body and when the readings are interpreted in that light, the standard is not broken.

Third, both the high and low band radios were determined to be safe. The Patrol advised S&H that the maximum time duration of

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III Bureau of Workers' Compensation, Division of Safety & Hygiene, 2-21-1991, pg 1.

communication is ten minutes. Also, out of every minute a maximum of thirty seconds is spent transmitting. Remember that significant irradiation occurs during transmission. Taking into consideration that the ANSI standards are based on an averaged continuous exposure over six minutes, the radios remain within all applicable limits.

Fourth, the MR-7 radar was designed for external mounting only. The readings that exceeded ANSI standards were based on having the unit installed inside the rear window. The readings were taken in the front seat position directly forward of the unit. Logically when the unit is operated according to manufacturers external specifications, the excessive irradiation effects are corrected.

Finally, S&H stated that there is a potential for localized effect or burning from holding onto an antenna. This statement refers to the communications antennas. Officers would not handle damaged appliance electric cords while connected to current. Antennas are similar to bare wires when transmitting. It is important that officers respect the energy sources utilized.

All of the testing to date has been with only one unit operating at a time. Under normal conditions more than one device will be used at a time. In these cases the power exposures would be additive. However, consideration must be given to each frequency and how the human organism absorbs it. In the strongest

case with a MR-7 operating outside the rear window and normal radio use, "...no cumulative overexposures would be expected."[IV]

Questions were also raised concerning Patrol radio technicians who are exposed to radar units at close range. In reality, these is insufficient evidence to prescribe any specific safety requirements. S&H, however, suggests that there may be some potential hazard. One crucial factor to be considered is that technicians will be working with units that are malfunctioning. Therefor, technicians potentially are at higher risk to excessive exposure than any field officer.

The remainder of the response to S&H reiterates topics and issues already discussed. Simply, there is no known reason to discontinue the use of radar. It is important that these units are used according to manufacturer's specifications. As with any device, improper use can result in health hazards.

IV Bureau of Workers' Compensation, Division of Safety and Hygiene, pg 3.

Conclusions

To draw any concrete conclusions from the some ten thousand publications concerning nonionizing radiation would be a Herculean feat. The greatest scientists, researchers, professors and physicians of our time have not been able to agree on hardly anything concerning this issue. Reflecting on the extremely small number of articles mentioned in this document, either mankind is plunging headlong into it's own demise or we are harnessing a media that provides the answer to many of our aspirations for future exploration and development.

Continuous exposure to anything has an effect. Increased running, jogging or walking will cause calluses on the foot to thicken and joints to wear. Yet, medical experts recommend people increase these activities to prevent heart disease and hopefully extend life. Any activity will have a different effect on each person engaging in it. Every person will absorb nonionizing energy differently depending on many factors known and unknown.

With few exceptions, every officer using radar speed monitoring devices also owns and operates a microwave oven. These ovens operate at 2.45 GHz and according to ANSI standards must comply with the same maximum power density exposures as traffic radar devices. However, microwave ovens produce electromagnetic

fields at five hundred (500) to seven hundred (700) watts which is hundreds of times more powerful than traffic radar. Durations of operation widely vary, but the potential for excess irradiation is far more likely. For those highly sensitized, these risks would be unacceptable.

Realistically speaking, uncontrolled fear of what is currently unknown is less than prudent. It is wise to challenge accepted standards to ensure they remain abreast of current situations. However, without a consistent body of evidence for or against the current ANSI standard, knee-jerk reactions should be avoided.

From the information suggesting the safe use of nonionizing energy in police applications, or the lack of consistent proof of it's danger, there is no reason to substantially alter the way police radar is used <u>at this time</u>. With the continued outcry from the American public for an answer to the question of nonionizing energy safety, rather than more scientific double-talk, eventually governmental safety standards will be established. When these standards will be established is not even being addressed by individuals with the power to do so.

Recommendations

1. Any radar units being considered for acquisition should be checked for certification by the IACP's Consumer Products List[V].

2. It is imperative that patrol radar equipment continue to be checked on a consistent basis. Any unit found operating outside of manufacturers specifications and/or ANSI standards should be taken out of service immediately. Efforts should be made to determine what event(s) occurred to cause the malfunction and corrective action taken.

3. Patrol proactive initial and ongoing training must be performed to ensure radar equipment is being used for speed monitoring as intended by the manufacturer such as:

    a. Ensuring officers avoid any unnecessary exposure. For instance, turning the unit off whenever officers are not checking for speed compliance.

    b. Avoid directing the unit in such a manner that it is transmitting directly at any vehicle occupant.

V Appendix K, Testing of Police Traffic Radar Device, IACP, Appendix A.

4. Keep officers informed of any changes in the current situation. The Patrol must maintain a proactive position in keeping employees informed of all relevant nonionizing radiation developments. Up front and candid communication should remove any disproportionate feeling of anxiety.

5. It is essential that the National Highway Traffic Safety Administration establish standards for safe exposure of officers to nonionizing radiation. There is no such standard at this time.

Glossary

Alzheimers disease - a degenerative disorder typified by a buildup of aluminium in brain tissue supposed to have a genetic origin.

Amplitude-Modulated (adj.) - to vary the maximum wave length, associated with radio.

Carcinogenic - any substance that produces cancer.

Cytotoxicity (adj.) - a substance having a toxic effect on cells.

DNA (Deoxyribonucleic Acid) - an essential component of all living matter and a basic material in the chromosomes of the cell nucleus: it contains genetic code and transmits the hereditary pattern.

Gigahertz - GHz - One billion cycles per second. see hertz.

Hertz - the international unit of frequency, equal to one cycle per second.

Ionizing - to change or be changed into ions (charged particles).

Kinase - a substance that converts a zymogen into an enzyme.

Leukocytes - a white or colorless nucleated cell that appears for our purposes in areas of inflammation or infection. Example: white blood cells

Lymphocyte - a colorless weakly motile cell constituting 20 to 30% of normal human blood. See Leukocyte.

Megahertz - MHz - One million cycles per second. see hertz.

Microwave - designating or of that part of the electromagnetic spectrum generally regarded as lying between 300 GHz and 300 MHz.

Milliwatts per centimeter squared - one thousandth of a watt for each centimeter squared.

Mutagen - any agent or substance capable of noticeably increasing the frequency of some inheritable characteristic (Mutation).

Nonionizing - cannot change or be changed into ions (Charged particles).

Radar - any of several systems or devices using transmitted and reflected radio waves for detecting a reflecting object.

Sinusoidally - (adj.) the curve whose ordinates are proportional to the sines of the abscissas with the equation y = a sin x.

Teratesm - anomaly of organic form and structure.

Watts per kilogram - one watt for each kilogram (appx. 2.2 Lbs.).

Wavelength - The distance, measure in the direction of progression of a wave, from any given point to the next point characterized by the same phase.

Zymogen - a protecolytic enzyme that is secreted in living cells and can be activated by catalyses (as by a kinase. see kinase).

ANSI C95.1-1982
(Revision of ANSI
C95.1-1974)

American National Standard Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 300 kHz to 100 GHz

Co-Secretariat

Institute of Electrical and Electronics Engineers
US Department of the Navy
(Naval Electronic Systems Command)

Approved July 30, 1982

American National Standards Institute

The Institute of Electrical and Electronics Engineers, Inc
345 East 47th Street, New York, NY 10017

.A51

American National Standard

An American National Standard implies a consensus of those substantially concerned with its scope and provisions. An American National Standard is intended as a guide to aid the manufacturer, the consumer, and the general public. The existence of an American National Standard does not in any respect preclude anyone, whether he has approved the standard or not, from manufacturing, marketing, purchasing, or using products, processes, or procedures not conforming to the standard. American National Standards are subject to periodic review and users are cautioned to obtain the latest editions.

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Foreword

(This Foreword is not a part of ANSI C95.1-1982, American National Standard Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 300 kHz to 100 GHz.)

In 1960, the American Standards Association approved the initiation of the Radiation Hazards Standards project under the co-sponsorship of the Department of the Navy and the Institute of Electrical and Electronics Engineers. The scope was defined as follows:

"Hazards to mankind, volatile materials and explosive devices which are created by man-made sources of electromagnetic radiation. The frequency range of interest extends presently from 10 kHz to 100 GHz. It is not intended to include infrared, X-rays, or other ionizing radiation."

Presently, the Co-Secretariat of the American National Standards Committee C95 are the Naval Electronic Systems Command of the Department of the Navy and the Institute of Electrical and Electronics Engineers. The committee has six subcommittees concerned with:

I Techniques, Procedures, and Instrumentation
II/III Terminology and Units of Measurements
IV Safety Levels and/or Tolerances with Respect to Personnel
V Safety Levels and/or Tolerances with Respect to Electro-Explosive Devices
VI Safety Levels and/or Tolerances with Respect to Flammable Materials
VII Medical Surveillance

To date, three standards, one guide and one recommended practice of the C95 Committee have been issued. They are:

ANSI C95.1-1974, American National Standard Safety Level of Electromagnetic Radiation With Respect to Personnel

ANSI C95.2-1981, American National Standard Radio Frequency Radiation Hazard Warning Symbol

ANSI C95.3-1979, American National Standard Techniques and Instrumentation for the Measurement of Potentially Hazardous Electromagnetic Radiation at Microwave Frequencies

ANSI C95.4-1978, American National Standard Safety Guide for the Prevention of Radio-Frequency Radiation Hazards in the Use of Electric Blasting Caps

ANSI C95.5-1981, American National Standard Recommended Practice for the Measurement of Hazardous Electromagnetic Fields — RF and Microwave

This standard prescribes recommended radiation protection guides to prevent biological injury from exposure to electromagnetic radiation. Major revisions of the original version of this standard, ANSI C95.1-1974, were made to take into account the significant expansion of the data base, improvement in the dosimetry, and the increasing number of people in the general population exposed to radio-frequency fields. The changes in the standard include a wider frequency coverage, frequency dependence resulting from the recognition of whole body resonance and incorporation of dosimetry, expanded application to the general population and guidelines for partial body and near field exposures.

The standard contains a detailed discussion (Section 6) of both the rationale and the limitations of the recommended guidelines based on the present data base. Sufficient information concerning the effects of various parameters such as modulation and long-term exposure on the Radio Frequency Protection Guide is not available to substantiate further refinement of the guide. However, as more knowledge is gained, the guide may be modified.

Suggestions for improvement gained from using this standard are welcomed. They should be sent to

The American National Standards Institute
1430 Broadway
New York, NY 10018.

The Standards Committee on Radio Frequency Radiation Hazards, C95, which reviewed and approved this standard had the following membership at the time of approval:

Saul Rosenthal, *Chairman* Stephen Caine, *Secretary*

Organization Represented	*Name of Representative*
Alliance of American Insurers	W. Uber
American Industrial Hygiene Association	C. W. Bickerstaff
American Insurance Association	J. Philip Berggren
	F. Schadt (Alt)
American Petroleum Institute	L. R. Yoder
Armed Forces Institute of Pathology	James Zimmerly
Association of Home Appliance Manufacturers	John Osepchuk
	John Weizerorick (Alt)
Consumers Union	A. Eckhause
	M. Florman (Alt)
DoD (Defense Nuclear Agency)	P. Tyler
Electronic Industries Association	H. Johnson
	A. Wilson (Alt)
Environmental Protection Agency	David E. Janes
	William A. Mills (Alt)
Health Physics Society	William Ham, Jr
	C. S. Parker (Alt)
Institute of Electrical and Electronic Engineers	R. Baird
	C. Frederick
	L. H. Montgomery
	S. Rosenthal
	N. H. Shepherd
International Microwave Power Institute	C. Buffler
Mine Safety Appliance Company	J. P. Lytle
National Council for Radiation Protection	Edwin Alpen
	J. A. Spahn (Alt)
Natural Resources Defense Council	L. Slesin
Office of the Assistant Secretary of Defense (H&E)	G. Kush
Secretary of Automotive Engineers	D. Weber
Telephone Group	R. C. Petersen
	R. Patterson (Alt)
Underwriters Laboratories, Inc.	L. S. Homa
	J. Drengenberg
US Department of Commerce	R. C. Baird
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Contents

American National Standard Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 300 kHz to 100 GHz

1. Scope and Purpose

Recommendations are made to prevent possible harmful effects in human beings exposed to electromagnetic fields in the frequency range from 300 kHz to 100 GHz. These recommendations are intended to apply to non-occupational as well as to occupational exposures. These recommendations are not intended to apply to the purposeful exposure of patients by or under the direction of practitioners of the healing arts.

2. Definitions

radio frequency protection guides (RFPG). The radio frequency field strengths or equivalent plane wave power densities which should not be exceeded without (1) careful consideration of the reasons for doing so, (2) careful estimation of the increased energy deposition in the human body, and (3) careful consideration of the increased risk of unwanted biological effects.

specific absorption rate (SAR). The time rate at which radio-frequency electromagnetic energy is imparted to an element of mass of a biological body.

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4. Recommendations

4.1 Radio Frequency Protection Guides. For human exposure to electromagnetic energy at radio frequencies from 300 kHz to 100 GHz, the protection guides, in terms of the mean squared electric (E^2) and magnetic (H^2)

Table 1
Radio Frequency Protection Guides

1 Frequency Range (MHz)	2 E^2 (V^2/m^2)	3 H^2 (A^2/m^2)	4 Power Density (mW/cm^2)
0.3 — 3	400 000	2.5	100
3 — 30	4000 ($900/f^2$)	0.025 ($900/f^2$)	$900/f^2$
30 — 300	4000	0.025	1.0
300 — 1500	4000 ($f/300$)	0.025 ($f/300$)	$f/300$
1500 — 100 000	20 000	0.125	5.0

Note: f = frequency (MHz).

field strengths and in terms of the equivalent plane-wave free-space power density, as a function of frequency, are given in Table 1.

For near field exposures, the only applicable protection guides are the mean squared electric and magnetic field strengths as given in Table 1, columns 2 and 3. For convenience, these guides may be expressed as the equivalent plane wave power density, given in Table 1, column 4.

For mixed or broadband fields at a number of frequencies for which there are different values of protection guides, the fraction of the radio frequency protection guide incurred within each frequency interval should be determined, and the sum of all such fractions should not exceed unity.

4.2 Exclusions

(1) At frequencies between 300 kHz and 100 GHz, the protection guides may be exceeded if the exposure conditions can be shown by laboratory procedures to produce specific absorption rates (SARs) below 0.4 W/kg as averaged over the whole body, and spatial peak SAR values below 8 W/kg as averaged over any one gram of tissue.

(2) At frequencies between 300 kHz and 1 GHz, the protection guides may be exceeded if the radio frequency input power of the radiating device is seven watts or less.

4.3 Measurements

(1) For both pulsed and non-pulsed fields, the power density, the squares of the field strengths, and the values of specific absorption rates (SARs) or input power, as applicable, are averaged over any 0.1 h period. The time-averaged values should not exceed the values given in Table 1 or in the Exclusions, 4.2.

(2) Measurements to determine adherence to the recommended protection guides shall be made at distances 5 cm or greater from any object (refer to ANSI C95.3-1979 [39][1]).

5. Explanation

Exposure to electromagnetic fields in the frequency range under consideration is but one of the several sources of energy input into the body, which requires wide ranges of energy production and dissipation in order to function. For situations involving unrestricted exposure of the body, the radio frequency protection guides are believed to result in energy deposition averaged over the entire body mass for any 0.1 h period of about 144 joules per kilogram (J/kg) or less. This is equivalent to a specific absorption rate (SAR) of about 0.40 watts per kilogram (W/kg) or less, as spatially and temporally averaged over the entire body mass.

Biological effects data applicable to humans for all possible combinations of frequency and modulation do not exist. The radio frequency protection guide, therefore, has been based on the best available interpretations of the literature and is intended to eliminate adverse effects on the functioning of the human body.

Exclusion criterion (2) to the protection guides can be used in relation to fields from low power devices such as hand-held, mobile, and marine radio transceivers. These devices may emit localized fields exceeding the protection guides, but will result in a significantly lower rate of energy absorption than allowed for the whole body average. Thus, exposure to

[1] The numbers in brackets correspond to the References listed in Section 3 of this standard.

fields emitted by devices operating at 1 GHz or lower and at less than 7 W output power would not be restricted. Exposure to fields from devices with greater output power or operating at frequencies above 1 GHz require a case-by-case analysis to determine if exclusion criterion (1) is applicable.

Because of the limitations of the biological effects data base, these guides are offered as upper limits of exposure, particularly for the population at large. Where exposure conditions are not precisely known or controlled, exposure reduction should be accomplished by reliable means to values as low as are reasonably achievable. Exposures slightly in excess of the radio frequency protection guides are not necessarily harmful, however, such exposures are *not* desirable and should be prevented wherever possible.

6. Rationale

American National Standards Institute (ANSI) policy requires that each of its standards or guides shall be reviewed at five year intervals. At the time of expiration, the standard or guide may be retained, revised, or rescinded in accord with the consensus of the reviewing body. In 1974, the members of the reviewing body, ANSI Subcommittee C95-IV, retained most of the provisions of the previous guide, but qualified the recommended exposure limits on power densities by specifying limits on strengths of both field components (electrical and magnetic) of radio-frequency electromagnetic (rfem) fields.

During 1978, 1979, and 1980, members of Subcommittee C95-IV met on several occasions to discuss the 1974 guide and to review data and developments that had been forthcoming since its publication. From these discussions and reviews consensus was reached on a number of issues and concerns. First, no verified reports exist of injury to or adverse effects on the health of human beings who have been exposed to rfem fields within the limits of frequency and power density specified by previous ANSI guides. Second, in spite of the absence of verified reports of injury, the physical and biological data upon which earlier guides have been based are quite limited. Moreover, previous guides were based on the assumption that only gross thermal effects, those borne of elevations of core temperature, are potential causes of biological reactivity. While recognizing the dangers of excessive elevations of temperature [1], the subcommittee also recognized the dangers of prejudgment in light of unsettled questions of field-body mechanisms of interaction and of emerging data that indicate the existence of athermal effects [3], [17], [25], [27]. Third, the subcommittee recognized that previous ANSI guides have been interpreted widely as occupational standards, applicable only to settings where the health status of exposed personnel is known and the working environment is under control of the operator of a source of rfem fields. In view of the rapidly expanding use of private and public sources such as citizen's band radio, and mobile and marine transmitters, and in recognition that FM and TV broadcasts constitute dominant sources of rfem fields in the environment of the average citizen, the subcommittee recognized the need for a general-population guide. And fourth, the subcommittee recognized that previous ANSI guides have provided the basis for almost all national and industrial standards of human exposure to rfem fields. Accordingly, withdrawal of an ANSI guide was considered highly undesirable. Retention of the 1974 guide was also viewed as undesirable in the light of new data and developments [16]. The decision was made to revise the guide in spite of acknowledged gaps that persist in the existing base of data [17], [22], [27].

The 1982 Radio Frequency Protection Guide (RFPG) is an extension of its 1974 predecessor with several notable refinements.

6.1 Recognition of Whole-Body Resonance. As is true of the 1974 guide, the 1982 RFPG is based on recommendations of maximal permissible limits (MPL) of field strength or of plane-wave-equivalent power densities of incident fields, but these limits are based on now well established findings that the body as a whole exhibits frequency-dependent rates of absorbing rfem energy [1], [10], [11], [13]. Whole-body-averaged absorption rates approach maximal values when the long axis of a body is parallel to the *E*-field vector and is four tenths of a wavelength of the incident field. At 2450 MHz, for example, Standard Man (long axis 175 cm) will absorb about half of the incident rfem 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 sevenfold increase of absorption relative to that in a 2450 MHz field [14], [15]. In consideration of this dependency, recommended MPLs of field strength have been reduced across the range of frequencies in which human bodies from small infants to large adults exhibit whole-body resonance.

6.2 Incorporation of Dosimetry. Dosimetry is the fundamental process of measuring physical quantities of energy or substances that are imparted to an absorbing body [23], [24]. In 1972, The National Council on Radiation Protection and Measurements (NCRP) convened Scientific Committee 39 to deliberate and recommend dosimetric quantities and units applicable to rfem fields [30]. In keeping with the NCRPs recommendations, the ANSI subcommittee adopted the unit-mass, time-averaged rate of rfem energy absorption as specified in SI units of watts per kilogram ($W \cdot kg^{-1}$). The quantity expressed by these units is termed the specific absorption rate (SAR) and 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),[2] that is, Joules per kilogram = $J \cdot kg^{-1} = W \cdot s \cdot kg^{-1}$.

Formally defined, the specific absorption rate is the time rate at which radio-frequency electromagnetic energy is imparted to an element of mass of a biological body.

The SAR is applicable to any tissue or organ of interest (that is, can be applied to any macromolecular element of mass) or, as utilized in the 1982 RFPG, is expressed as a whole-body average. Ideally, anatomical distributions of SARs would be used explicitly in formulating a guide in recognition that absorption of rfem energy from even the most uniform field can result in highly variable anatomical depositions of energy. Guy and his colleagues have established through thermographic analyses of models of rats and man, and cadavers of rabbits, that peaks of the SAR can range more than an order of magnitude above a whole-body average [18], [19], [20]. Comparable findings have been reported by Gandhi [14]. However, several factors preclude explicit use of peak SARs: (1) the availability of data on distributive SARs is limited, and (2) SAR distributions are highly variable since they are dependent on wavelength, polarization, and zone of the incident field, and on the mass and momentary geometry of the biological body. The number of the complex family of SAR distributions approaches infinity. It is recognized, however, that a whole-body-averaged SAR is the mean of a distribution, the high side of which is an envelope of electrical hotspots. These localized SARs range from mean to peak and when integrated with localized SARs of less than the mean value, equal the whole-body average. Moreover, for any given orientation of a given species in a given field, the *correlation* between the magnitude of a whole-body-averaged SAR and that of any lower or higher part-body SAR must approach unity, that is, if the power density of an incident rfem field is increased, the relative increase of the whole-body SAR will be directly proportional to the increase of any part-body SAR. Because of the invariable presence of electrical hotspots in the irradiated body and the inherent correlation between magnitudes of whole-body and part-body SARs, a biological effect induced by a localized SAR that is well above the whole-body average will be reflected to some extent by that average. The predictive utility of the correlation between part and whole has long served clinical and experimental medicine in which a whole-body, unit-mass dosimetry underlies therapeutic administration of pharmacological agents.

6.3 Expanded Data Base. The data base from which the 1982 RFPG was developed is considerably broader than that of the 1974 guide [16], [17]. After several hundred reports in the biomedical literature on rfem fields were reviewed by members of working groups of ANSI Subcommittee C95-IV, a select list of experimental reports was compiled in accord

[2] Some authors have used the terms *dose rate* ($\dot{D}$) and *dose* (D) instead of, respectively, *specific absorption rate* and *specific absorption*. The rfem-energy dose rate and the SAR are identical in meaning and definition (as are D and SA). Absorbed power density, when expressed as a volume integral, has also been used as a synonym for the SAR. The SAR is used exclusively in the 1982 RFPG to prevent confusion with dosimetric terminology used in the study and application of ionizing radiations.

with several criteria; they are: demonstrability (that is, positive data), relevance, reproduceability, and dosimetric quantifiability. The reports in the select list (see Table A1, Appendix) cover a wide range of external and internal field strengths associated with positive findings but are not representative of the data base because of the bias toward positive findings. This bias is justified on two grounds: (1) while negative findings are useful in evaluating the generality of data, they cannot displace the positive findings upon which a rational standard must be based, and (2) selection of positive findings injects a degree of worst-case conservatism into the guide and therefore constitutes an additional factor of safety. In compiling the select list of reports, the members of the subcommittee often screened several studies from different laboratories that had yielded positive findings of relevance to a common end point. For example, data from teratological studies of microwave irradiation have been reported in many papers by Roberts Rugh and his associates [35], [36], [37] and by M. E. O'Connor and her associates [31], [32], [33] yet only the report by Berman was chosen for inclusion [4]. This choice was made because positive findings were claimed at much lower field strengths than those employed by other investigators and because the periods of exposing animals are among the longest of any reported in the literature on microwave teratology.

6.4 Broadened Assessment Criteria. In assessing positive reports of biological effects, the subcommittee emphasized studies that had generated evidence of morbidity or debilitation, chronic or acute. The most sensitive measures of biological effects were found to be based on behavior [9], [12], [15], [21], [25], [26]. Because of the paucity of reliable data on chronic exposures, the subcommittee focused on evidence of behavioral disruption under acute exposures, even that of a transient and fully reversible character. The assumption is that reversible disruption during an acute exposure is tantamount to irreversible injury during chronic exposure. The whole-body-averaged SARs associated with thresholds of reversible behavioral disruption were found to range narrowly between 4 and 8 W/kg in spite of considerable differences in carrier frequency (600 MHz to 2.45 GHz), species (rodents versus primates), and mode of irradiation (cavity, waveguide, and plane wave). In contrast, the time-averaged power densities of incident radiation associated with these thresholds of disruption ranged (by calculation or measurement) from 10 to 50 mW/cm^2. During the assessment procedure, classification and judgment of findings were made without prejudgment of mechanisms of effects. The subcommittee's intent was that of protecting exposed human beings from harm by *any* mechanism, including those arising from excessive elevations of body temperature.

6.5 Two-Tier Assessment. After the select list of reports was compiled, each report was evaluated on a case by case basis by the subcommittee's biologically trained scientists. The subcommittee's physical and biological scientists then evaluated the reports in terms of reliability and evidence of adverse effects. The discussion focused on thresholds of adverse effects, the extent to which findings had been verified in independent investigations, and roles played by confounding factors. There was general agreement that adverse effects of acute exposures are associated with whole-body specific absorption rates (SAR) above 5 W/kg. On the other hand, whole-body SARs below 4 W/kg were not by consensus associated with effects that demonstrably constitute a hazard. Some effects reported in the Eastern European literature were discounted because of questionable control procedures and lack of information on environmental parameters and physical measurements. In addition, modulation-specific effects, such as efflux of calcium ions from brain materials [2], [5] were not considered adverse because of the inability of the subcommittee's members to relate them to human health. The narrow ranges of power density and the low and narrow range of modulation frequencies associated with field-induced efflux of calcium ions, and the authors' findings that the phenomenon is reversible, are factors that entered into the subcommittee's deliberations. The consensus remained that reliable evidence of hazardous effects is associated with whole-body-averaged SARs above 4 W/kg.

6.6 Safety Factor. To ensure a wide margin of safety, an order-of-magnitude reduction in the permissible whole-body-averaged specific absorption rate (SAR) to 0.4 W/kg was invoked.

This decision was nearly unanimous; one biological scientist dissented on the grounds that a specific justification should be given for the power-of-ten reduction. Different biological scientists offered different reasons, but beyond the need for a wide margin of safety, no quantitative justification was advanced. None of the members of the subcommittee offered an argument to widen the margin of safety.

It is noted in Fig A3 (see Appendix) that the majority of case reports are in the range of microwave frequencies; most of these reports are based on a frequency at or near 2450 MHz. This narrow data base of frequencies sheds little light on the relative biological effectiveness of rfem radiation as a function of frequency. However, no verified theory that would predict frequency specificity because of possible athermal effects has been advanced. In the absence of any contrary experimental or theoretical evidence the subcommittee assumed no wavelength dependencies beyond those of depth of penetration and whole-body resonance. Given these assumptions, the physical scientists of the subcommittee were asked to determine frequency-dependent limits of exposure. The results of theoretical calculations and experimental modeling of absorbed energy for various conditions of human exposure as reported by several authors are shown in Fig A2 of the Appendix (see legend to Fig A2).

The SAR envelope for plane-wave exposures at 1 mW/cm^2 as a function of frequency was determined for human beings from small infant to large adult (see curve 16, Fig A2). The maximal permissible limit (MPL) was determined from 300 kHz to 100 GHz. The results are shown in Fig A1 of the Appendix. Above 1.5 GHz the curve is assumed to be flat. It should be noted that curve 16, Fig A2, is not extended above 1 GHz, but the general trend of flattening with frequency is indicated by all other curves of Fig A2. As frequency decreased below 30 MHz, the quantity of rfem energy absorbed by human beings of any size decreases substantially. Nonetheless, it was recommended that field strengths at frequencies below 3 MHz be limited to those associated with a plane-wave-equivalent power density of 100 mW/cm^2. This limit is intended to prevent reactions at the body's surface that can occur in *E* fields of high intensity.

The limiting rate of energy absorption of 0.4 W/kg is predicated on a biological body that is located in a linearly polarized plane-wave field, that is, the case in which the long axis of the body is parallel to the vector of the *E* field. This case presumes near-maximal absorption of rfem energy. By adopting the principle of maximal coupling, the subcommittee intended to introduce yet another element of conservatism into the guide.

6.7 Near-Field Exposures. The subcommittee recognized that the assumption of a plane-wave exposure is simplistic and is not a realistic approximation of most exposures that pose a risk to health; such exposures occur in relative close proximity to rfem sources in the near field [8], [28]. Fortunately, because of the highly localized nature of the fields in the near zone, the whole-body-averaged SARs associated with them will be below those induced by plane waves of equivalent field strengths.

6.8 Other Factors. It was recognized by the subcommittee that the specific absorption rate (SAR), which provides the basis for limiting power densities, does not contain all of the factors that could be of importance in establishing safe limits of exposure. First, other characteristics of an incident field such as modulation frequency and peak intensity may pose a risk to health. Again, the data base does not provide the evidence of adversity by which to recommend special provisions for modulated fields. There was an *intuitive* concern by some members of the subcommittee that caution should be exercised when individuals are exposed to a pulse-modulated field of high peak but low averaged density, or to a sinusoidally-modulated field, when either field has a recurrence rate in the range of bioelectric rhythms. A supportable way of expressing this concern, which would be applicable to all exposed populations, could not be reached.

A considerable degree of conservatism has been incorporated in the RFPG to make it applicable to the control of non-occupational as well as to occupational exposures. Accordingly, the need for special considerations of environmental conditions such as extremes of temperature and humidity is averted. Previous guides have recommended reduction of maximum permissible limits (MPLs) of power density in hot, humid environments in recognition of the potential thermal burden imposed thereunder by 10 mW/cm^2 fields [29]. This

RFPG effectively controls the thermally adverse environment by limiting the permissible rate of energy absorption to a level that precludes excessive elevations of body temperature [38].

Some of the members of the subcommittee expressed a concern for toxic chemical agents that might be present in the environment and might, in combination with exposure to rfem fields, constitute a hazard. However, the absence of evidence that toxic agents are potentiated by weak rfem fields led the subcommittee to concur that the 1982 RFPG is sufficiently conservative to make additional precautions unnecessary and, particularly in view of the difficulty of administering such precautions effectively, to advise against their inclusion as an adjunct to the RFPG.

6.9 Restriction on Measurement. The subcommittee recognized that objects immersed in an electromagnetic field at strengths below those specified in Table 1 of the RFPG can produce a scattered field of apparent intensity greatly exceeding that of a primary source. The apparent strength of a scattered field can be enhanced by many orders of magnitude in close proximity to an object and is an inverse function of distance from the object. The apparent strength is also dependent on the geometry of the object. Valid measurement of scattered fields is difficult due to the finite size of the field sensor and to its interaction with the object. It is also recognized that the quantity of energy that can be coupled from a scattered field to a large body (that is, that of a human being) is small compared with that from the primary source. Thus, based on fundamental considerations of scattering properties of absorbing or reflecting objects in an rfem field and on consideration of the practical limitations of measuring instruments, it was agreed that measurements of field strengths to determine adherence to the RFPG are to be made at distances 5 cm or greater from any object.

6.10 Special Exclusion. The subcommittee recognized that many low-power devices that are used by a large segment of the general population, such as citizen's band radio, and amateur, public-safety, land-mobile and marine transmitters, may generate localized fields that appear to exceed the RFPG but result in a significantly lower rate of whole-body-averaged energy absorption as a result of the limited area of exposed tissue. For example, calculation of field strengths and localized power densities in proximity to an ideal 150 MHz quarter-wave antenna mounted on a ground plane with an input power of two watts (Table A2, Appendix) reveal that human exposure at a distance 20 cm or less from the antenna would be prohibited by the RFPG even though the highest possible rate of energy absorption is less than seven percent of that allowed for a whole-body exposure to a plane wave.

6.11 Exclusion. The subcommittee agreed that the only practical way to cope with the problem of low-power devices was to enter an exclusion clause in the RFPG that would allow the power density (and local strengths) of incident fields to be exceeded under certain conditions. The exclusion is based on the following considerations:

(1) It would not violate the general provisions of the RFPG. The whole-body-averaged rate of energy absorption during localized exposure should be less than 0.4 W/kg, and anatomically localized rates should not exceed those that are expected from a whole-body exposure to a plane wave that results in an average specific absorption rate (SAR) of 0.4 W/kg. By implication and demonstration, peak SARs in a biological body can range more than an order of magnitude above the average SAR over a limited mass of the exposed tissue.

(2) It would be unlikely for devices such as low-power hand-held radios operating at frequencies below 1 GHz and radiating at rfem power levels below 7 W to couple enough energy into any size human body to violate the general provisions of the RFPG.

Therefore, the subcommittee included in the standard a provision for the exclusion of a particular source from the general RFPG, provided it could be competently shown that for any individual that might be exposed to emissions from that source the whole-body-averaged SAR would not exceed 0.4 W/kg and that any spatial peak value of the SAR would not exceed 8 W/kg as averaged over any one gram of tissue and over any time period of 0.1 h.

It was also recognized by the subcommittee that to determine whether a particular rf source would meet these absorption criteria

would be difficult and could be done only by a properly qualified laboratory or by an appropriate scientific body for a general class of equipment. In no case could a routine field survey determine conformance with the criteria of this part of the exclusion.

The subcommittee further recognized that it would be unnecessary to validate the dosimetry criteria for the application of the exclusion clause if the maximal input power of the radiating device is seven watts or less. The seven watts that is allowable under the exclusion clause is, by way of comparison, more than an order of magnitude below power levels of equipment that is routinely used in the clinic for part-body treatment by diathermy [40]. Furthermore, it is difficult to envision any operating conditions where more than a small fraction of the rfem energy from a 7 W device could be absorbed by a human body. The 7 W exclusion should be limited to frequencies below 1 GHz to prevent known, adverse biological consequences of exposure to intense collimated beams.

6.12 Time Averaging. The subcommittee retained 0.1 h (6 min) as the significant period of time over which exposures, the values of specific absorption rates (SAR) and input power are to be averaged.

Appendix

(This Appendix is not a part of ANSI C95.1-1982, American National Standard Safety Levels with Respect to Human Exposure to Radio Frequency Electromagnetic Fields, 300 kHz to 100 GHz.)

List of Selected Reports Biological Effects of RFEM Fields[3]

A1. Environmental Factors

JOHNSON, R. B., MIZUMORI, S., LOVELY, R. H., and GUY, A. W. Adaptations to Microwave Exposure as a Function of Power Density and Ambient Temperature in the Rat. *Abstracts, 1978 Symposium on Electromagnetic Fields in Biological Systems.* International Microwave Power Institute, Edmonton, Alberta, 1978, p 30.

MONAHAN, J. C. and HO, H. S. The Effect of Ambient Temperature on the Reduction of Microwave Energy Absorption by Mice. *Radio Science*, 12(6S), 1977, pp 257—262.

SHANDALA, M. G., RUDNEV, M. I. and NAVAKATIAN, M. A. Patterns of Change in Behavioral Reactions to Low Power Densities of Microwaves. *Abstracts, 1977 International USNC/URSI Symposium on the Biological Effects of Electromagnetic Waves.* National Academy of Sciences, Washington, DC, 1977, p 88.

A2. Behavior

D'ANDREA, J. A., GANDHI, O. P. and LORDS, J. L. Behavioral and Thermal Effects of Microwave Radiation at Resonant and Non-Resonant Wavelengths. *Radio Science*, 12(6S), 1977, pp 251—256.

FREY, A. H. Behavioral Effects of Electromagnetic Energy. *Biological Effects and Measurement of Radio Frequency/Microwaves*, D. H. Hazzard (Ed.), HEW Publication (FDA) 77-8026. U.S. Government Printing Office, Washington, DC, 1977, pp 11—22.

FREY, A. H., FELD, S. R. and FREY, B. Neural Function and Behavior: Defining the Relationship. *Annals of the N.Y. Academy of Sciences*, 247, 1975, pp 433—438.

KING, N. W., JUSTESEN, D. R. and CLARKE, R. L. Behavioral Sensitivity to Microwave Irradiation. *Science*, 172, 1971, pp 398—401.

LIN, J. C., GUY, A. W. and CALDWELL, L. R. Thermographic and Behavioral Studies of Rats in the Near Field of 918 MHz Radiation. *IEEE Transactions on Microwave Theory and Techniques*, MTT-25, 1977, pp 833—836.

THOMAS, J. R., FINCH, E. D., FULK, D. W. and BURCH, L. S. Effects of Low-Level Microwave Radiation on Behavioral Baselines. *Annals of the N.Y. Academy of Sciences*, 247, 1975, pp 425—431.

A3. Immunology

CZERSKI, P. Microwave Effects on the Blood-Forming System With Particular Reference to the Lymphocyte. *Annals of the N.Y. Academy of Sciences*, 247, 1975, pp 232—241.

HUANG, A. T., ENGLE, M. E., ELDER, J. A., KINN, J. B. and WARD, T. R. The Effects of Microwave Radiation (2450 MHz) on the Morphology and Chromosomes of Lymphocytes. *Radio Science*, 12(6S), 1977, pp 173—177.

SHANDALA, M. G., RUDNEV, M. I., VINOGRADOV, G. K., BOLONOZHIKO, H. G. and GONCHAR, N. M. Immunological and Hematological Effects of Microwaves at Low Power Densities. *Abstracts, 1977 USNC/URSI International Symposium on the Biological Effects of Electromagnetic Waves*, National Academy of Sciences: Washington, DC, 1977, p 85.

SMIALOWICZ, R. J., KINN, J. E. and ELDER, J. A. Exposure of Rats *In Utero* Through Early Life to 2450 MHz (CW) Microwave Radiation: Effects on Lymphocytes. *Radio Science*, 14(6S), 1979, pp 147—154.

[3] A number of preprints, either published as abstracts or available as editorially accepted manuscripts, were used in preparation of the select list. In each case where a report has been published in an archival journal, reference is to the published paper.

A4. Teratology

BERMAN, E., KIN, J. B. and CARTER, H. B. Observations of Mouse Fetuses After Irradiation with 2.45 GHz Microwaves. *Health Physics*, 35, 1978, pp 791—801.

A5. Central Nervous System/Blood-Brain-Barrier

ALBERT, E. N. Light and Electron Microscope Observations on the Blood-Brain Barrier after Microwave Irradiation. *Biological Effects and Measurements of Radio Frequency/Microwaves*, Hazzard, D. G. (Ed), HEW Publication (FDA) 77-8026 U.S. Government Printing Office, Washington, DC, 1977, pp 294—304.

BAWIN, S. M., KACZMAREK, L. K. and ADEY, W. R. Effects of Modulated VHF Fields on the Central Nervous System. *Annals of the N.Y. Academy of Sciences*, 247, 1975, pp 74—81.

BLACKMAN, C. F., ELDER, J. A., WEIL, C. M., BENANE, S. G., EICHINGER, D. C. and HOUSE, D. E. Induction of Calcium-Ion Efflux from Brain Tissue by Radio-Frequency Radiation: Effects of Modulation Frequency and Field Strength. *Radio Science*, 14(6S), 1979, pp 93—98.

FREY, A. H., FELD, S. R. and FREY, B. Neural Function and Behavior: Defining the Relationship. *Annals of the N.Y. Academy of Sciences*, 247, 1975, pp 433—438.

A6. Cataracts: None ≤ 10 mW/cm^2

A7. Genetics: None ≤ 10 mW/cm^2

A8. Human Studies: None

A9. Thermoregulation and Metabolism

ADAIR, E. R. and ADAMS, B. W. Microwaves Modify Thermoregulatory Behavior in Squirrel Monkeys. *Bioelectromagnetics*, 1, 1980, pp 1—20.

DE LORGE, J. W. Operant Behavior and Colonic Temperature of Squirrel Monkeys During 2450 MHz Microwave Irradiation. *Radio Science*, 14(6S), 1979, pp 217—226.

LOVELY, R. H., MYERS, D. E. and GUY, A. W. Irradiation of Rats by 918 MHz Microwaves at 2.5 mW/cm^2: Delineating the Dose-Response Relationship. *Radio Science*, 12(6S), 1977, pp 139—146.

LU, S. and T., LEBDA, N., MICHAELSON, S. M., PETTIT, S. and RIVERA, D. Thermal and Endocrinological Effects of Protracted Irradiation of Rats by 2450 MHz Microwaves. *Radio Science*, 12(6S), 1977, pp 147—156.

STERN, S., MARGOLIN, L., WEISS, B., LU, S. and T. and MICHAELSON, S. M. Microwaves: Effect on Thermoregulatory Behavior in Rats. *Science*, 206, 1979, pp 1198—1201.

A10. Biorhythms

LU, S. and T., LEBDA, N., MICHAELSON, S. M., PETTIT, S. and RIVERA, D. Thermal and Endocrinological Effects of Protracted Irradiation of Rats by 2450 MHz Microwaves. *Radio Science*, 12(6S), 1977, pp 147—156.

A11. Endocrinology

LOVELY, R. H., Guy, A. W., JOHNSON, R. B. and MATHEWS, M. Alterations of Behavioral and Biochemical Parameters During and Consequent to 500 μW/cm^2 Chronic 2450 MHz Microwave Exposure. *Abstracts, 1978 Symposium on Electromagnetic Fields in Biological Systems*, International Microwave Power Institute: Edmonton, Alberta, 1978, p 34.

LU, S. and T., LEBDA, N., MICHAELSON, S. M., PETTIT, S. and RIVERA, D. Thermal and Endocrinological Effects of Protracted Irradiation of Rats by 2450 MHz Microwaves. *Radio Science*, 12(6S), 1977, pp 147—156.

TRAVERS, W. D. Low Intensity Microwave Effects on the Synthesis of Thyroid Hormones and Serum Proteins, *Health Physics*, 33, 1978, p 662.

A12. Development

GUILLET, R. and MICHAELSON, S. M. The Effect of Repeated Microwave Exposure on Neonatal Rats. *Radio Science*, 12(6S), 1977, pp 125—129.

JOHNSON, R.B., MIZUMORI, S. and LOVELY, R. H. Adult Behavioral Deficits in Rats Exposed to 918 MHz Microwaves. *Developmental Toxicology of Energy Related Pollutants*, M. SIKOV and D. MALUM (Eds), 17th Hanford Symposium. Department of Energy, Washington, DC, 1979, pp 281—299.

MC REE, D. I. and HAMRICK, P. E. Exposures of Japanese Quail Embryos to 2.45 GHz Microwave Radiation during Development. *Radiation Research*, 71, 1977, pp 355—366.

A13. RF Hearing: None

A14. Hematology

MIRO, L., LUBIERE, R. and PFISTER, A. Effects of Microwaves on the Cell Metabolism of the Reticulo-Histiocytic System. *Biologic Effects and Health Hazards of Microwave Radiation*, Czerski, P. (Ed), Polish Medical Publishers, Warsaw, 1974, pp 89—97.

MITCHELL, D. S., SWITZER, W. G. and BRONAUGH, E. L. Hyperactivity and Disruption of Operant Behavior in Rats After Multiple Exposures to Microwave Radiation. *Radio Science*, 12(6S), 1977, pp 263—271.

A15. Cardiovascular

REED, J. R., LORDS, J. L. and DURNEY, C. H. Microwave Irradiation of the Isolated Rat Heart After Treatment with ANS Blocking Agents. *Radio Science*, 12(6S), 1977, pp 161—165.

Table A1
Calculation of Whole-Body SARs for Data in Selected Reports

Research Paper	Reference	Subject and Mass	Orientation to *E* Field	Frequency (GHz)	W/kg per mW/cm²	Modulation	Average Power Density (mW/cm²)	Peak Power Density (mW/cm²)	Duration of Exposure	Average SAR (W/kg)
M. Shandala	A1	Rat (310 g)	Not Specified	2.375	0.21 (medium rat)	CW	0.01 — 0.05	Not Applicable	7 h/day 90 days	0.0021 0.0195
R. Johnson	A1	Rat Embryo (in utero) (445 g)	(Circularly Polarized Field)	0.918	0.30 (large rat)	CW	5	Not Applicable	380 h total	1.5
Monahan and Ho	A1	Mouse (30—34 g)	Waveguide	2.45	SAR Measured Directly	CW	Indeterminate	Not Applicable	20 min	0.6
D'Andrea	A2	Rat (420—450 g)	Long Axis ∥ to E Vector	0.60	0.61	CW	10	Not Applicable	≤ 55 min Repeated, Variable	6.1
Thomas	A2	Rat	Free-moving Animal	2.86 9.6	0.20 — 0.16	1 μs Pulses 500 pps	5	Unknown	1 h	1.0 Far Field 0.8
Frey	A2	Rat (250 g)	Horizontal	1.3	0.36 (medium rat)	0.5 ms Pulses 1000 pps	0.65	1.3 mW/cm²	5 min	Average: 0.23 Maximum: 0.47
King	A2	Rat (400 g)	(Cavity)	2.45	0.22 (estimate)	Sinusoid 60 Hz	Indeterminate	Indeterminate	60 s Repeated	0.6
Frey	A2	Rat (medium)	Various Orientations	1.2	0.35	Pulsed CW	0.2 2.4	2.1 mW/cm² 2.4 mW/cm²	30 min 30 min	Average: 0.07 Maximum: 0.86
Lin	A2	Rat	Near Field	0.918	0.9	CW	32 40	Not Applicable	15 min 5 min	7.2 8.9
M. Shandala	A3	Rat (medium)	Dorsal, Group	2.375	0.21	CW	0.01	Not Applicable	90 days	0.0021
Czerski	A3	Mouse (20 g)	Dorsal, Group	2.95	1.1	1 μs Pulses 1200 pps	0.5	420.0	6—12 wks	0.55
Huang	A3	Hamster (35 g)	Dorsal, Group Far Field	2.45	1.1	CW	5	Not Applicable	15 min/day for 5 days	5.5
Smialowicz	A3	Neonatal Rat (50—90 g)	Dorsal, Individual	2.45	0.7 — 4.7	CW	5	Not Applicable	60 days	0.7 — 4.7
Berman	A4	Mouse (25—33 g)	Dorsal, Group	2.45	0.8	CW	3.4 — 28.0	Not Applicable	100 min/day	2.0 — 22.2
Bawin	A5	Chick Brain *in vitro*	(Parallel Plate)	0.147	< .003	Sinusoid (0.5 — 32 Hz) AM > 90%	< 1	—	20 min	0.003
Blackman	A5	Chick Brain *in vitro*	(Crawford Cell)	0.147	< .002	Sinusoid (0, 3, 9, 16, 30 Hz)	0.75	—	20 min	0.0023

Table A1 (Continued)
Calculation of Whole-Body SARs for Data in Selected Reports

Research Paper	Reference	Subject and Mass	Orientation to *E* Field	Frequency (GHz)	W/kg per mW/cm²	Modulation	Average Power Density (mW/cm²)	Peak Power Density (mW/cm²)	Duration of Exposure	Average SAR (W/kg)
Frey	A5	Rat (225 g)	Various Orientations	1.2	0.36 0.36	Pulsed CW	0.2 2.4	2.1 2.4	30 min 30 min	0.06 0.72
Albert	A5	Chinese Hamster (35 g)	Not Specified	2.45	1.0	CW	10	Not Applicable	1 h or 8 h for 1 day	10
Lovely	A9	Rat (316—388 g)	(Circularly Polarized Waveguide)	0.918	0.36	CW	2.5	Not Applicable	8/h/day for 13 weeks	0.9
Stern	A9	Rat (385—400 g)	Dorsal	2.45	0.18	CW	5	Not Applicable	15 min Intermittent	0.9
Adair	A9	Squirrel Monkey (1 kg)	Long Axis ‖ to E Vector	2.45	0.13	CW	6	Not Applicable	15 min Intermittent for 3 h	0.78
DeLorge	A9	Squirrel Monkey	Dorsal to Head	2.45	0.13	AM (120 Hz)	50	—	30—60 min	6.5
Lu	A9 A10 A11	Rat (150 g)	Dorsal	2.45	0.36	CW	1	Not Applicable	8 h	0.35
Travers	A11	Rat	Not Specified	2.45	0.21	CW	8	Not Applicable	8 h/day 0, 7, 14 or 21 days	1.65
Lovely	A11	Rat (300—350 g)	(Circularly Polarized Waveguide)	2.45	0.21	CW	0.5	Not Applicable	7 h/day for 3 months	0.11
Guillet and Michaelson	A12	Rat (Neonatal) (10—25 g)	Dorsal	2.45	1.3	CW	10	Not Applicable	1 h	13
McRee and Hamrick	A12	Japanese Quail Embryo (10 g)	Long Axis ‖ to E Vector	2.45	0.8	CW	5	Not Applicable	12 days	4
Johnson	A12	Rat (290—310 g)	(Waveguide)	0.918	0.5	CW	5	Not Applicable	20 h/day for 19 days	2.5
Mitchell	A14	Rat (300 g)	15/Group (Cavity)	2.45	0.5	CW	2.3	Not Applicable	1 or 5 h/day 110 days	1.2
Miro	A14	Large Mouse (26—38 g)	Horizontal	3.10	0.9	Pulsed 5 Hz—1 ms	2	Not Applicable	145 h	1.8
Reed	A15	Isolated Rat Heart	Parallel-Plate Exposure	0.96	—	CW	Indeterminate	Not Applicable	10 min	1.5

Table A2
Field Strengths (E and H) and Power Densities in Proximity to a Current-Fed, Quarter-Wave, Radiating Monopole Antenna

A 50 cm antenna operating at 150 MHz and 2 W of input power is assumed, as an input impedance of 36 Ω. Each asterisk associated with a set of field measurements denotes the spatial point of measurement in relation to the antenna. The vertical and horizontal distance between adjacent points of measurement is 10 cm. (Calculations based on [41].)

	Distance in Centimeters			
0	10	20	30	40
E = 64.3 V/m H = 0.00 A/m PD = 1.10 mW/cm²	48.7 0.0217 0.628	32.7 0.0317 0.287	24.2 0.0341 0.156	19.3 0.0348 0.0985
*	*	*	*	*
	95.1 0.0524 2.40	45.7 0.0524 0.553	29.8 0.0489 0.236	22.4 0.0456 0.133
	*	*	*	*
	121 0.136 3.88	53.4 0.0856 0.757	33.3 0.0682 0.295	24.5 0.0533 0.159
	*	*	*	*
	110 0.229 3.72	51.7 0.124 0.708	33.3 0.089 0.300	25.0 0.0716 0.193
	*	*	*	*
	83.6 0.306 3.54	43.0 0.157 0.931	30.2 0.108 0.437	24.1 0.0531 0.261
	*	*	*	*
	50.1 0.358 4.82	32.0 0.180 1.22	26.2 0.120 0.548	22.7 0.0910 0.312
	*	*	*	*
	27.7 0.375 5.31	26.3 0.188 1.33	24.3 0.125 0.589	22.1 0.0938 0.332
	*	*	*	*

50 cm Monopole Antenna

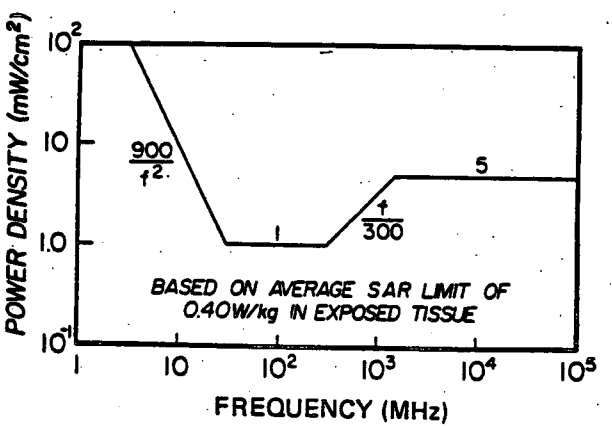
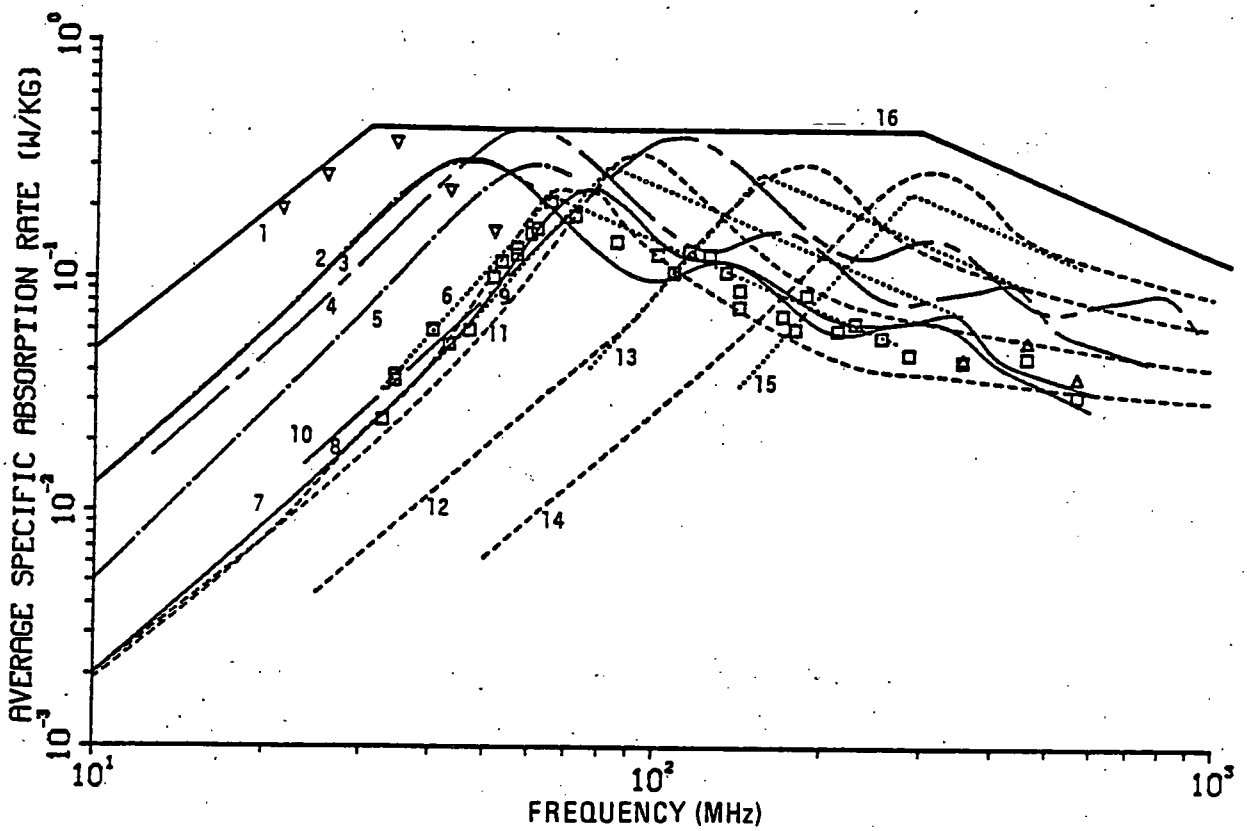


Fig A1
Radio Frequency Protection Guide for Whole-Body Exposure of Human Beings



(1) (Inverted Triangles). Experimental results scaled from a saline-filled, realistic model of an adult human being under grounded conditions.**

(2) (Solid Curve). Numerical calculations based on a block model of man in conductive contact with ground.†

(3) (Chain-Dot). Experimental results based on a realistic model of a human adult in conductive contact with ground.*

(4) (Chain-Dash). Scaling of Curve 2 for ten year old child in conductive contact with ground.

(5) (Chain-Dot). Experimental results based on a realistic model of a human adult 3 cm from a ground plane.*

(6) (Dotted Line). Empirical equation developed for a human adult in free space.**

(7) (Solid Line). Numerical calculations for a block model of man in a free field; experimental data are shown as open squares and experimental data on models are shown as open triangles.**†

(8) (Dashed Line). Prolate spheroidal model of man in a free field.*

(9) (Dotted Line). Empirical equations for a ten year old child.**

(10) (Chain-Dash). Scaling of Curve 2 for a one year old child in conductive contact with ground.

(11) (Dashed Line). Prolate spheroidal model for a ten year old child.*

(12) (Dashed Line). Prolate spheroidal model for a one year old child.*

(13) (Dashed Line). Empirical equations for a one year old child.

(14) (Dashed Line). Prolate spheroidal model of a human infant.*

(15) (Dot). Empirical equation for a human infant.**

(16) Upper limit of the SAR for human beings of all ages and body mass.

*DURNEY, C. H., JOHNSON, C. C., BARBER, P. W., MASSOUDI, H., ISKANDER, M. F., LORDS, J. L., RYSER, D. K., ALLEN, S. J. and MITCHELL, J. C. *Radiofrequency Radiation Dosimetry Handbook*, Second Edition, May 1978, Report SAM-TR-78-22, USAF School of Aerospace Medicine: Brooks Air Force Base, Texas, 1978.

**GANDHI, O. P., HUNT, D. L. and D'ANDREA, J. A. Deposition of Electromagnetic Energy in Animals and in Models of Man With and Without Grounding and Reflector Effects. *Radio Science*, 12(6S), 1977, pp 39—48.

†HAGMANN, M. J. and GANDHI, O. P. Numerical Calculations of Electromagnetic Energy Deposition in Models of Man with Grounding and Reflector Effects. *Radio Science*, 14(6S), 1979, pp 23—29.

Power Density = 1 mW/cm². The results of various investigators are used for cross comparison. The outer envelope, curve 16, is the upper-limit SAR for the range of human beings from infant to adult (see legend).

Fig A2
Whole-Body-Averaged SAR for a Human Adult, a 10 Year Old Child, a 1 Year Old Child, and a Human Infant

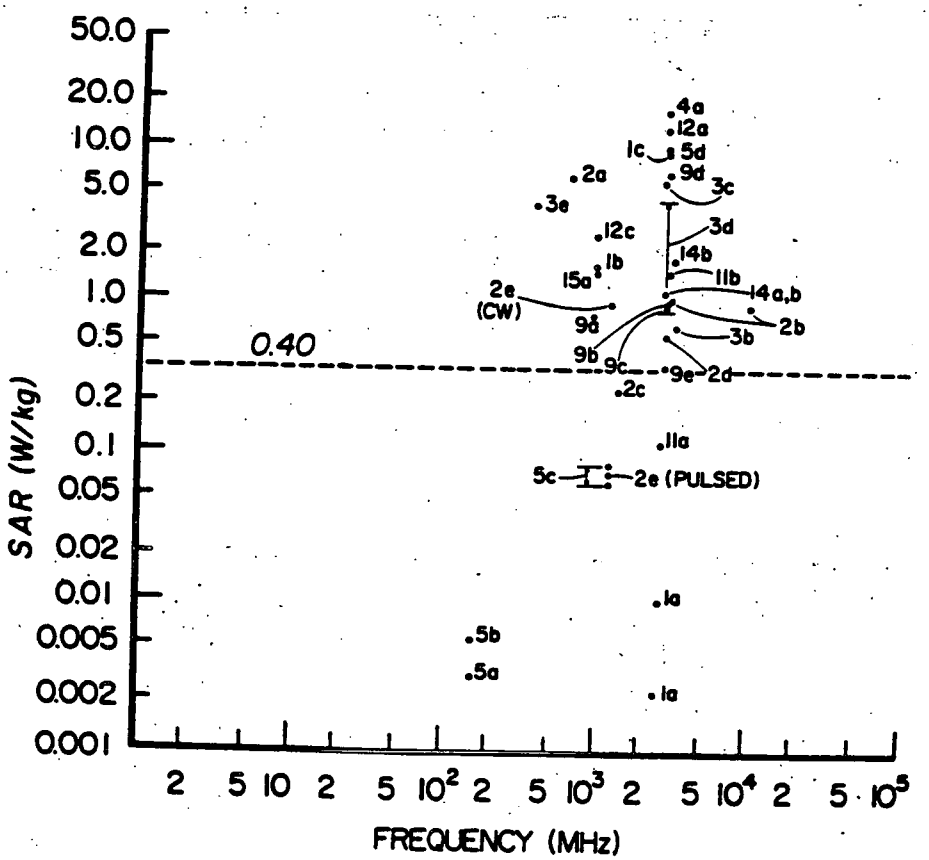
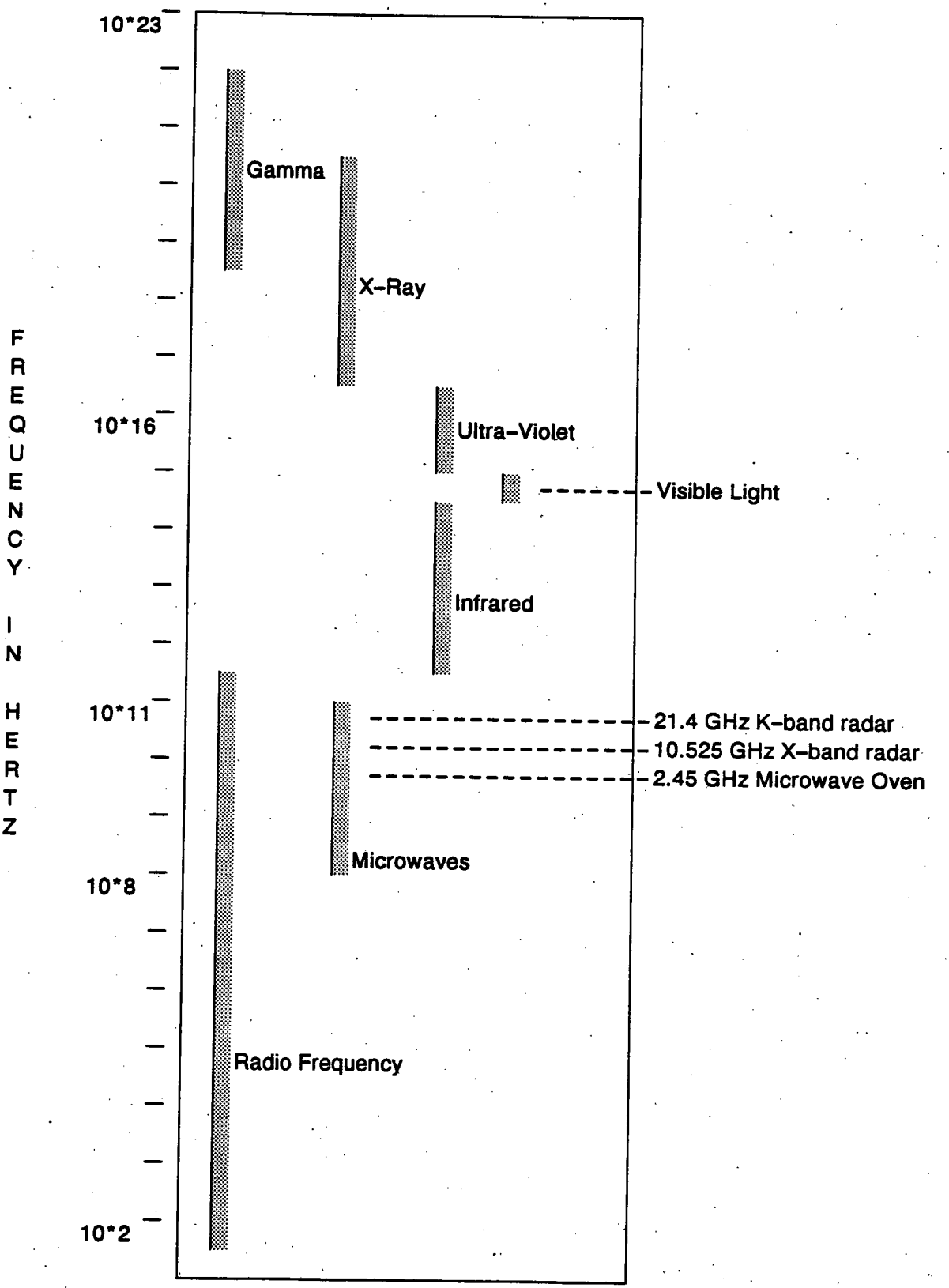


Fig A3
Whole-Body-Averaged SAR
Corresponding to Biological Effects Reported in
Various References of Appendix

Electromagnetic Spectrum
General Approximations



Based on graph published in Scientific American September 1986 vol 225 num 3.

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Field Strength Measurements of Speed Measuring Radar Units

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Prepared by
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U.S. DEPARTMENT OF COMMERCE, Malcolm Baldrige, *Secretary*
NATIONAL BUREAU OF STANDARDS, Ernest Ambler, *Director*

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Foreword

The National Highway Traffic Safety Administration (NHTSA) is engaged in a continuing effort to fulfill the objectives of the Highway Safety Act of 1968. Accordingly, NHTSA has a particular interest in devices used for measuring vehicle speeds for law enforcement purposes. In August 1977 the NHTSA entered into an interagency agreement, DOT-HS-7-01697, with the National Bureau of Standards (NBS) for the purpose of developing performance standards and a qualified product list of speed measuring devices used for law enforcement purposes.

Modification 7 to the interagency agreement, initiated in July 1979, provided for the measurement of the strength of microwave fields surrounding typical speed measuring radar devices, both in free space and in a typical working environment. This research and testing has been performed by the personnel of the Electromagnetic Fields Division under the cognizance of the Law Enforcement Standards Laboratory (LESL) of NBS. The results are included in this document.

Among the tasks performed by the LESL for the NHTSA is the preparation of technical reports on the results of research and testing. This document is one such report. Technical comments and suggestions are invited from interested parties. They may be addressed to the authors, or to the undersigned at the National Bureau of Standards, Washington, DC 20234.

Lawrence K. Eliason, Chief
Law Enforcement Standards Laboratory

Contents

FIELD STRENGTH MEASUREMENTS OF SPEED MEASURING RADAR UNITS

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The objective of this project was to measure the microwave radiation emitted by speed measuring radar units to obtain a data base for evaluating the potential radiation hazards of these devices. Measurements were taken both in free-space and with the radar units mounted in typical operating positions inside or attached to a four-door sedan. The free-space measurements were made at four different distances to determine the field strength as a function of distance from the radar units. Calibrated radiation level probes were used to measure the field strength inside the automobile and scan the interior volume of the four-door sedan with particular attention to the driver and passenger locations. Twenty-two radar units were involved, and the data are presented in a power density format.

Key words: Doppler radar; field intensity; hazard; microwave radiation; power density; radar unit; radiation level; speed measuring radar.

1. INTRODUCTION

The objective of this project was to accurately measure the microwave radiation emitted by speed measuring radar units in order to obtain a data base for evaluating the potential radiation hazards of such devices. To meet this objective, the following two measurement tasks were undertaken.

Task 1. Free-Space Measurements: Direct measurements of the field strength levels surrounding 20 different radar units were made in the laboratory under approximate free-space conditions. In order to obtain data in the side- and back-lobe regions as well as in the direction of the main beam, measurements were made at selected points on an imaginary spherical surface centered at the aperture of the radar unit. Further, measurements were made at several distances in order to determine the field strength as a function of distance from the radar units.

Task 2. Vehicular Measurements: The radar units were mounted inside or attached to a four-door sedan in normal operational configurations. Calibrated radiation level probes were used to measure the field strength inside the automobile and to scan the entire interior volume of the vehicle, with particular attention to the driver and passenger locations. This task provided data on field strengths inside the automobile under simulated operating conditions.

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2. TASK 1 MEASUREMENTS

2.1 General Approach

The purpose of this task was to measure the field intensity in the region surrounding each radar unit and to determine and display the power density as a function of direction and distance from the radar. The measurements were performed at the National Bureau of Standards (NBS) near-field scanning facility employing standard antenna measurement techniques.

The experimental arrangement and coordinate system are shown in figure 1. The radar was mounted on a model mount aimed at a small receiving antenna (probe), and aligned so that the aperture was in the x-y plane and the direction of the main beam was along the z-axis which coincided with the axis of the receiving probe. The radar was then rotated (scanned) a full 360° about the y-axis (θ rotation) and the received signal was recorded at 5° intervals in θ. Next, the radar was rotated by 10° about its own axis (φ rotation) and the θ scan was repeated. (Note that, as shown in figure 1, the φ-rotation axis coincides with the z axis for θ=0.) This process of stepping in φ and scanning in θ was repeated until data had been obtained over the entire measurement sphere enclosing the radar. The complete process was repeated for several separation distances, d.

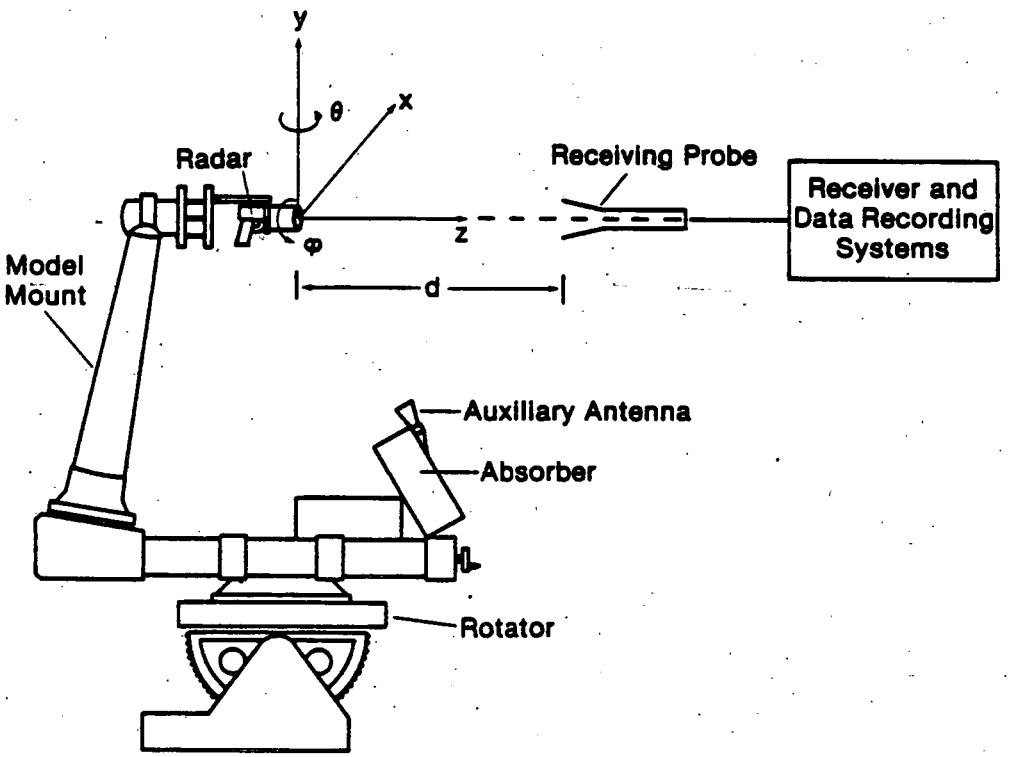


Figure 1. Experimental setup used for measuring electromagnetic field strength in the vicinity of speed radar units.

The received power, P_R, picked up by the probe was determined by means of a calibrated receiving system. The power density, W, incident on the receiving probe, was determined from the relation

$$W = \frac{4\pi P_R}{\lambda^2 G} \qquad (1)$$

In this equation, G is the gain of the probe and λ is the free-space wavelength of the energy emitted by the radar unit [1][1].

2.2 Description of Measurement System

The measurement system is shown schematically in figure 2 as connected for power-density measurements. The signal generator, rotary-vane attenuator (RVA), thermistor, and power meter were only used in the calibration process to be described later. The rest of the system functioned as follows. The electromagnetic (EM) field emitted by the radar was sampled by a small probe antenna. The probe output signal was detected in the mixer and amplified and measured in the receiver. Finally, the amplitude was digitized and recorded on magnetic tape for later computer processing. A coaxial mixer was used for the X-Band measurements and a waveguide mixer was used at K-Band. The receiver used was a calibrated, three-channel, wide-band, phase-amplitude model.

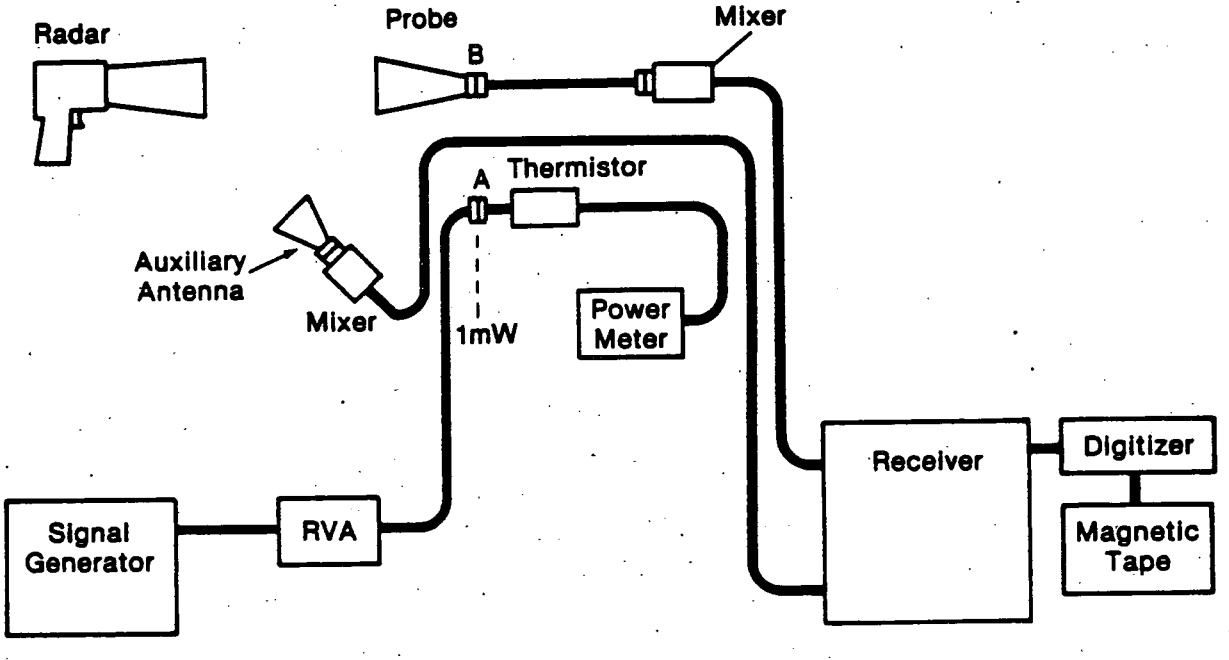


Figure 2. Schematic diagram of measurement system used to determine power density. As shown here, the system is connected for measuring the probe output produced by an incident electromagnetic field. The auxiliary antenna and circuit provide a stable reference signal to which the receiver is phase locked. A 1 mW calibrating signal is established at point A by means of the calibrated thermistor, power meter, and rotary-vane attenuator (RVA).

[1]Numbers in brackets refer to references in appendix A.

In order for the receiver to have sufficient sensitivity to detect the weak signals existing in the side- and back-lobe regions, it must be phase locked to the radar. Since it was not feasible to connect a cable to the radar, the phase-lock signal was provided by means of an auxiliary antenna and mixer which responded to the side-lobe energy radiated by the radar and provided a constant signal for phase locking the receiver. Note that the auxiliary antenna and mixer were mounted on the rotator (fig. 1) so that, as the radar was rotated in azimuth (θ), the relative positions of the radar and auxiliary antenna remained unchanged and the phase-lock signal remained constant within +1.0 dB. As the radar was rotated in ϕ, changes of as much as +3 dB in the amplitude of the phase-lock signals were observed, due mainly to the change in polarization. However, the receiver remained locked during these changes and, since the data were recorded only during θ scans with constant ϕ, these changes of amplitude in the locking signal were of no consequence. The auxiliary antenna was mounted below the probe and well out of the main beam, so there was no significant interaction between them; that is, the auxiliary antenna did not noticeably perturb the field measured by the probe.

The auxiliary antenna used for the X-Band measurements was a broadband conical spiral with Type N connector and it was connected to a coaxial mixer. The K-Band auxiliary system consisted of a standard gain horn and waveguide mixer.

2.3 Calibration of Measurement System

2.3.1 Calibration Procedure

Absolute calibration of the power density measurement equipment was accomplished by means of the signal generator, attenuator, thermistor, and power meter of figure 2. The signal generator provided a calibrating signal of constant amplitude and frequency. The power of this signal at point A was measured by the calibrated thermistor and power meter combination, and accurate changes in this power level were accomplished by means of the RVA. The procedure for carrying out the calibration was as follows.

The radar was set at $\theta=0$ and $\phi=0$, in which case the probe measures the power in the main beam and the probe output signal was near a maximum for that distance. Under these conditions the receiver amplitude was set at 00.00 dB. Next, the signal generator frequency was set equal to the radar frequency and, with the RVA set at 0 dB, the signal generator amplitude was adjusted so that the power meter indicated 1 mW. This means that 1 mW of power was available at A if the thermistor and power meter had been calibrated correctly and all mismatch effects taken into consideration.

The next step was to disconnect the auxiliary mixer from the auxiliary antenna and connect the mixer to the signal generator as shown in figure 3. This link provided the required phase-lock signal for the receiver. The transmission line connecting the RVA to the thermistor was disconnected at A and connected to the primary mixer at B in place of the probe (see fig. 3). Following these connections, the RVA was adjusted until the receiver again read approximately 00.00 dB. The attenuator reading was noted, along with the offset from zero on the receiver. Finally, the circuits were reconnected as in figure 2 to make sure that no significant drift occurred during the calibration.

The above procedure determined the output power from the probe at B with respect to 1 mW. In other words, if the final RVA setting was K dB and the receiver offset was L dB, then the probe output signal would be (K+L) dB below 1 mW. Eq (1) can then be used to calculate the incident on-axis power density, and the power densities at all other points are known since they were measured with respect to the on-axis value.

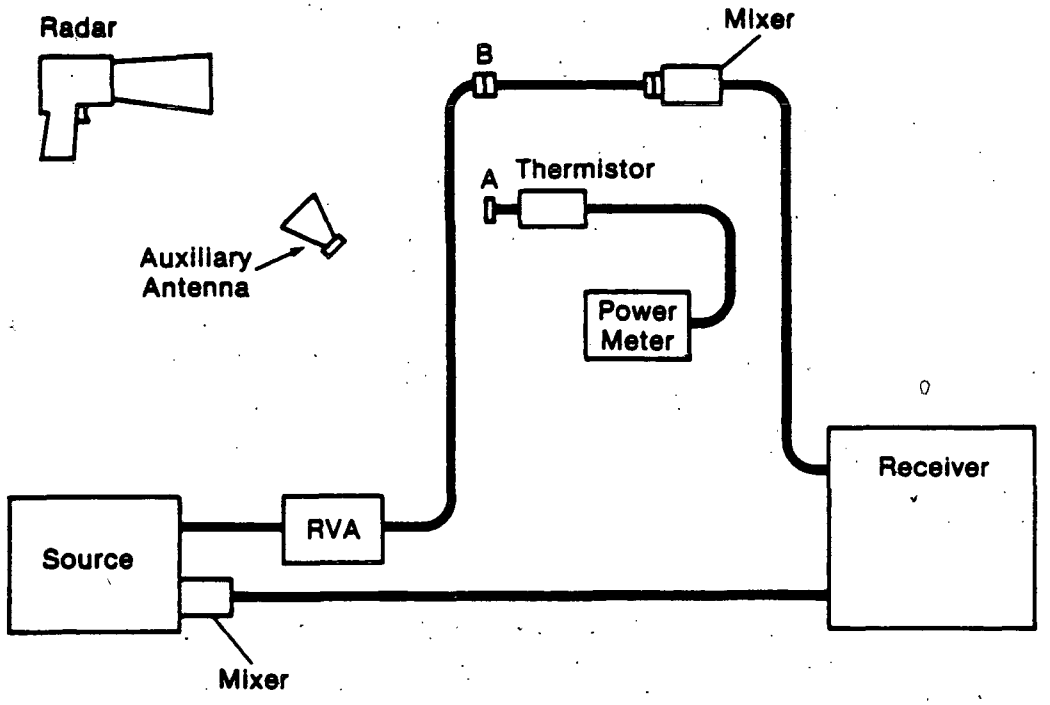


Figure 3. Schematic diagram of measurement system as it is connected for calibrating the probe output signal. The 1 mW calibrating signal is inserted at B and compared with the signal produced by the probe antenna (see sec. 2.3.1 for details).

This calibration procedure was repeated at every measurement distance used for each radar.

2.3.2 Estimated Measurement Accuracy

According to eq (1), the accuracy of the measured power density depends on how accurately the received power is determined by the calibration procedure of the preceding section, and on how well G_p, the gain of the probe, is known. The accuracy of the P_R calibration depends, in turn, on the accuracies of the attenuator, thermistor, power meter, and receiver. An additional factor is the degree of repeatability associated with the disconnection and connection of the transmission lines and probe at points A and B of figures 2 and 3. Any amplitude drifts in system components or in the radar itself will also affect the repeatability.

The RVA is a calibrated, precision laboratory standard with an uncertainty of less than +0.02 dB over the range 0 to 20 dB used for these measurements. The thermistor mounts were calibrated to account for mismatch effects, loss due to absorption within the mount but not in the thermistor elements, and the dc-to-microwave substitution error. The estimated uncertainty associated with the thermistor mount is $\pm$0.06 dB, and the uncertainty of the power meter used with the thermistor mount is $\pm$0.13 dB. The uncertainty due to non-linearity of the receiver is $\pm$0.04 dB.

The repeatability error was determined by repeating the procedure of section 2.3.1 and observing any variations in the attenuator setting and receiver readings. The final receiver reading, following reconnection of the probe, was especially significant for indicating drift and flange

connection errors. The uncertainty due to non-repeatability was estimated to be ±0.20 dB.

The theoretical gain value of each probe at the frequency of interest was used. These values are:

X-Band (10.53 GHz) 16.7 dB±0.3 dB

K-Band (24.15 GHz) 6.7 dB±0.3 dB

These gains could have been determined to within ±0.1 dB by careful calibration, but it was not considered worth the added effort and expense in view of the other errors associated with the measurement. In addition, there is undoubtedly some variation in the output of different radar units of the same model, so there is not much to be gained by testing a particular radar to such accuracy. A near-zone gain correction for the X-Band probe was required at the closest separation distance and has been included in the calculations.

The errors associated with these measurements are summarized below:

Source of error	Uncertainty (in decibels)
Rotary-vane attenuator	±0.02 dB
Thermistor mount	±0.06 dB
Power meter	±0.13 dB
Receiver non-linearity	±0.04 dB
Connector repeatability and system drift	±0.30 dB
Uncorrected mismatch error	±0.10 dB
Probe antenna gain	±0.30 dB
Total error sum:	±0.95 dB

The listed errors are approximate worst-case values and, since the errors are uncorrelated, the sum represents a conservative estimate for the total error.

Although an estimated error approaching ±1 dB may seem rather high, it should be noted that radiated power density is a particularly difficult parameter to measure with high accuracy because of the many variables and precise calibrations involved. In this case, the two largest sources of error were associated with the gain of the probe antenna and with connector repeatability and system drift. As has already been mentioned, the gain uncertainty could be reduced to ±0.10 dB by careful calibration, and the repeatability could probably be improved to 0.10 to 0.15 dB through the use of precision connectors. These improvements would result in an overall error of approximately ±0.5 dB, which is about the best one could expect. However, considering the variability that exists among radar units of the same type and the variable effects of the immediate operational radar environment on the radiated field strength, the approximate ±0.3 dB improvement in accuracy did not justify the use of the more expensive and time-consuming techniques required to achieve it.

2.4 Measurement Procedures and Results

2.4.1 Alignment and Measurements

The mounting arrangement is shown in figure 4. A ring mount was constructed which clamped to the radar being measured. The ring mount was attached to a tilt plate which was, in turn, attached to a translation plate. The translation plate was bolted to the model mount. A handgun is shown mounted in figure 4, but the same basic arrangement was used for all the radars tested. Care was taken to assure that the radar aperture was always well in front of the metal mounting ring to minimize perturbation of the pattern by the mount.

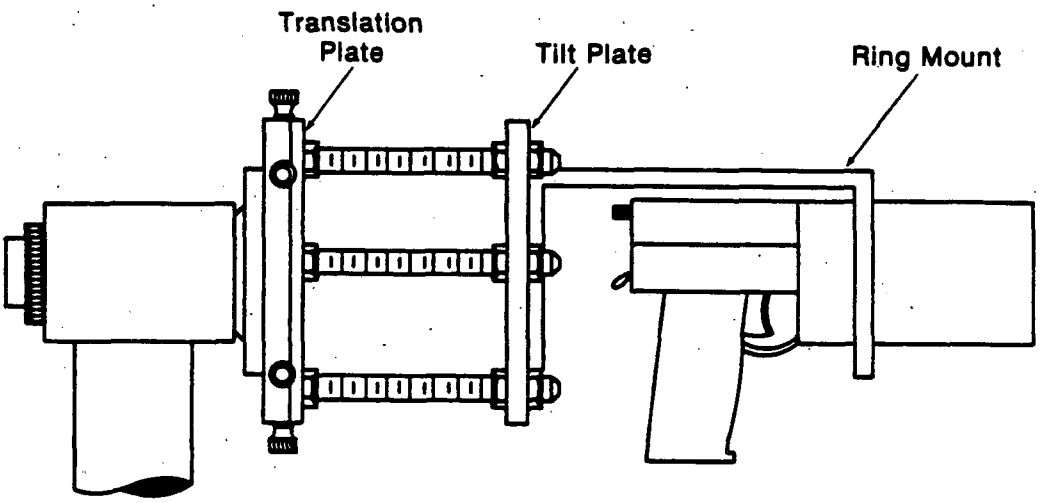


Figure 4. Mounting arrangement used to support radars so that they could be properly aligned for radiated power density measurements.

Although mechanical, optical, and electrical techniques for antenna alignment were employed, the final test involved only electrical measurements of the fields emitted by the test antenna. Proven methods were used for the precise alignment of each radar. The objective was to assure that the radar axis coincided with the ϕ rotation axis of the model mount and that for $\theta=0$, the axes of the probe, radar, and model mount were all coincident with the z-axis of the coordinate system of figure 1. Once this was accomplished, the z-position of the radar was adjusted slightly by moving the model mount with respect to the azimuth rotator until rotation in θ produced minimal phase variations. This adjustment was done to place the rotation axis at the phase center of the radar.

After the alignment was completed, a set of data was obtained by rotating the radar 360° about the θ axis, recording the field strength at 5° intervals in θ, and repeating the process for 10° increments in ϕ until the entire sphere was scanned. This procedure yielded data along the 10° meridians of a sphere.

The total power density is the sum of the power densities associated with two orthogonal field components. Therefore, at each measurement distance, two complete scans must be performed, one for each component. Since the probes were linearly polarized, they were oriented to respond to the vertical component for one scan and then rotated 90° to respond to the horizontal component for the second scan. The entire process was carried out at four distances for each radar, the distances being 1, 3, 6, and 12 ft (30, 91, 183, and 366 cm) for the X-Band radars and 1, 3, 6, and 10 ft (30, 91, 183, and 305 cm) for the K-Band radars. The largest K-Band distance was restricted to 10 ft (305 cm) because the probe assembly was longer than the X-Band probe and the overall distance available was limited by the length of the rails on which the rotator was mounted. At the shortest distance, 12 in (30 cm), the rotation about the θ axis was limited to the range -135° to +135° in order to avoid hitting the probe antenna with the rear of the model tower.

2.4.2 Results

The power density values were obtained as follows. Results of the calibration procedures described in section 2.3.1 were analyzed and used to calculate the received power at the on-axis reference point, which we shall call P_R'. The values of P_R obtained during the scanning process were measured relative to this on-axis value. Once P_R' had been calculated, the received power in mW at each measurement point was determined and the corresponding incident power density was then calculated by eq (1), for each point. The power densities for each component were summed to give the total power density at each point. These calculations were performed using the NBS computing facilities, and one of a variety of plotting routines was selected to display the results in graphical form.

In order to determine the symmetry of typical radiation patterns, contour plots like that in figure 10 (see sec. 5) were generated for selected radars. Note that the beam possesses circular symmetry down to about 23.4 dB below the peak (42 dB below 1 mW/cm^2). This symmetry is typical of all the radars tested. Because of the uniformity between radar units, it was decided not to provide contour plots for each distance for 20 radars.

A graphical display which presents the power density information in a more useful form is the polar coordinate format used in figures 11 through 50. These graphs display the power density in mW/cm^2 or dB with respect to 1 mW/cm^2 as functions of the angle off axis and the distance from the radar. Each figure has four curves, one for each measurement distance, and figures were prepared for both the vertical (elevation) and horizontal (azimuth) pattern cuts in order to display the effects of any significant asymmetries which might exist. The vertical and horizontal planes are defined with respect to the radars when mounted in their normal upright configurations and pointed horizontally as in figure 1. The horizontal and vertical cuts correspond to $\phi=0$ and 90°, respectively. In each figure the origin corresponds to a power density of 10^{-5} mW/cm^2. The power density in dB corresponding to a particular distance and elevation or azimuth angle (equal to θ for these principal plane cuts) is proportional to the length of the radius vector from the origin to the point selected on the particular distance curve. Therefore, the inner curve represents the smallest power density and is for the largest distance from the radar. The strongest fields are associated with the closest distance 12 in (30 cm) and are plotted on the outer curve. This format provides a good visual representation of the field patterns as viewed by an operator positioned behind the radar and looking in the direction the radar is aimed.

The nomenclature used to identify the radars (X-1, K-1, etc.) is identical to that used in task 2 so that results between tasks may be compared if desired. Fourteen X-Band and six K-Band radar units were tested during task 1 and 15 X-Band and seven K-Band radar units were tested during task 2.

3. TASK 2 MEASUREMENTS

3.1 General Approach

Since speed radar units are usually mounted on or inside an automobile, a complete investigation of microwave radiation levels requires that measurements be made inside the automobile under normal operating conditions. By this means it is possible to observe any field enhancement effects produced by the automobile enclosure. Each of the 22 radar units tested was mounted in the positions in which it is normally used, and calibrated field probes were used to measure the power density (mW/cm^2) throughout the interior of the car, paying particular attention to regions where the head and groin would be located.

3.2 Probes Used for the Vehicular Measurements

Two different probes were used for these measurements. An NBS Model EDM-1C Electric Energy Density Meter was used for the measurements involving the X-Band (10.525 GHz) radars. This NBS probe does not operate above X-Band, so a commerical Electromagnetic Radiation Monitor was used for the K-Band (24.15 GHz) measurements. The sensor antennas in both probes consisted of three orthogonal dipoles in order to achieve isotropic response patterns. The measurements are, therefore, quite insensitive to the orientation of the probe with respect to the field being measured, as long as the probe handle is not pointed toward the radiation source. The NBS probe uses diodes for the detecting elements, while the commerical unit employs thermocouples. Consequently, the NBS probe has greater sensitivity; its threshold response being 0.001 mW/cm^2. With the commerical meter, the minimum detectable power density was 0.01 mW/cm^2. Both probes make use of high-resistance leads between the sensor and metering unit to eliminate pickup by the leads which would generate an error signal.

The meters were calibrated by the free-space standard-field method which is illustrated in figure 5. The power density, W, at a point on the transmitting axis at a distance d from the standard gain horn is given by

$$W = \frac{P_T G}{4\pi d^2} ,$$

where P_T is the net power delivered to the horn and G is the effective gain of the horn. The gain was determined in advance [2] and P_T and d were measured as part of the calibration procedure. The incident power Pi and reflected power P_r were monitored with the coupler sidearms, and $P_T = P_i - P_r$. When W had been determined at a particular point, the probe being calibrated was placed at this same point in the known field and a probe correction factor was obtained from the ratio of the actual power density to the indicated power density.

The error in the basic calibration was estimated to be ± 0.5 dB (12%). However, in actual use, additional uncertainties arose due to such things as multipath effects, perturbation of the field by the operator, and the fact that the probe was calibrated in a plane-wave field but was used in a more complex field configuration. Taking all of these factors into consideration resulted in an estimated overall measurement uncertainty of ± 1.0 dB (25%). Although it may seem rather high, a ± 1.0 dB uncertainty is very good for electromagnetic field measurements performed in such a complicated electromagnetic environment.

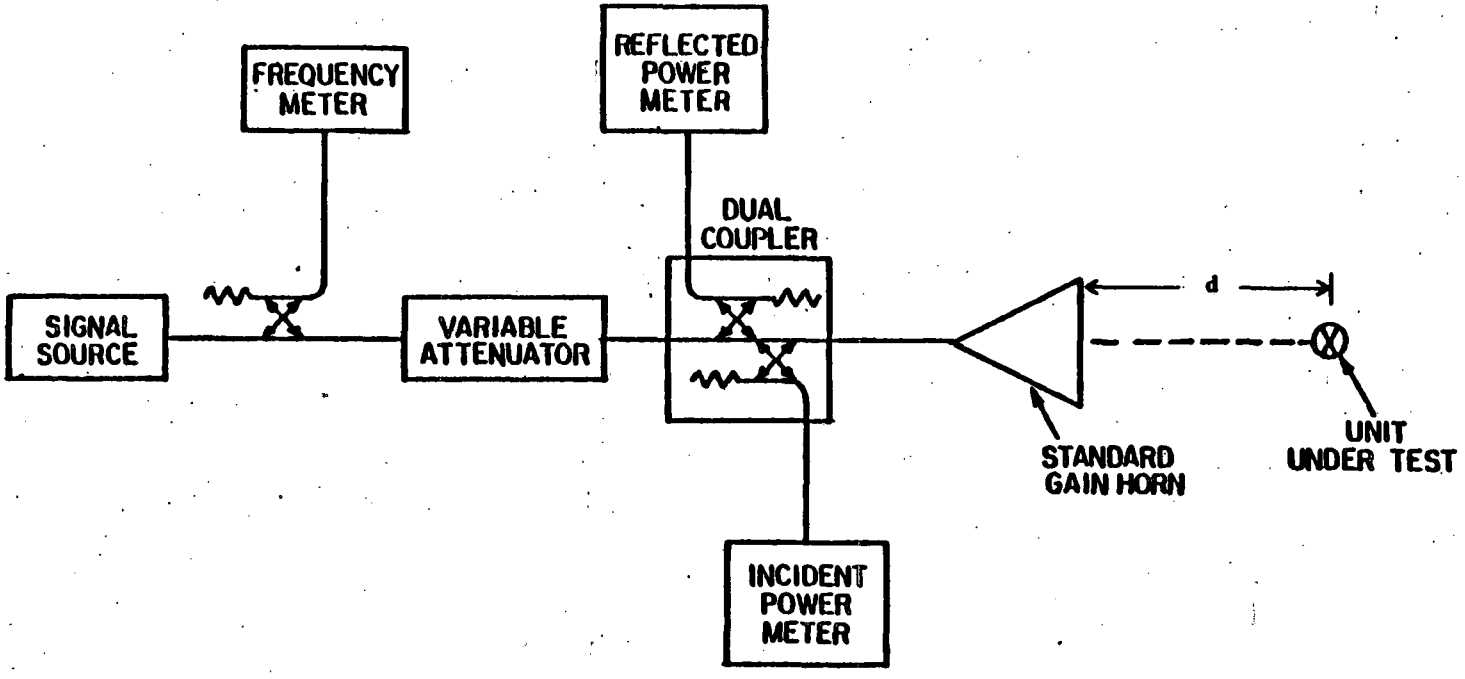


Figure 5. Diagram of the basic experimental arrangement required for the free-space standard-field method of calibrating microwave hazard meters.

3.3 Measurement Procedures and Results

Prior to making measurements inside the automobile, the power density in the main beam, close to the aperture, was determined by holding the probe directly in front of the aperture as shown in figure 6. These results are listed in column 2 of table 1. The maximum power density radiated to the sides and rear of each unit was also determined by moving the probe over the surface of the unit as shown in figure 7 and observing the maximum indication. These values are listed in column 3 of table 1.

Following the above tests, the radars were mounted in, on, or hand-held in a four-door sedan in the various operating positions indicated by the numbers in figure 8. In positions 1 and 2, the radars were hand-held and aimed through the windshield. Position 5 is the common dashboard mount with the radar aimed forward (fig. 9a), and 5R is the same arrangement with the radar aimed to the rear through the back window (fig. 9b). In positions 6 and 7 the radars were hand-held and aimed out the left and right front side windows, respectively. For position 3, the radar was attached to the inside of the right rear window and aimed forward through the windshield while, in position 4, the radar was attached outside the left rear window and aimed forward. In position 8, the radar was aimed to the rear through the back window.

Each radar unit was mounted in each of the positions in which it was designed to operate, as indicated in column 4 of table 1. With the radar in each position, the appropriate hazard probe was used to survey the field intensity throughout the interior of the automobile. The maximum power densities observed in the general regions occupied by the driver and three passengers (locations A through D of fig. 8) are given in table 1, columns 5-8. These recorded values represent the maximum levels observed, that is, there were no values of higher radiation intensity in locations other than those whose power density values are listed in table 1.

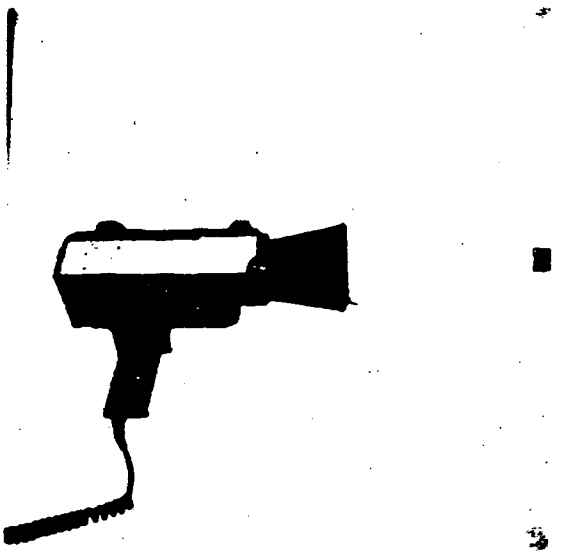


Figure 6. Measurement of main-beam power density in the aperture region.

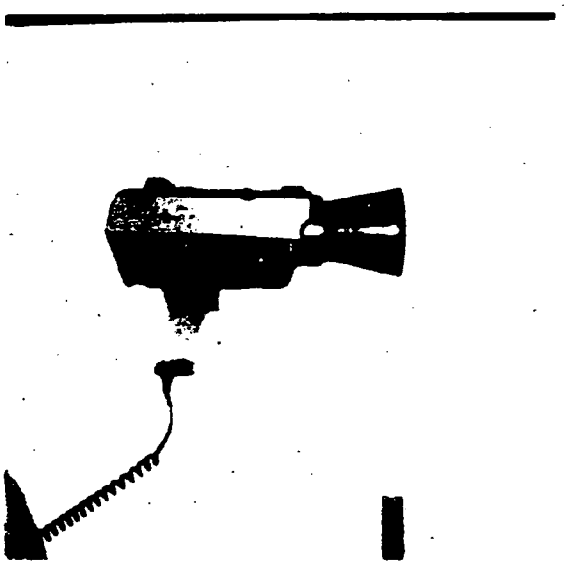


Figure 7. Measurement of radiation in the side- and back-lobe regions.

Table 1. Results of vehicular power density measurements.

This table contains results of measurements of field intensities produced by the various radar units inside an automobile. The radar code numbers are given in the first column, with the K-Band units (24.15 GHz) designated by K-1 to K-7 and the X-Band units (10.525 GHz) by X-1 to X-15. Column 2 gives the power density in the aperture, and column 3 the maximum power density in the back hemisphere. The mounting positions of column 4 correspond to the numbered positions of figure 8 as described in the text. The interior vehicular data are given in columns 5 to 8. Blank entries in these columns mean that the field intensities were too low to read with the meters used for these tests; i.e., the fields were <0.01 mW/cm^2 for K-Band and <0.001 mW/cm^2 for X-Band.

Radar code number	Aperture power density (mW/cm^2)	Maximum back-lobe power density (mW/cm^2)	Radar mounting position	Maximum power density at positions A, B, C, and D of figure 8 (mW/cm^2) A	B	C	D
K-1	1.97	<0.01	5				
			8				
K-2	2.40	0.02	1				
			2				
			5				
			5R[a]	0.01	0.01		
			8				
K-3	2.27	<0.01	1				
			2				
			5				
K-4	1.83	<0.01	1				
			2				
			5				
K-5	0.25	<0.01	1				
			2				
			5				
			5R[a]				
K-6	2.78	0.02	1				
			2				
			5				
K-7	1.64	<0.01	1				
			2				
			5				
X-1	0.55	<0.001	1	0.001			
			2				
			3	0.137	0.001	0.002	0.002
			4		0.001		
X-2	0.73	0.001	1	0.001	0.001		
			2				
			3	0.36	0.003	0.001	0.001
			4		0.002		
X-3	2.82	0.018	1	0.002	0.003		0.001
			2	0.001	0.021	0.001	0.001
			5	0.002	0.001	0.001	
X-4	2.55	0.018	1	0.002	0.001		0.001
			2	0.001	0.003	0.001	0.001
			5	0.001	0.001		0.001
X-5	0.36	0.001	1				
			2				
			5				

Table 1. Results of vehicular power density measurements (Continued)

Radar code number	Aperture power density (mW/cm^2)	Maximum back-lobe power density (mW/cm^2)	Radar mounting position	Maximum power density at positions A, B, C, and D of figure 8 (mW/cm^2) A	B	C	D
X-6	1.19	0.002	1				
			2				
			5				
			6		0.001		
			7				
X-7	1.10	0.004	1	0.001			
			7	0.002	0.001		
			2				
			6	0.001	0.002		
			5		0.001	0.001	
X-8	0.89	0.002	1				
			7				
			2				
			6				
			5				
X-9	0.96	0.004	1	0.001			
			7				
			2				
			6				
			5				
			5R [a]				
X-10	1.76	0.008	1	0.001			
			7				
			2				
			6				
			5	0.001			
X-11	0.46	0.001	1				
			7	0.001			
			2		0.001		
			6				
			5				
X-12	0.59	0.005	5				
X-13	0.93	0.003	1				
			2				
			5				
			6				
			7				
X-14	0.48	0.001	1				
			2				
			5				
			6				
			7				
X-15	2.29	0.018	1				
			2				
			5				
			6		0.002		
			7	0.002			

[a] Same location as radar mounting position 5 except that the radar is pointed toward the rear of the vehicle.

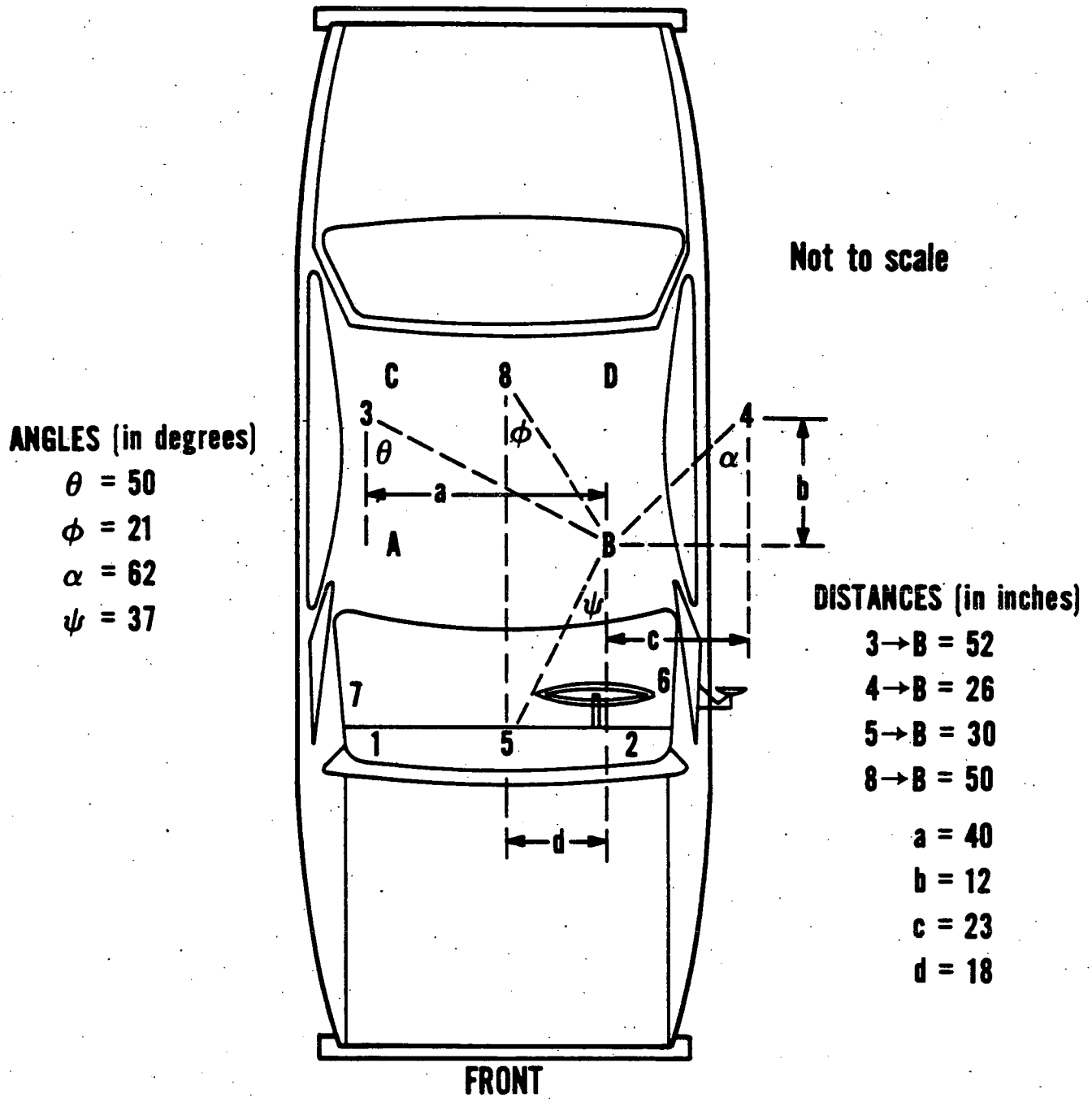
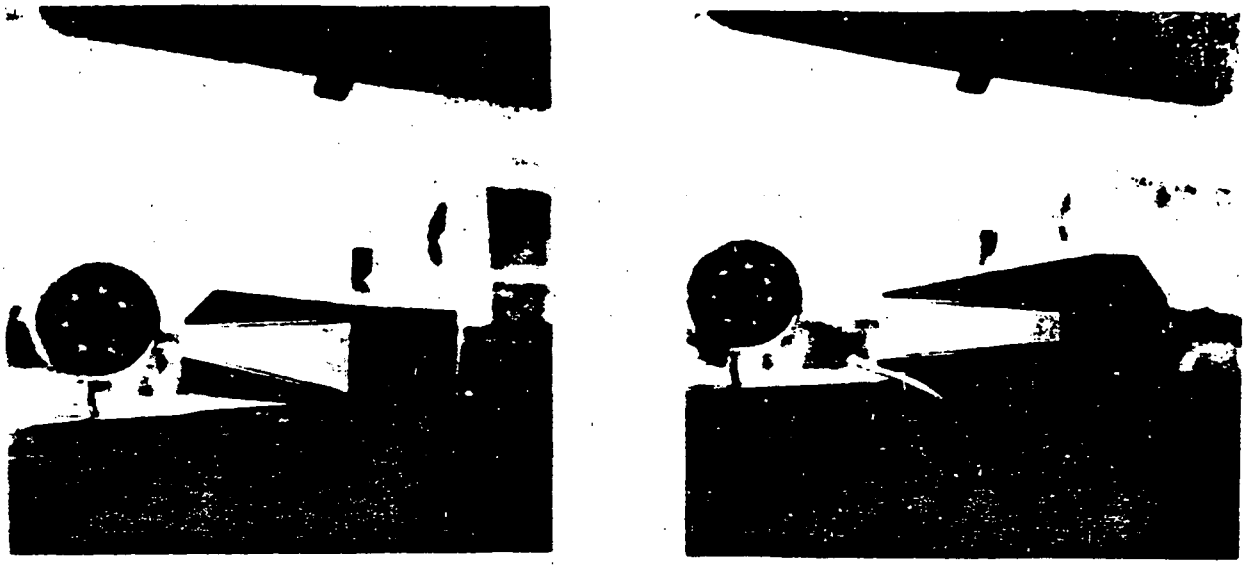


Figure 8. Diagram showing the location of radar mounting positions (1-8), seat locations (A-D), and distances between selected radar positions and seat locations used in describing field intensity distributions inside the automobile.



a) b)

Figure 9. Typical dashboard mounting arrangement: a) shows the radar pointing forward, and b) shows the radar aimed to the rear through the back window.

4. DISCUSSION OF MEASUREMENT RESULTS FOR TASKS 1 AND 2

It is not the purpose of this report to state what levels of electromagnetic fields constitute a health hazard. Such issues are left to those organizations and committees that have been established to perform and interpret research on the biological effects of electromagnetic waves and to set exposure limits based on the results of such research.

From the graphs of figures 11 through 50 one can obtain the expected power density in any desired region of space. For example, from the horizontal cut for X-1 (fig. 11), it is evident that, for distances greater than 12 in (30 cm) and for all angles, the power density is less than 0.2 mW/cm^2 (-7 dB). Another way of using the curves is to determine regions where a specified power density such as 0.1 mW/cm^2 is not exceeded. From figure 11, it is clear that the power density is less than 0.1 mW/cm^2 (-10 dB) at all angles for distances greater than about 36 in (91 cm) and also for distances greater than 12 in (30 cm) if the angle is greater than approximately $\pm 10°$ off axis. Similar determinations can be done for the other radars using the appropriate graphs.

Section 1910.97 of the Occupational Safety and Health Act (OSHA) contains a Radiation Protection Guide (RPG) which applies to exposure to electromagnetic radiation at various frequencies. At frequencies of 10 MHz to 100 GHz, the RPG allows exposure up to a power density of 10 mW/cm^2 over any 0.1-h period, or up to a power density of 10 mW/cm^2 averaged over any 0.1-h period or more. Concurrently, a voluntary Radio Frequency Protection Guide of 5 mW/cm^2 for the 1500 MHz to 100 GHz frequency range is under consideration for adoption by the American National Standards Institute. Whether the power density exposure limit remains at 10 mW/cm^2 or is lowered to 5 mW/cm^2, the power densities measured at a 12 in (30 cm) distance from the radars did not exceed either limit, even if operated continuously. The

K-Band radars tended toward higher powers and, since the antennas had more gain than the X-Band antennas, it was not surprising to observe that the power densities were generally higher for most K-Band units. In fact, all except K-5 had on-axis power densities of approximately 1 mW/cm^2 at the 12 in (30 cm) distance. For comparison, the U.S.S.R. defines safe exposure limits at 10 $\mu W/cm^2$ for a whole working day or exposure for not more than 15 or 20 min a day at 1 mW/cm^2 while wearing goggles [3].

The aperture power density for most of the units, measured in task 2 (col. 2 of table 1) is a significant fraction (25 to 50%) of the existing or proposed maximum permissible exposure levels. Based on the back-lobe data (col. 3 of table 1), one can conclude that all units are well-designed and packaged to provide shielding from leakage and back radiation. There was not a single case of reverse-hemisphere radiation of sufficient intensity to cause concern. In most cases, the power densities were $\frac{1}{1000}$, or less, of the exposure limit presently specified in the OSHA guide.

The data in columns 5 to 8 of table 1 indicate that the field levels inside the car do not exceed 0.002 mW/cm^2 for most radar positions. The major exception occurs in the data for X-1 and X-2 which show relatively strong fields at location A when the radar is mounted in position 3. This is to be expected since location A, the front seat passenger position, is directly in the main beam for this situation. Note, however, that the field strength recorded at the driver location is still low.

5. POWER DENSITY GRAPHIC DISPLAYS

The radiation pattern contour plot and power density polar coordinate plots discussed earlier (figs. 11-50) are grouped in this section for the convenience of the reader.

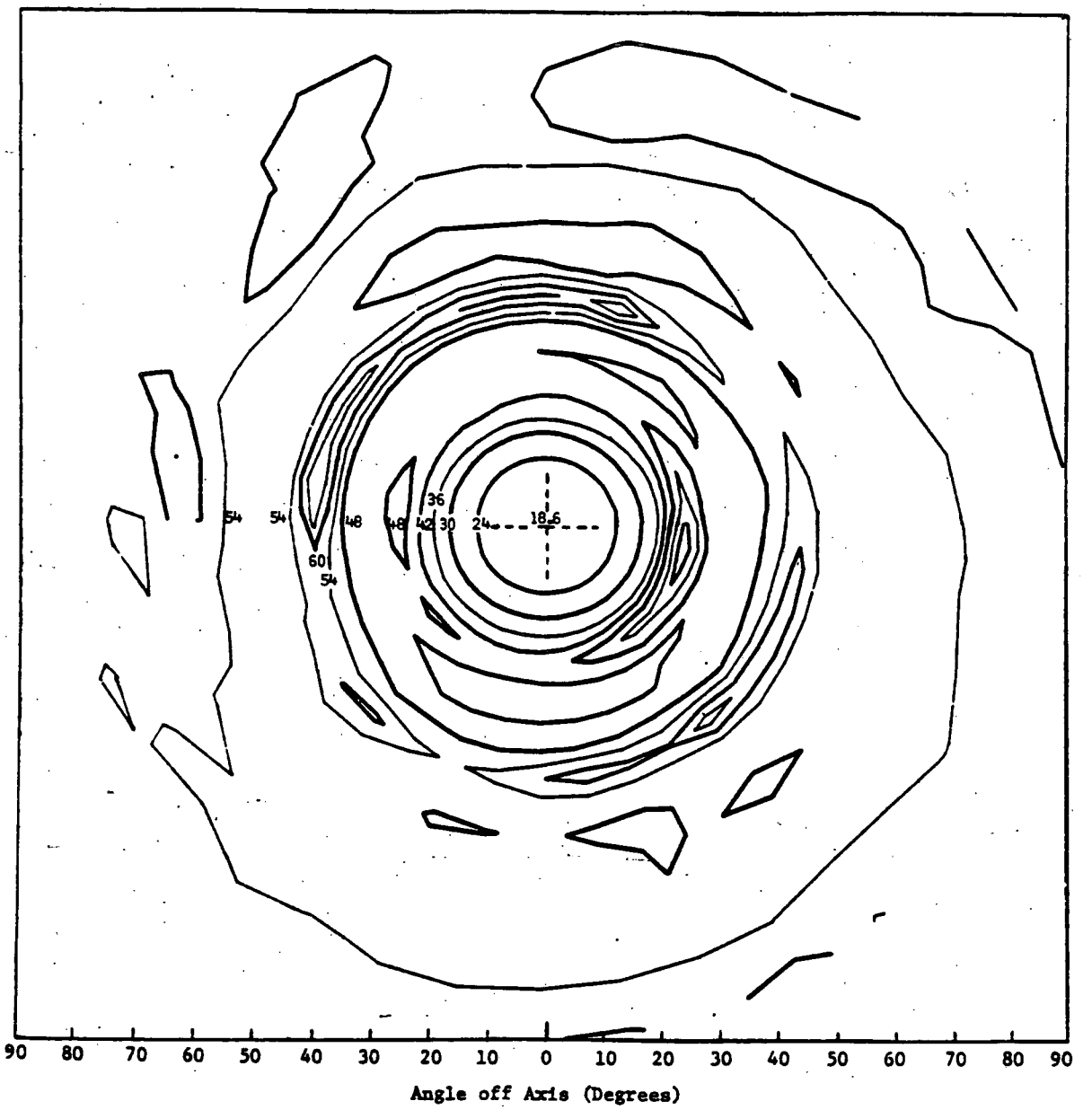
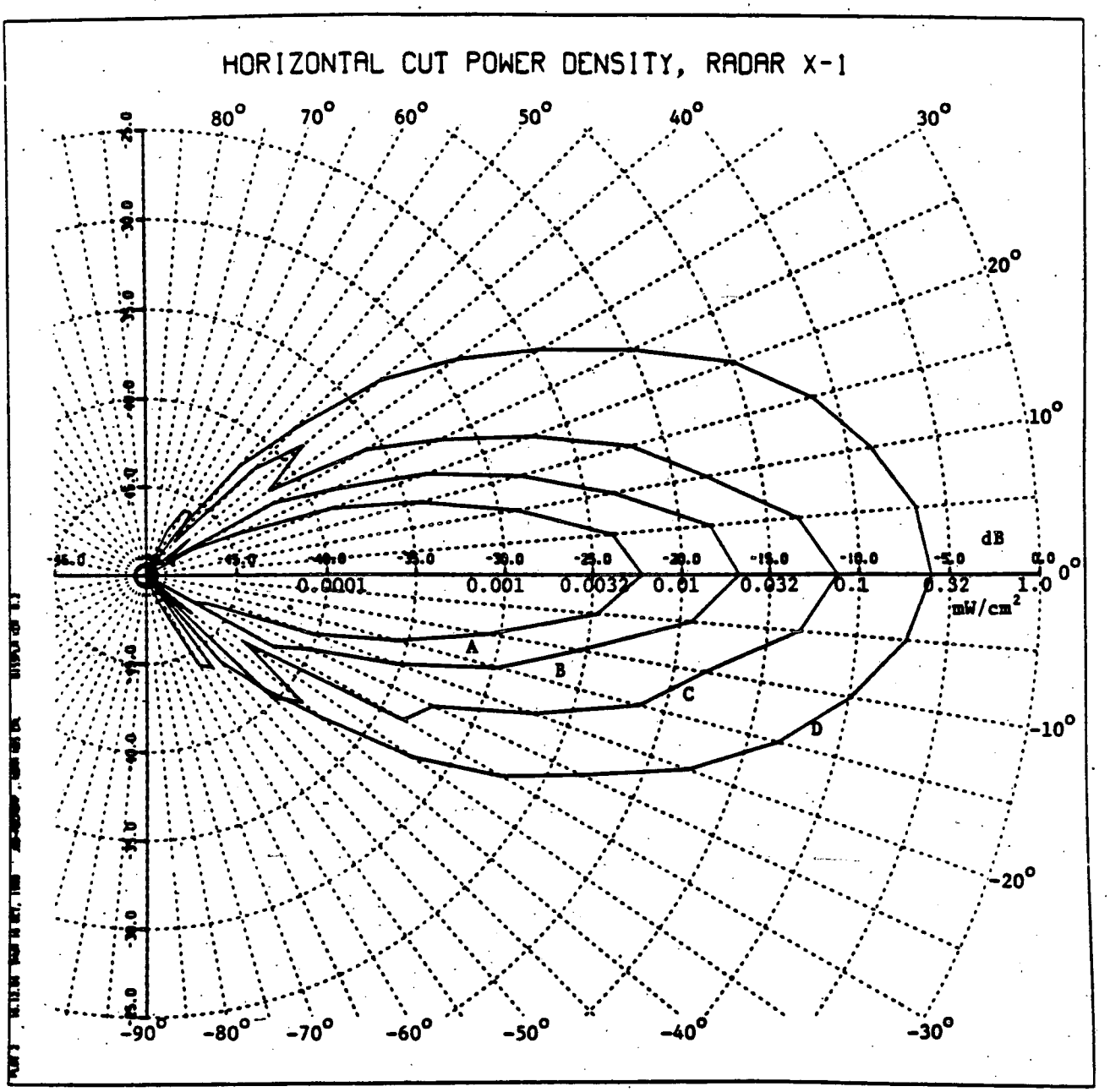
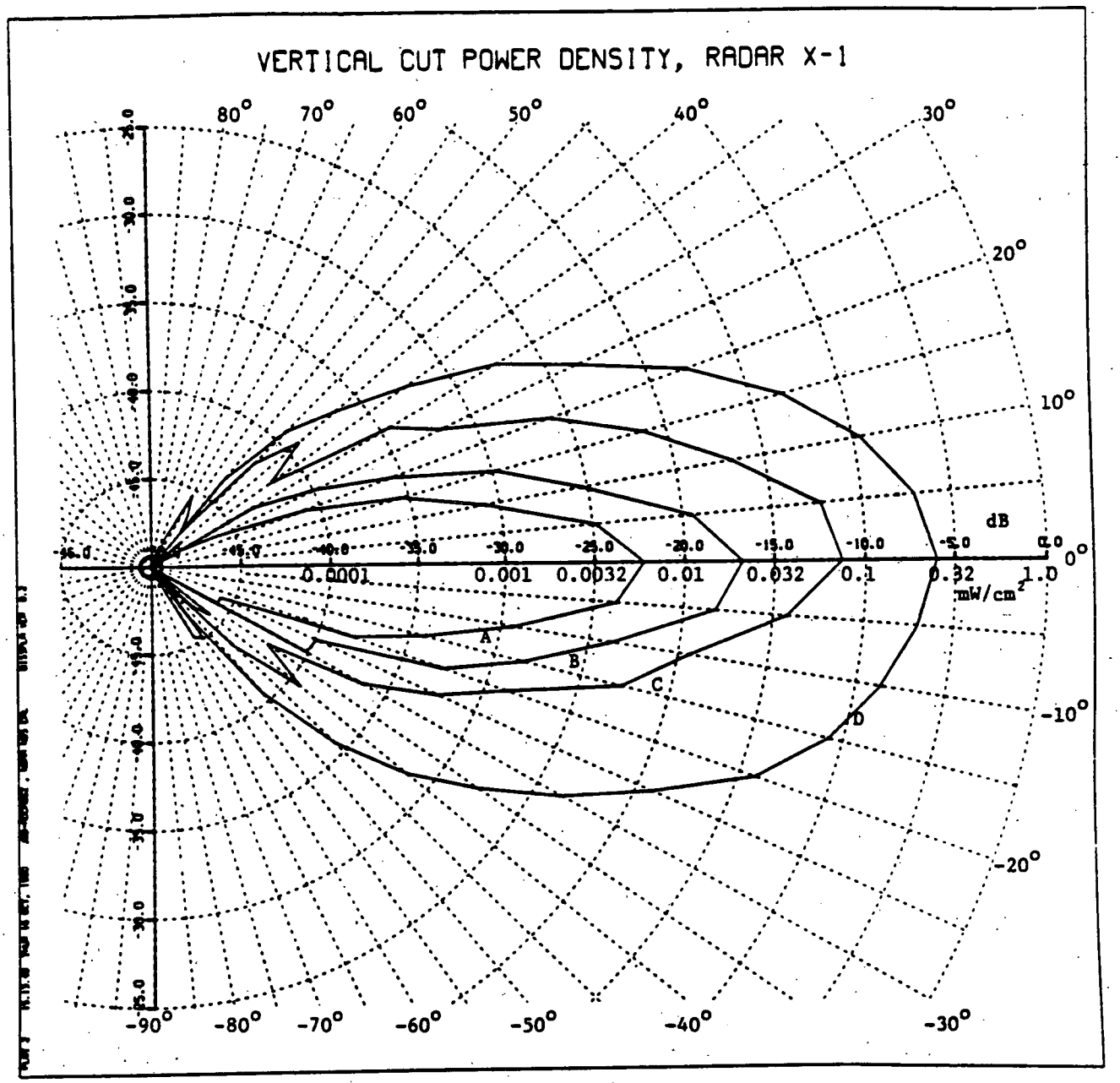


Figure 10. Power density contours for Radar X-4 at a distance of 2.1 m (82.5 in) from the antenna aperture. The view directly toward the radar. Numerical values of the contour lines indicate the number of decibels below 1 mW/cm . The peak, on-axis value is -18.6 dB (0.014 mW/cm^2), and the contour interval is 6 dB. The angular distance off axis is proportional to the radial distance from the center of the figure.



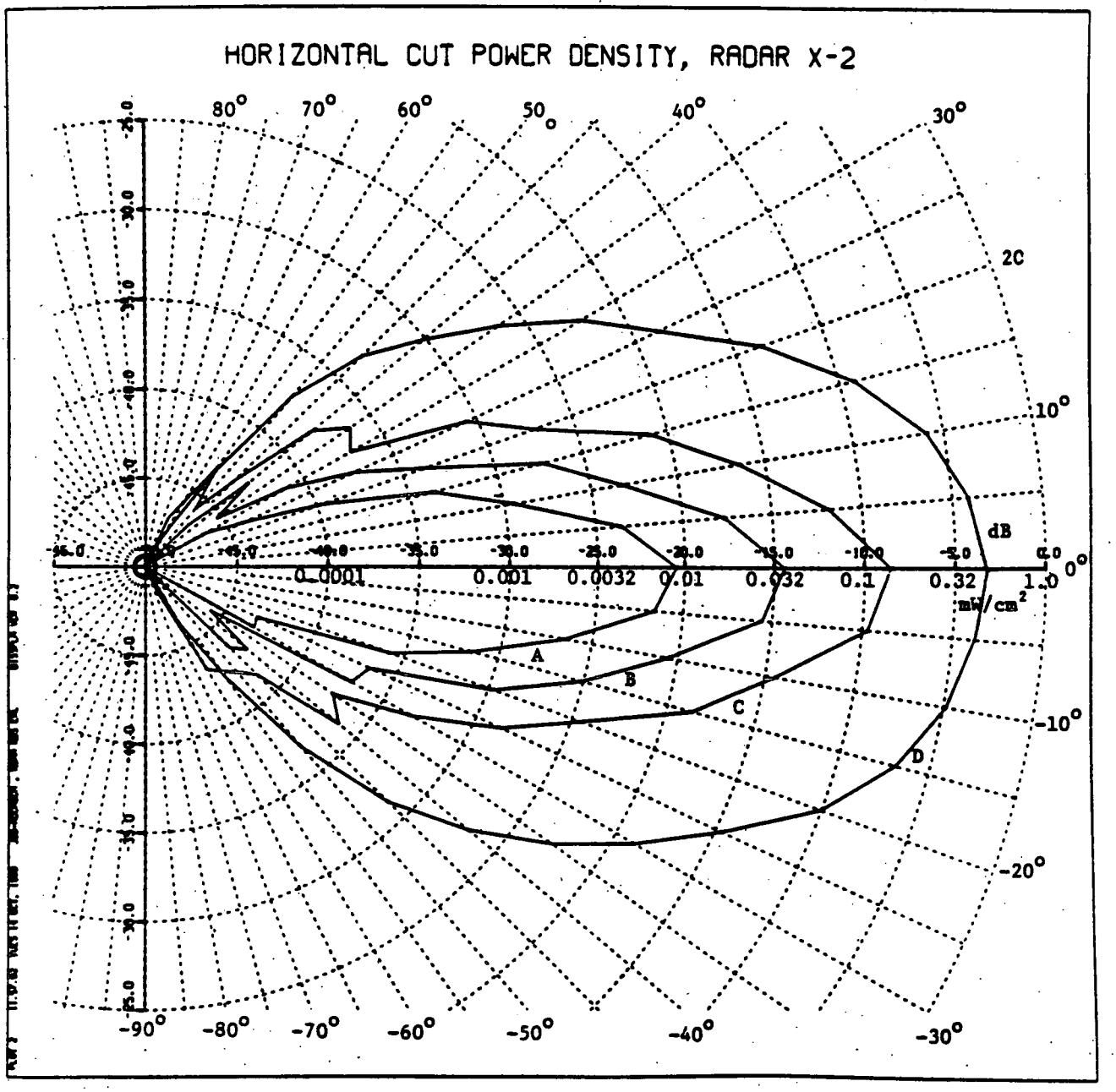
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 11. Horizontal power patterns for Radar X-1 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



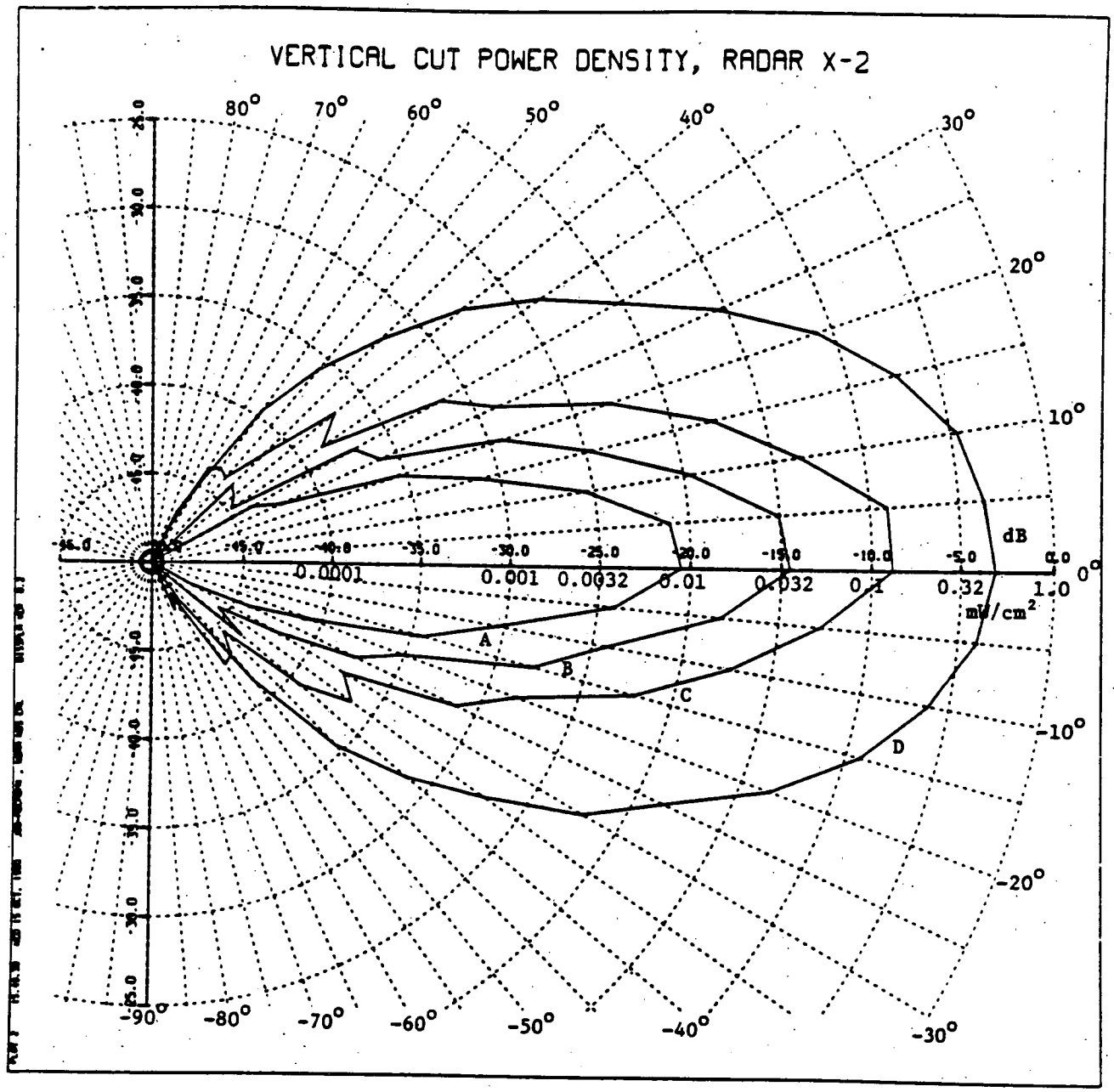
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 12. Vertical power patterns for Radar X-1 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



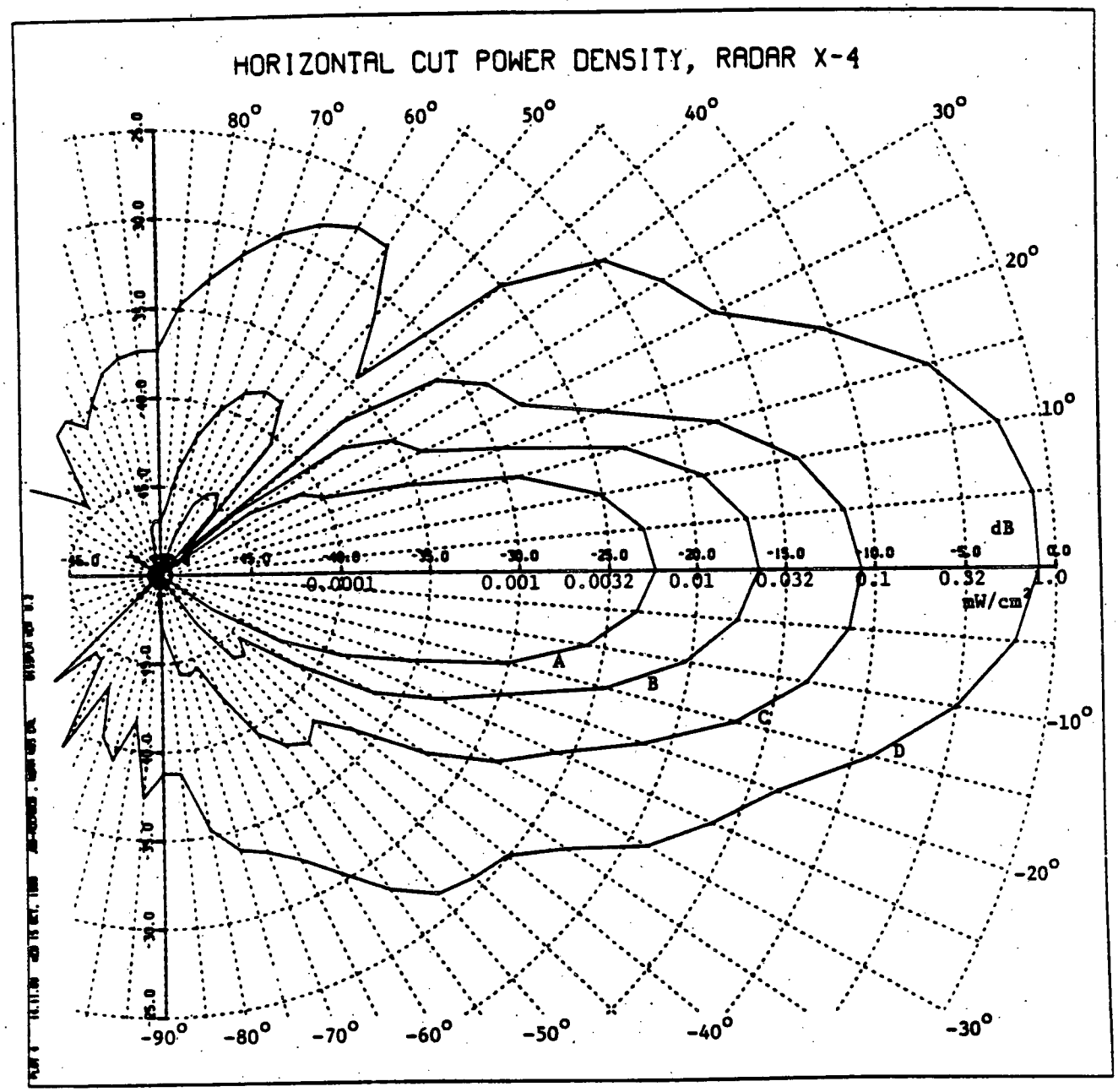
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 13. Horizontal power patterns for Radar-X-2 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



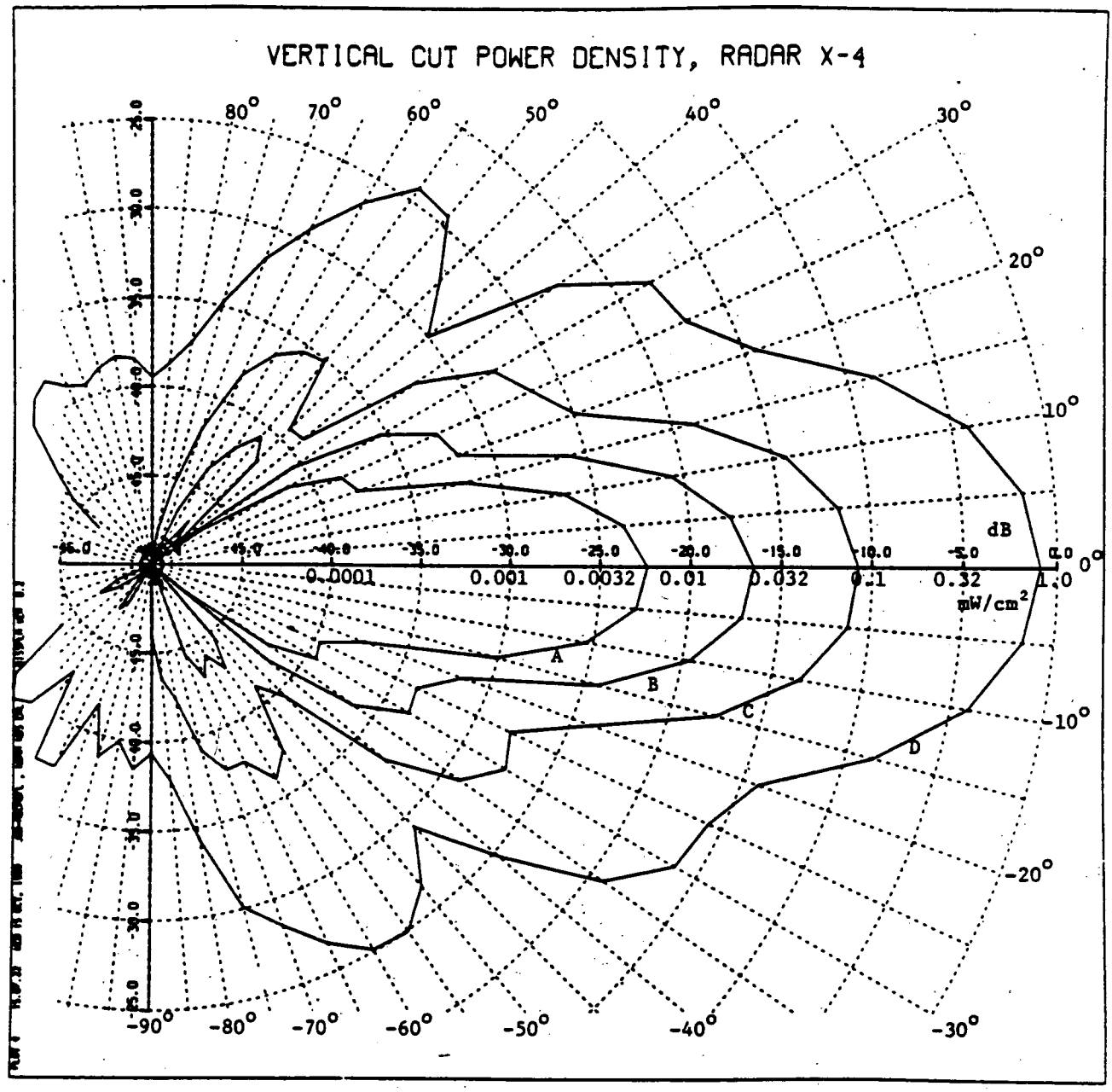
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 14. Vertical power patterns for Radar X-2 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



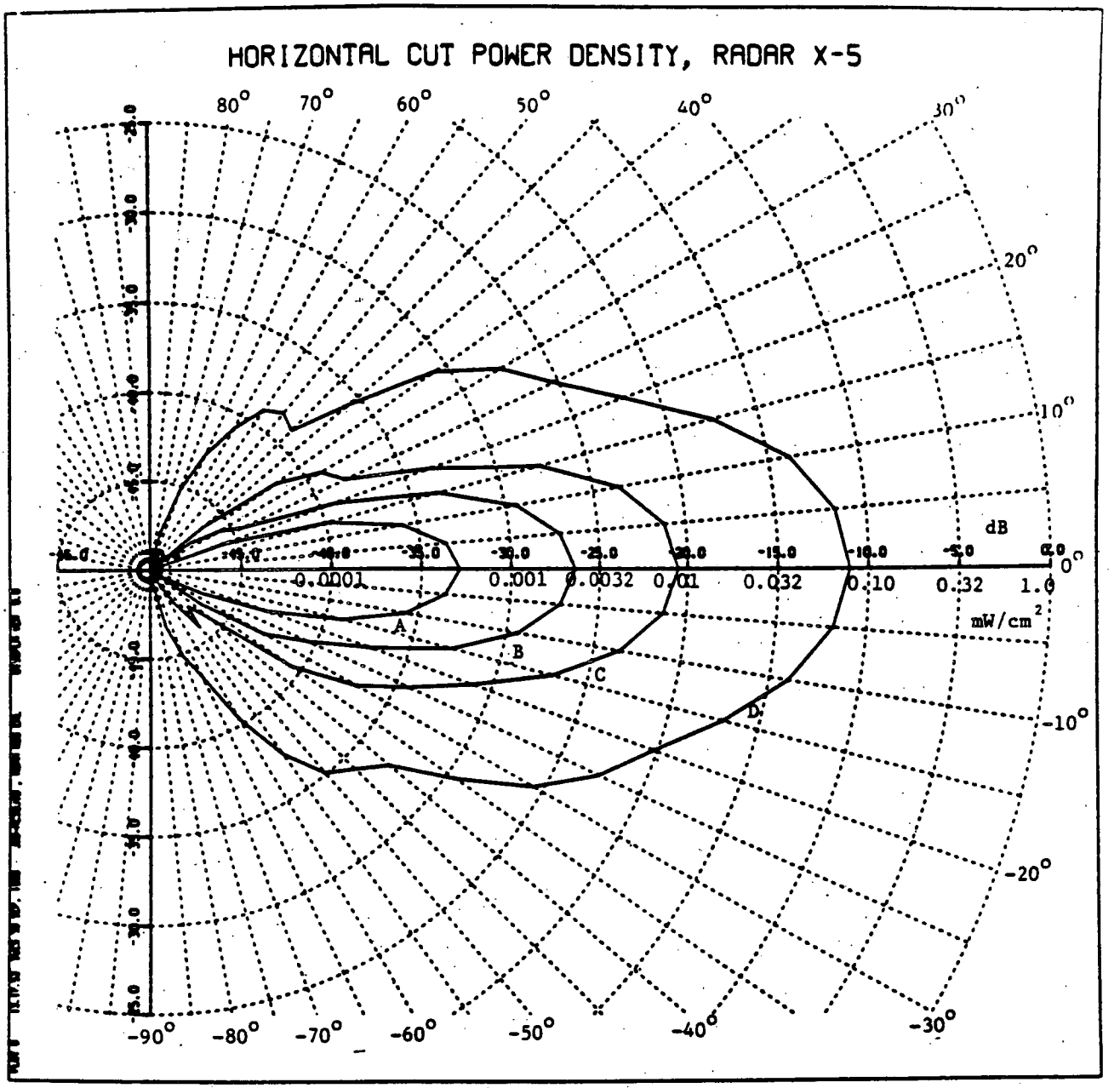
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 15. Horizontal power patterns for Radar X-4 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



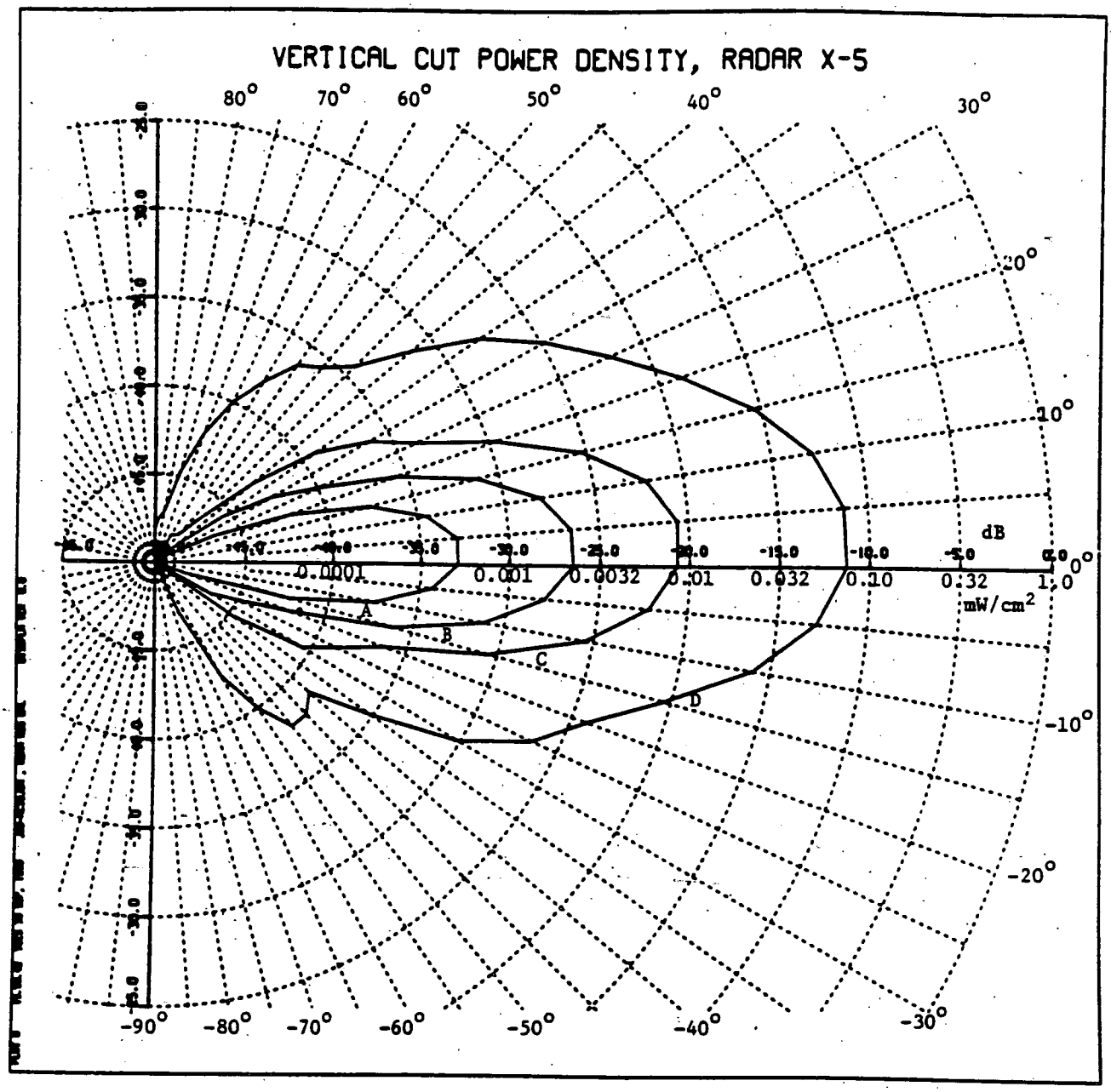
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 16. Vertical power patterns for Radar X-4 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



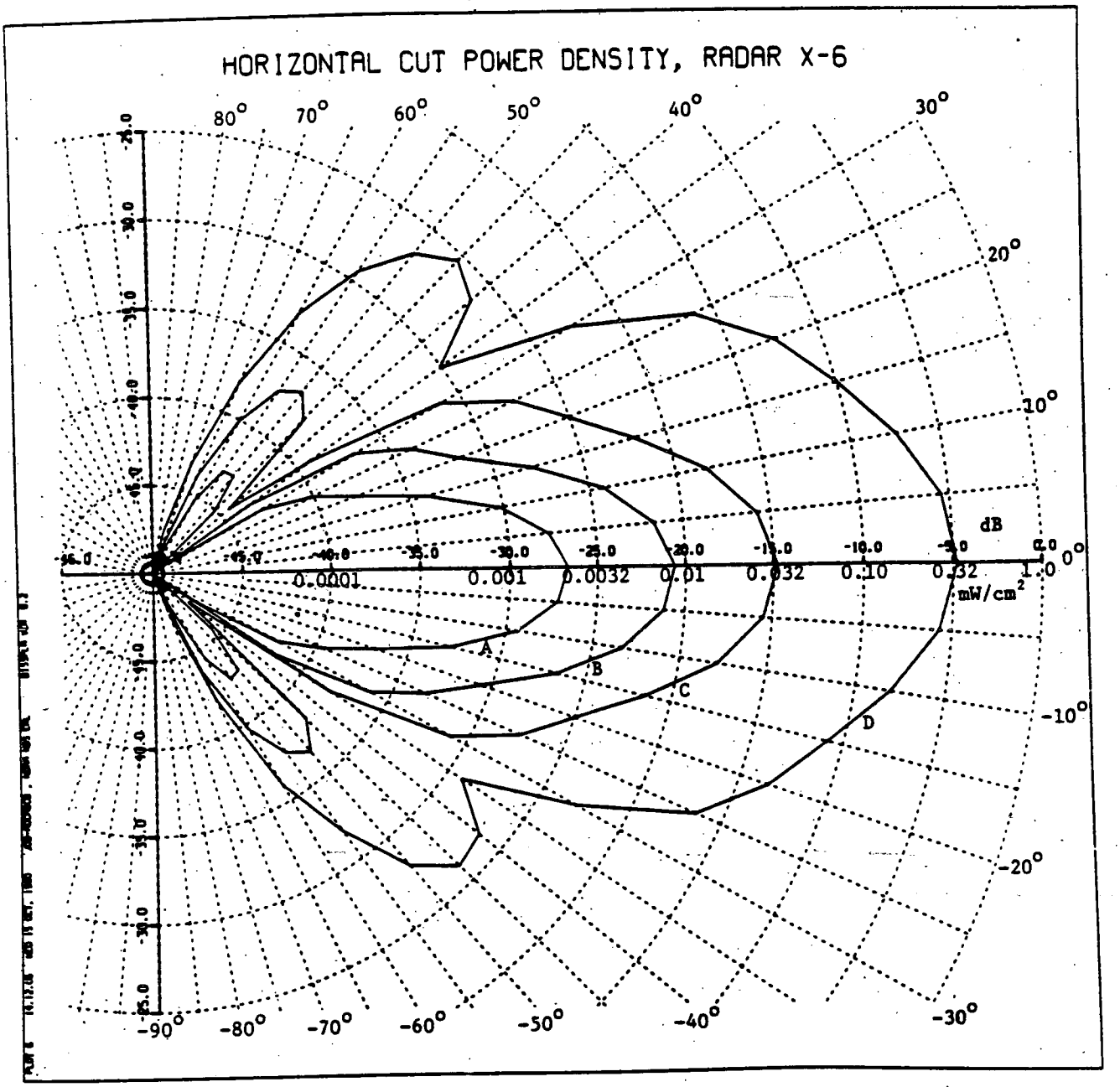
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 17. Horizontal power patterns for Radar X-5 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees with the direction of the main beam.



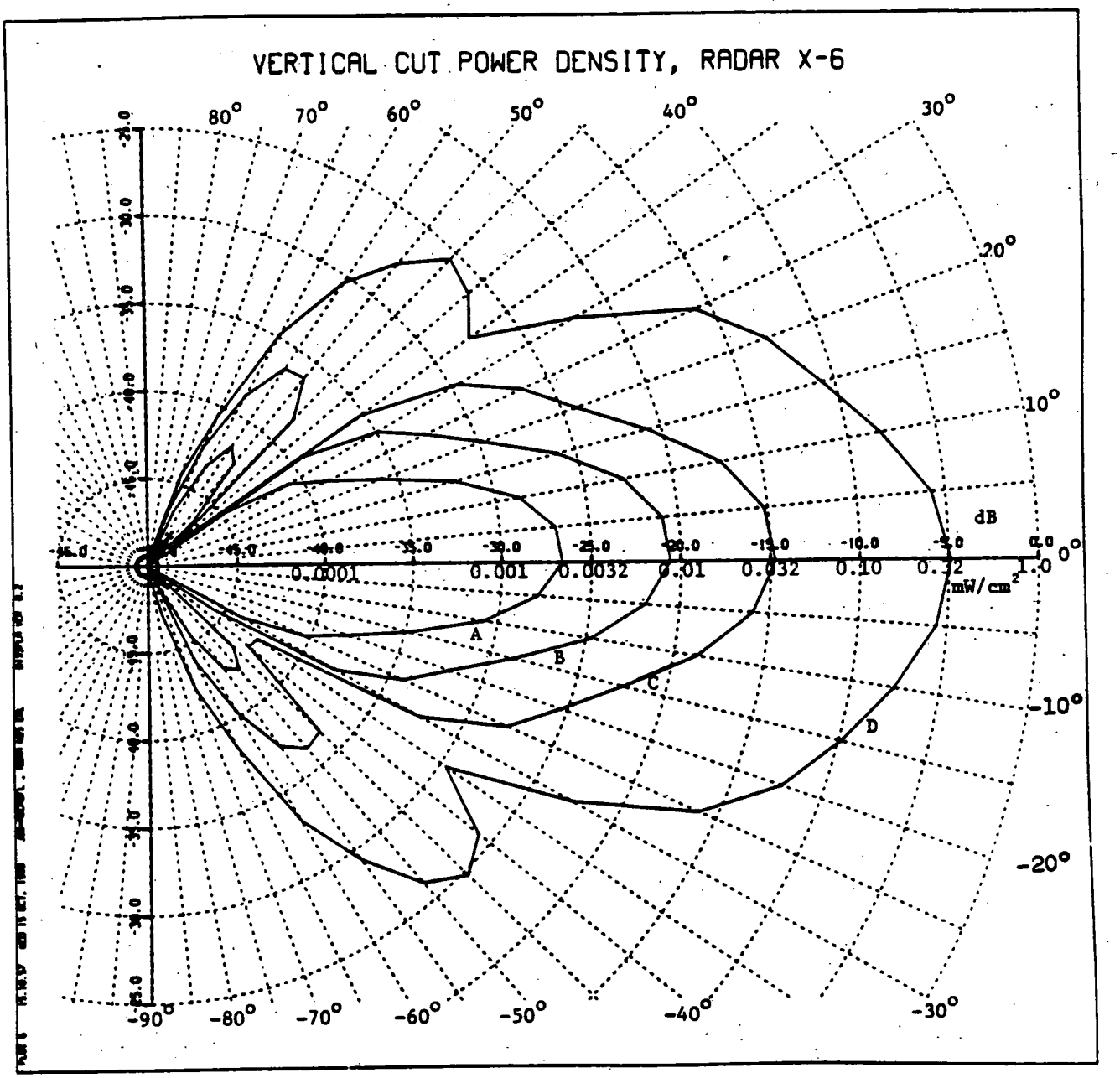
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 18. Vertical power patterns for Radar X-5 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



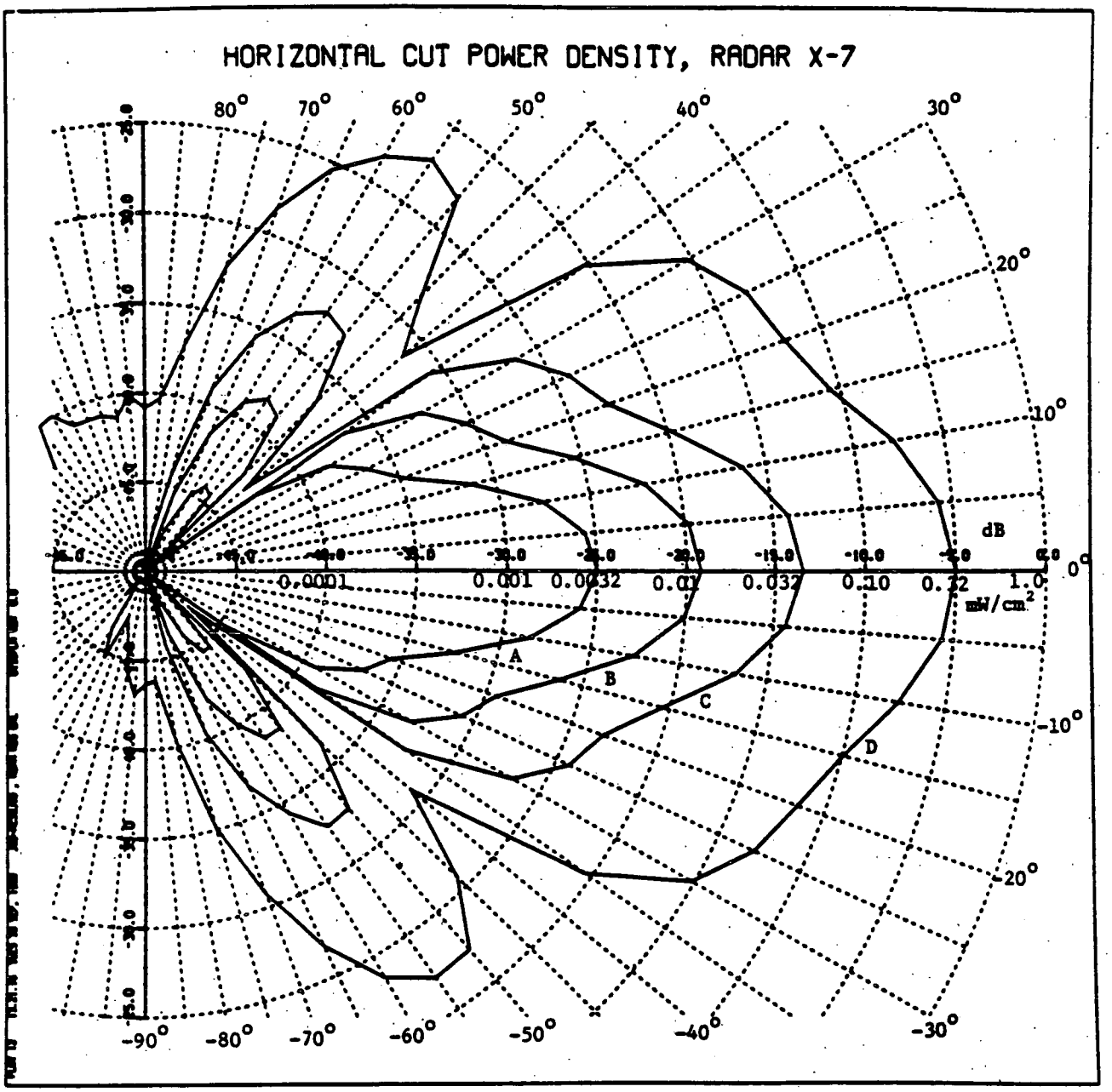
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 19. Horizontal power patterns for Radar X-6 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



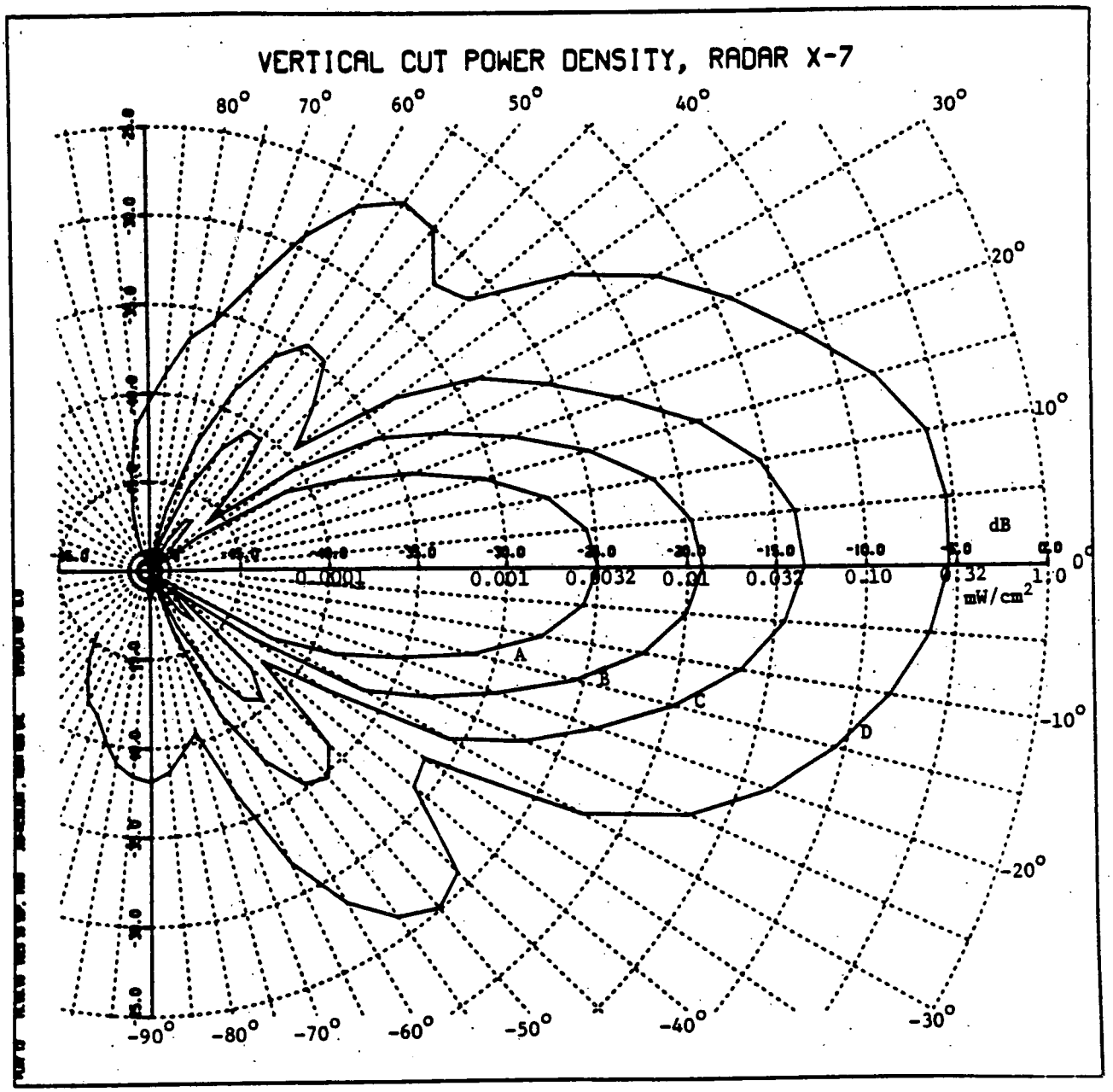
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 20. Vertical power patterns for Radar X-6 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



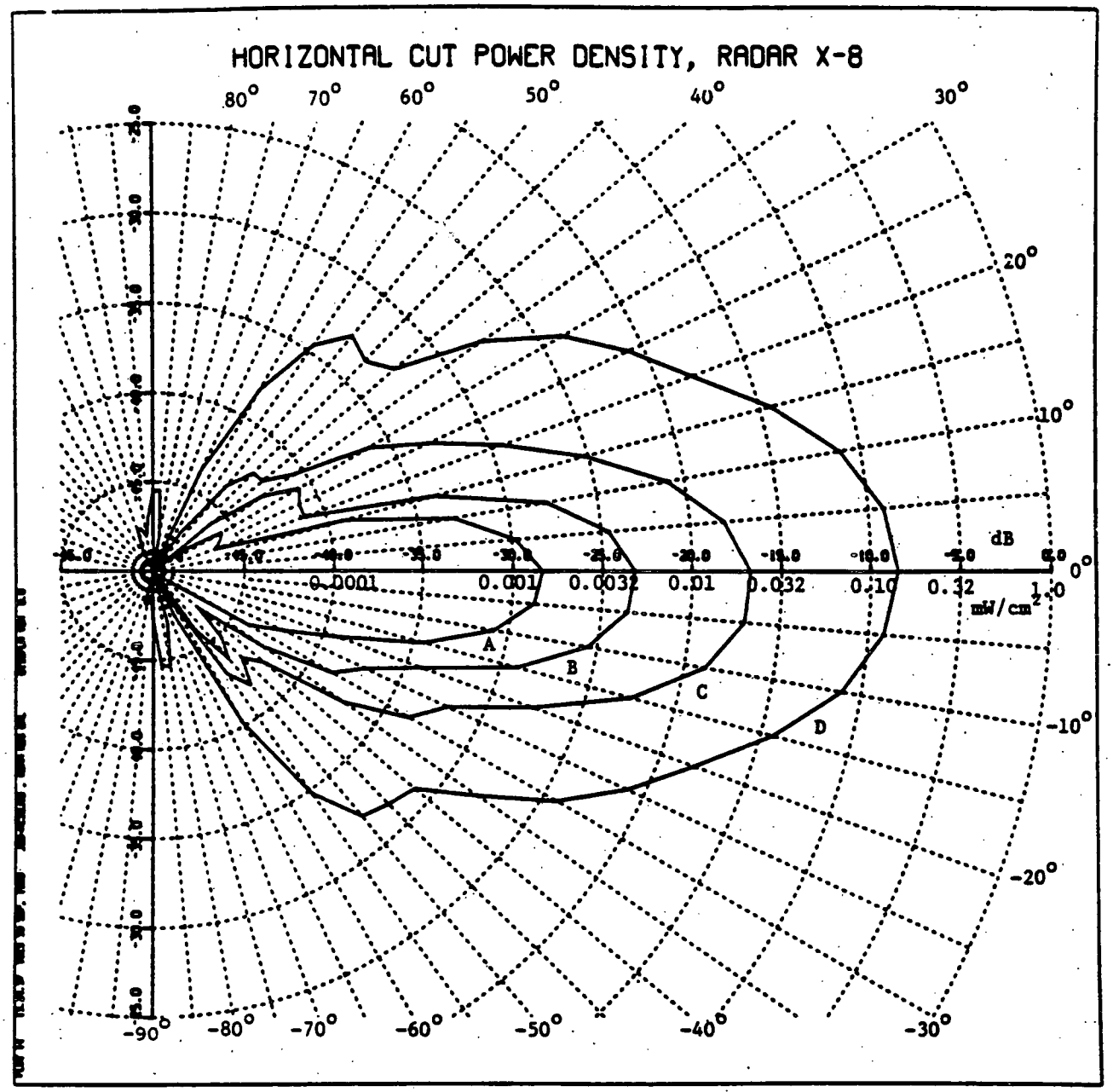
Distance From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 21. Horizontal power patterns for Radar X-7 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



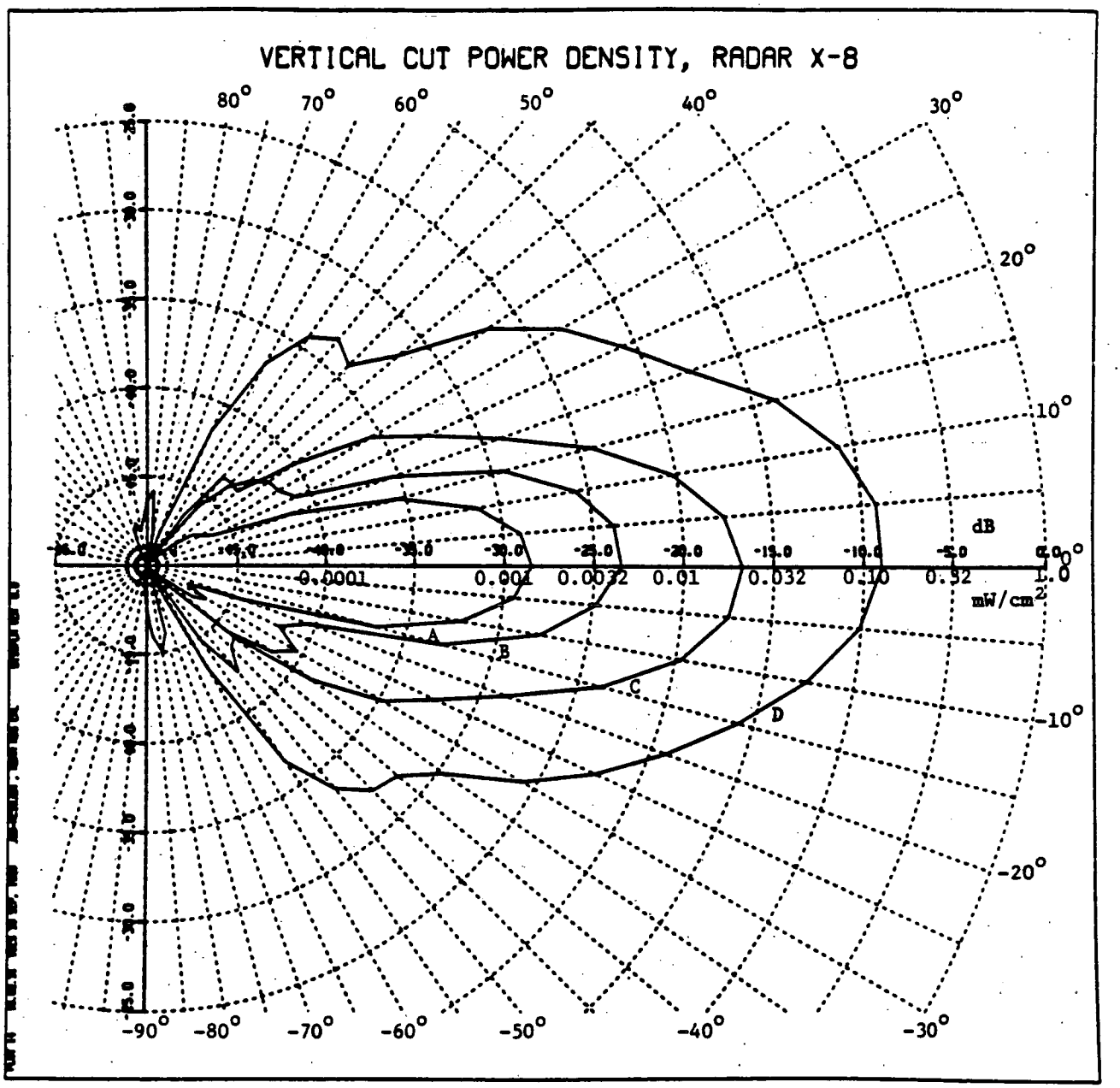
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 22. Vertical power patterns for Radar X-7 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



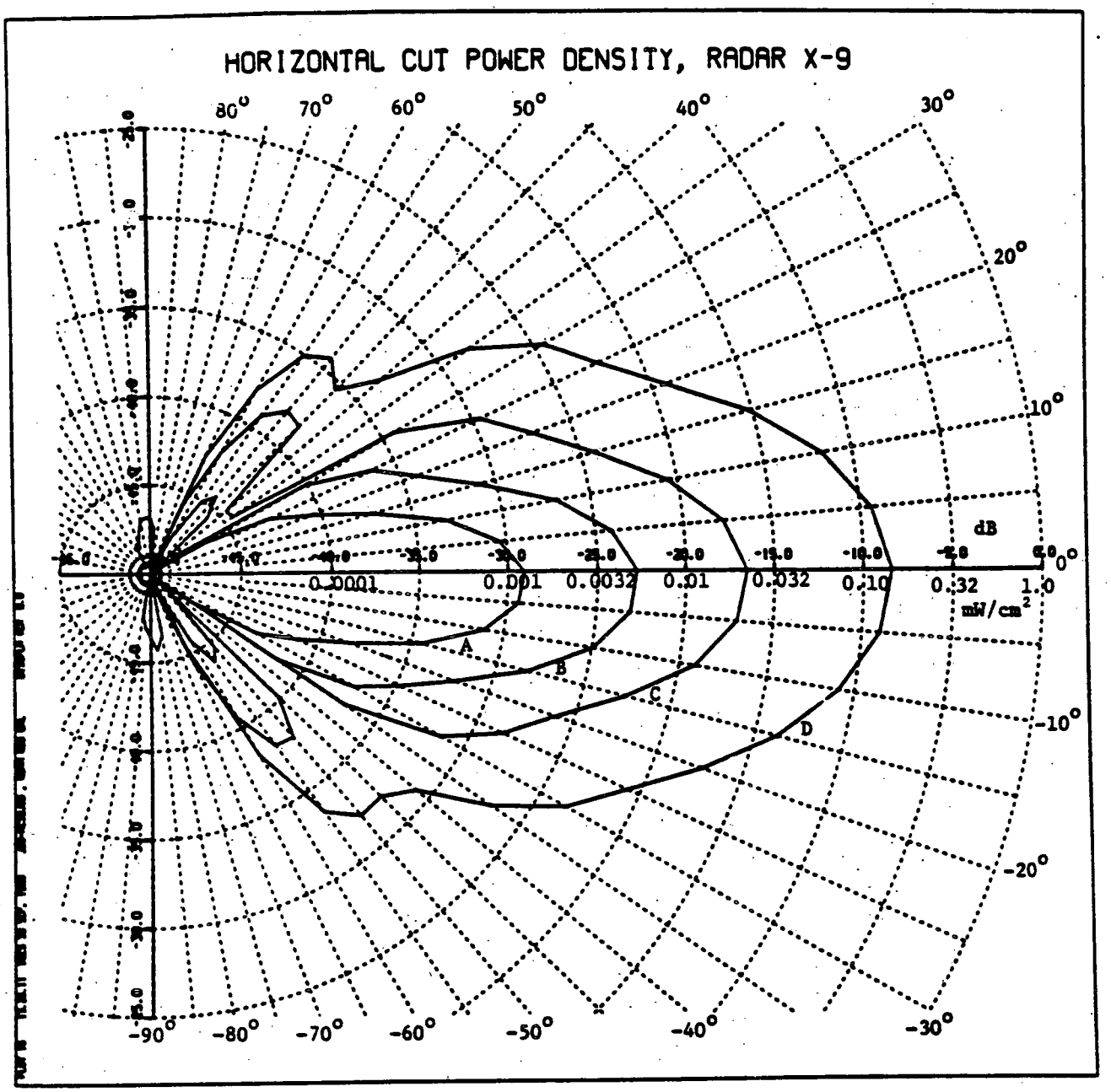
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 23. Horizontal power patterns for Radar X-8 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



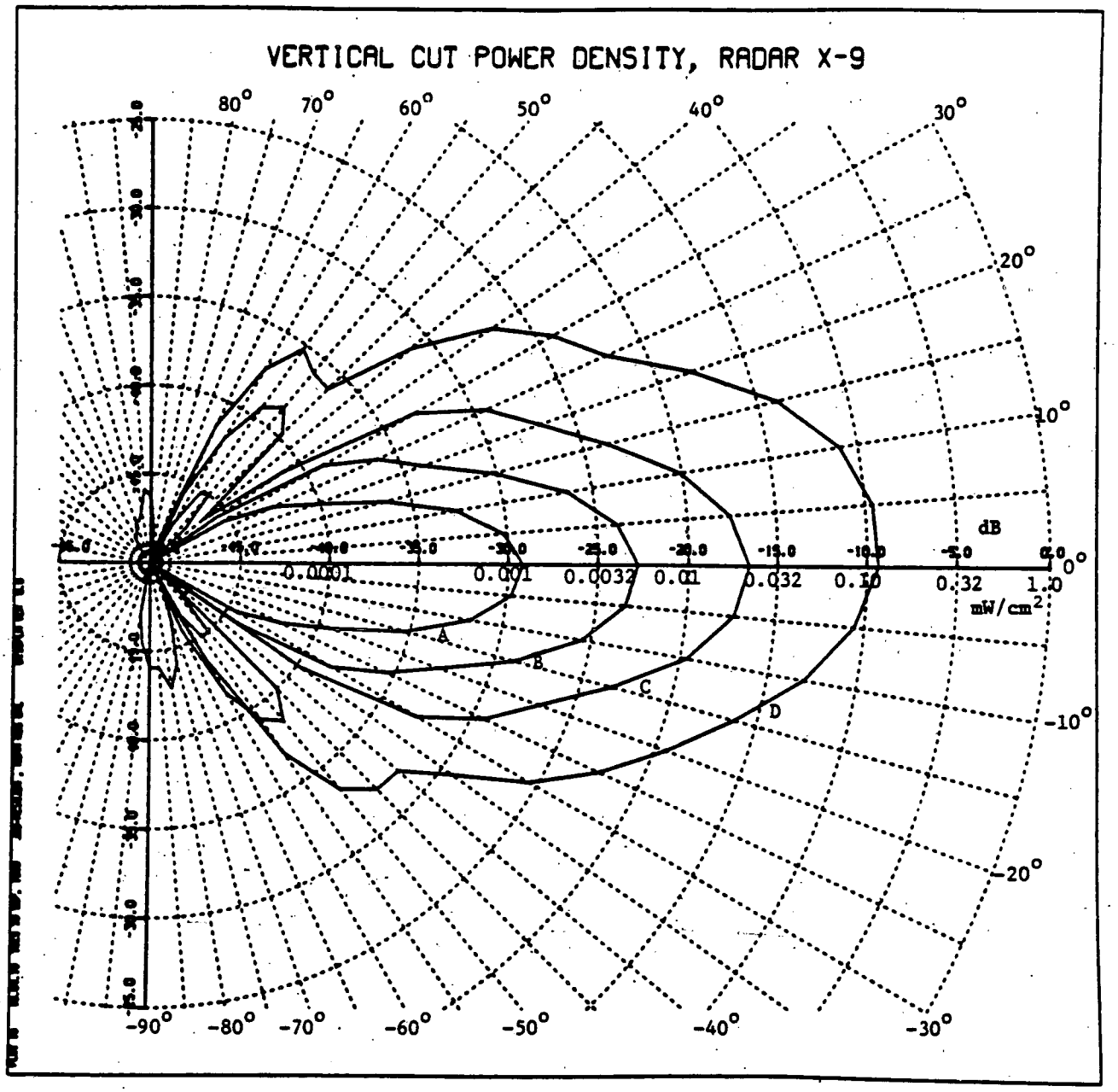
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 24. Vertical power patterns for Radar X-8 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



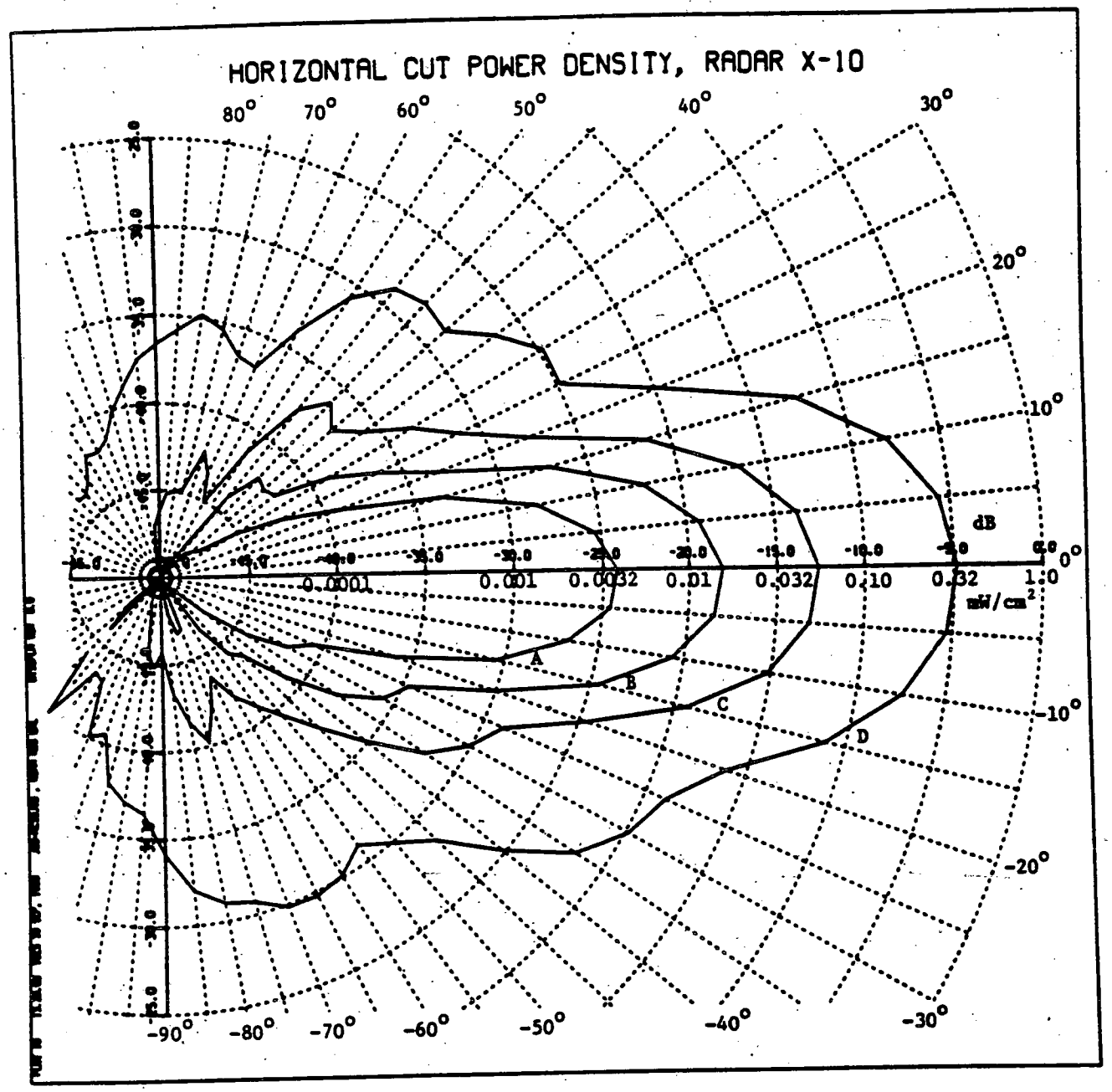
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 25. Horizontal power patterns for Radar X-9 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



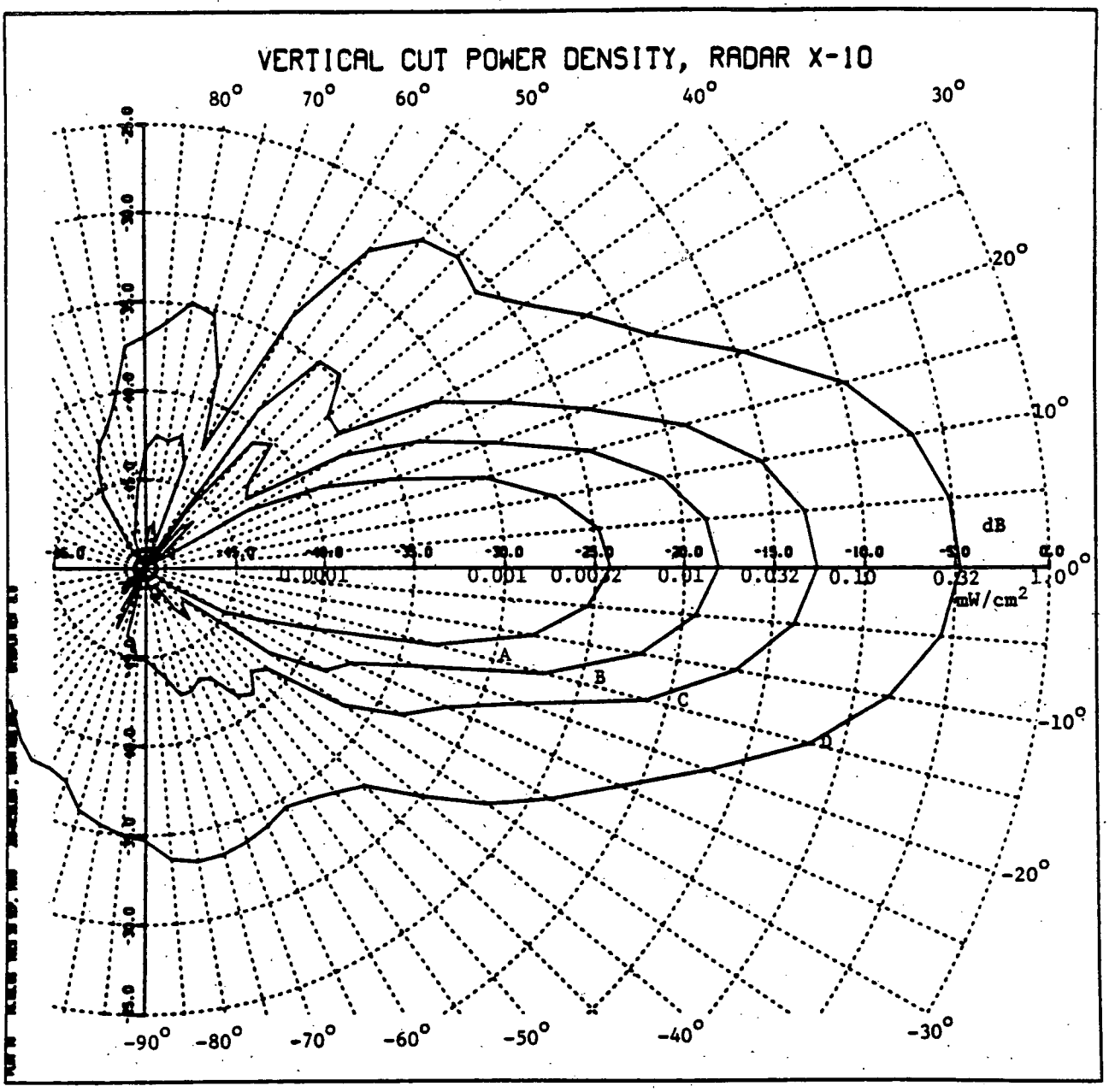
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 26. Vertical power patterns for Radar X-9 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



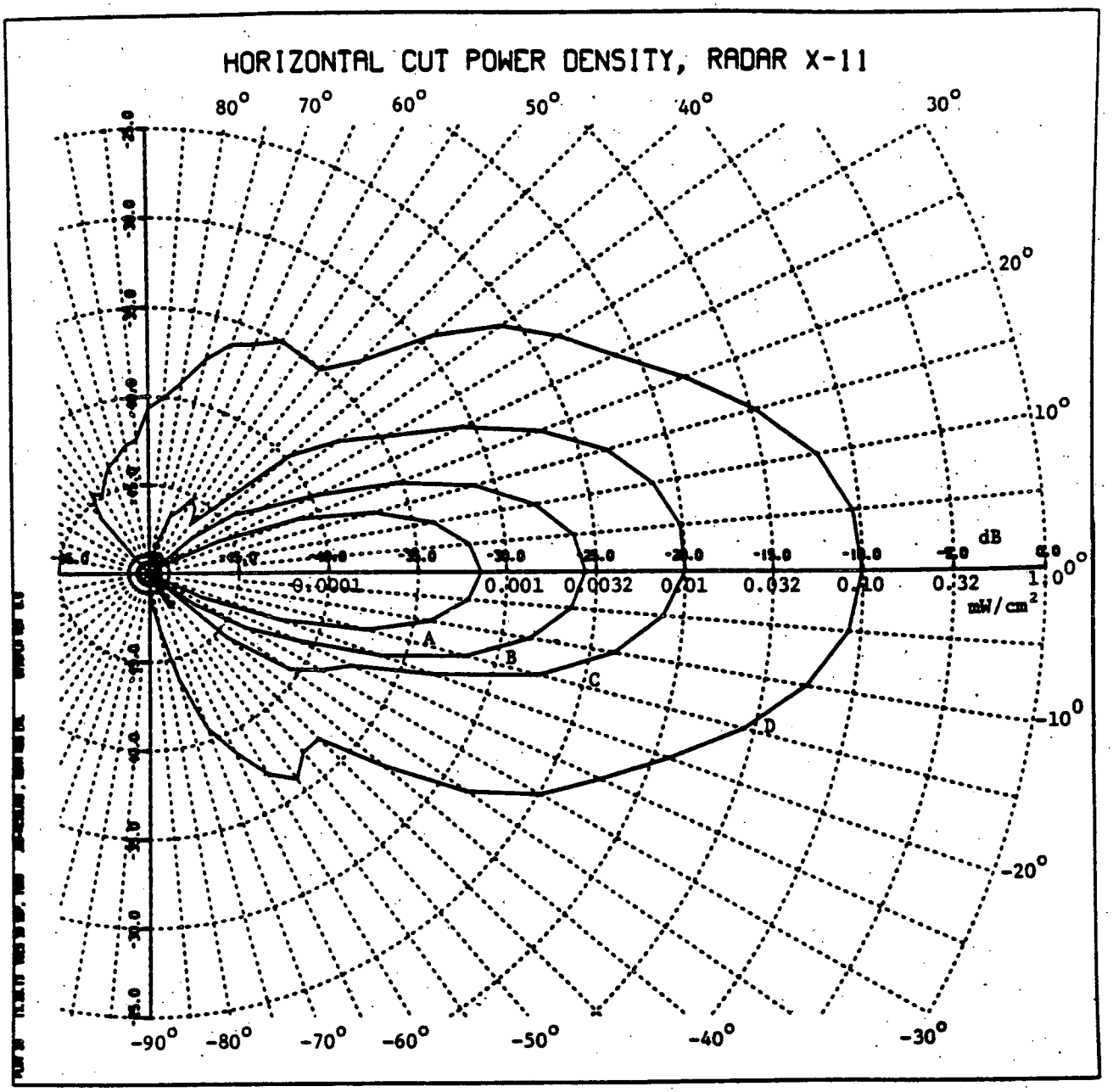
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 27. Horizontal power patterns for Radar X-10 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



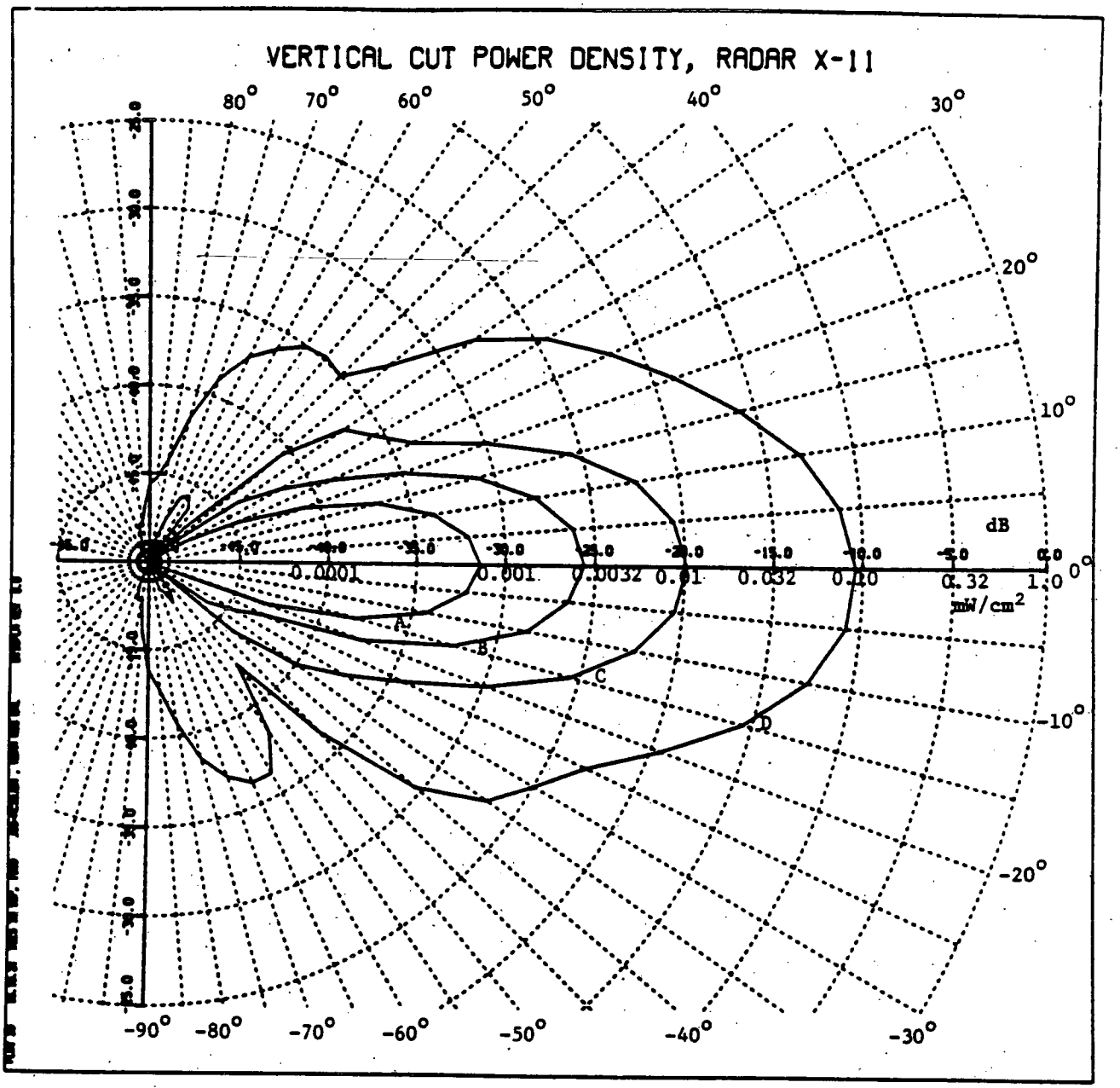
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 28. Vertical power patterns for Radar X-10 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



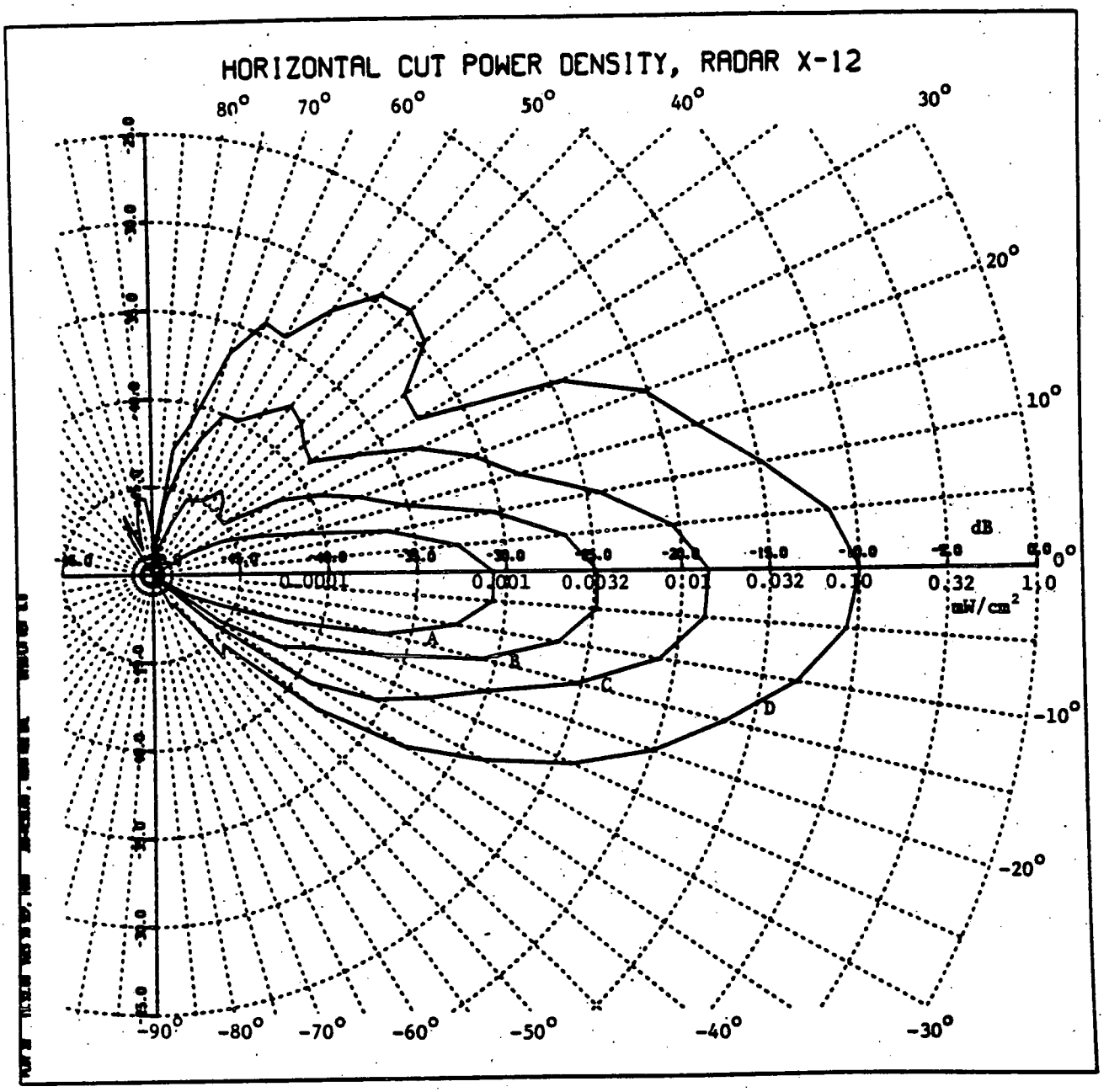
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 29. Horizontal power patterns for Radar X-11 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



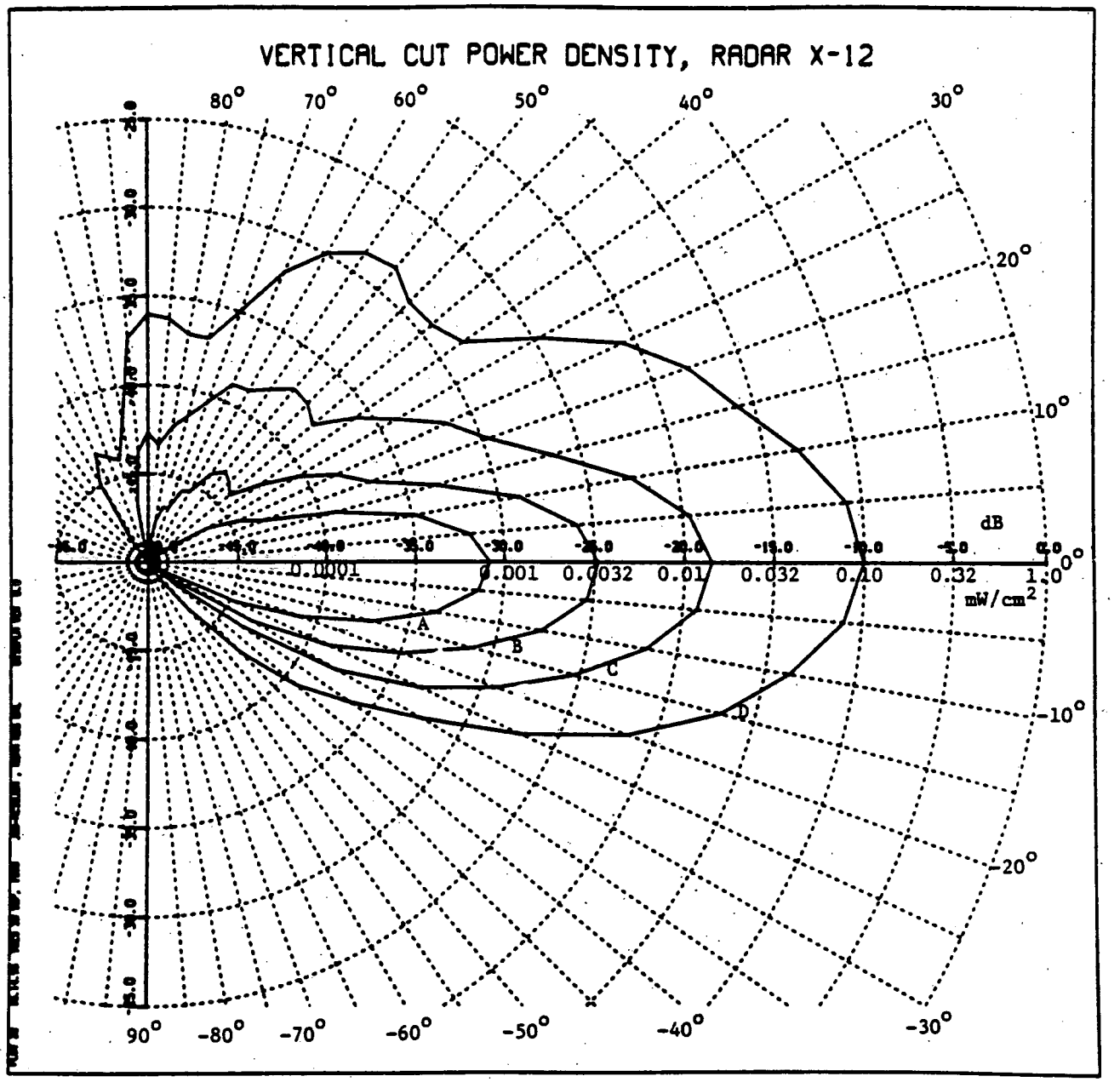
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 30. Vertical power patterns for Radar X-11 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



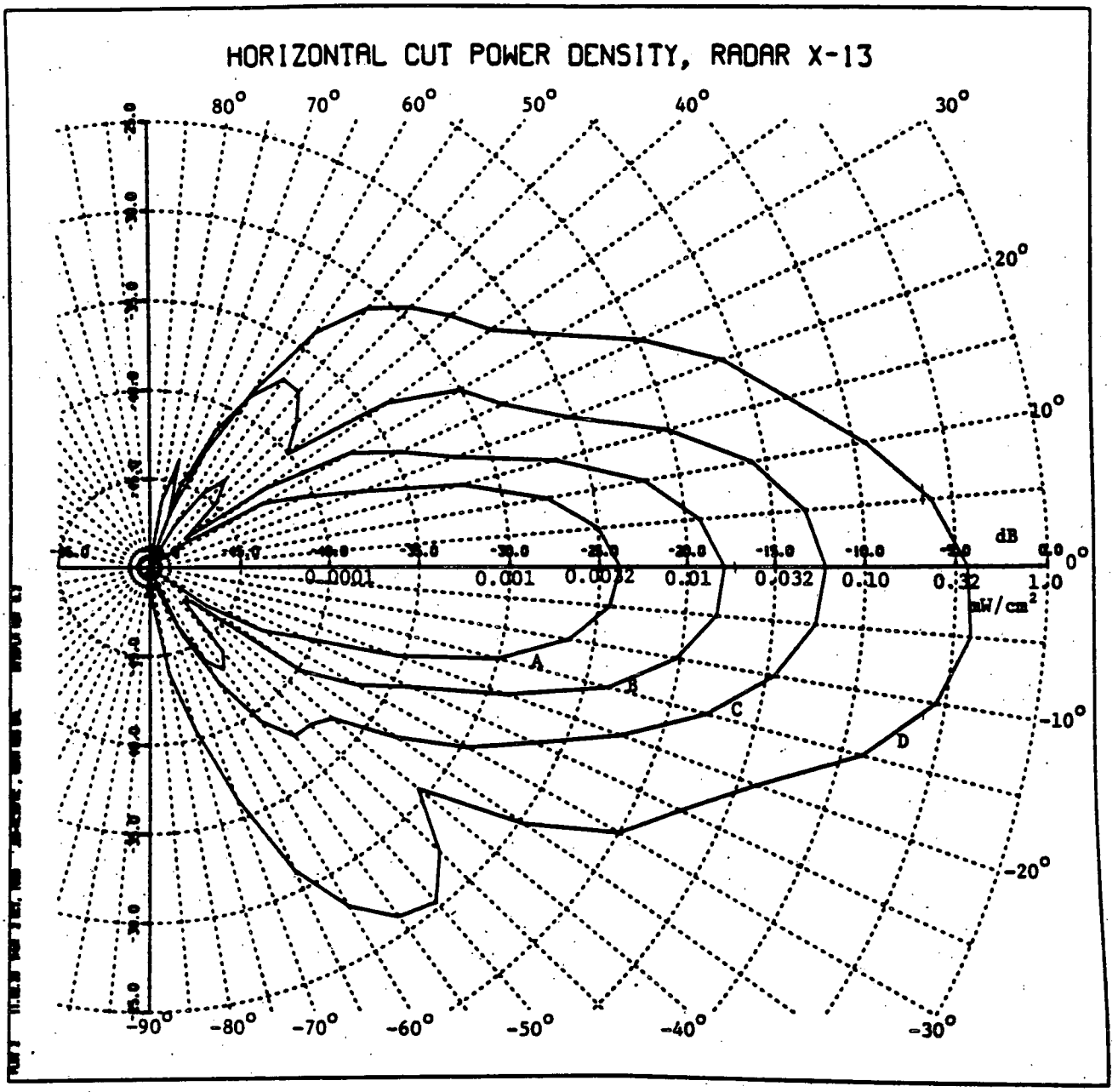
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 31. Horizontal power patterns for Radar X-12 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



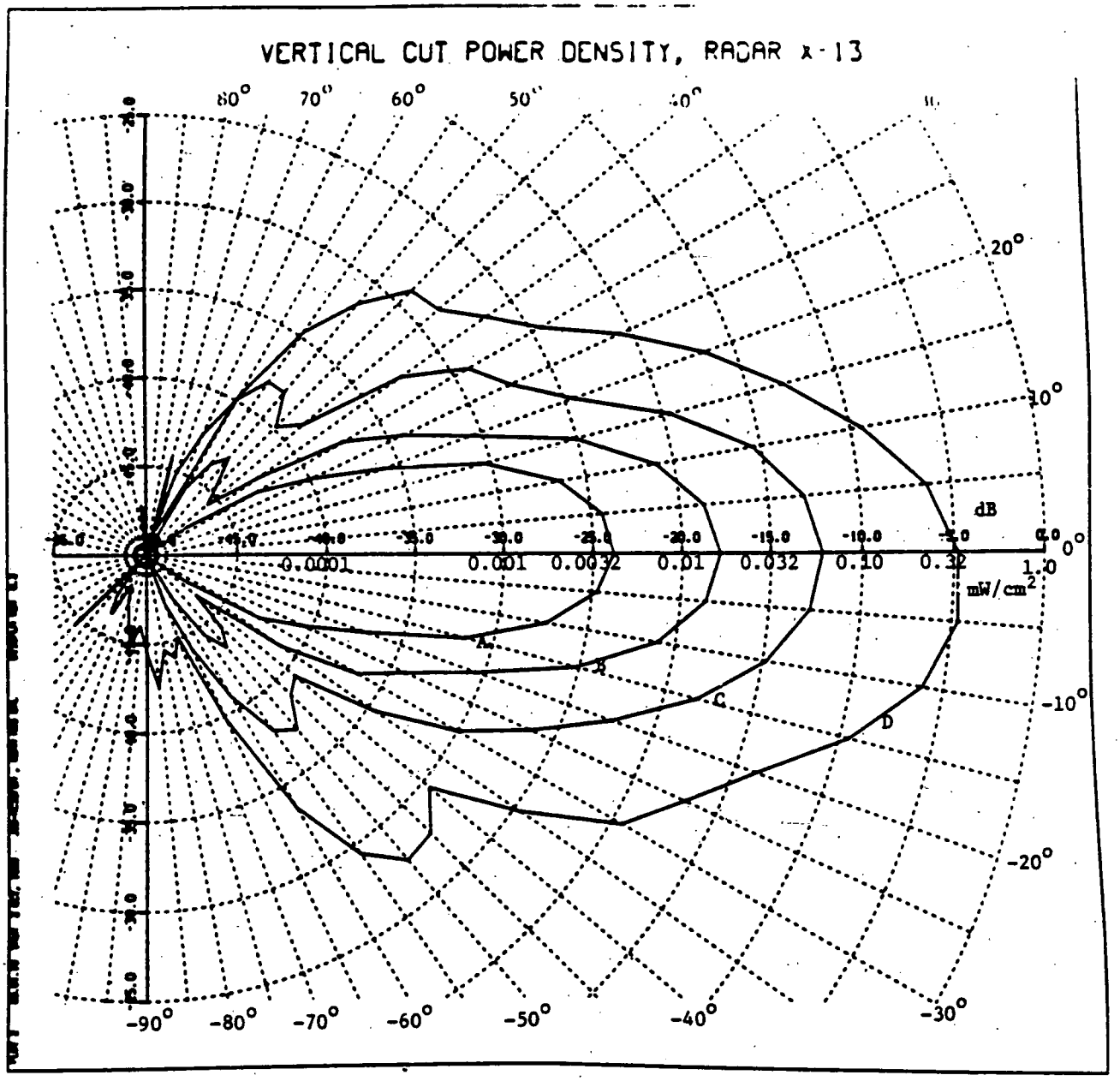
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 32. Vertical power patterns for Radar X-12 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



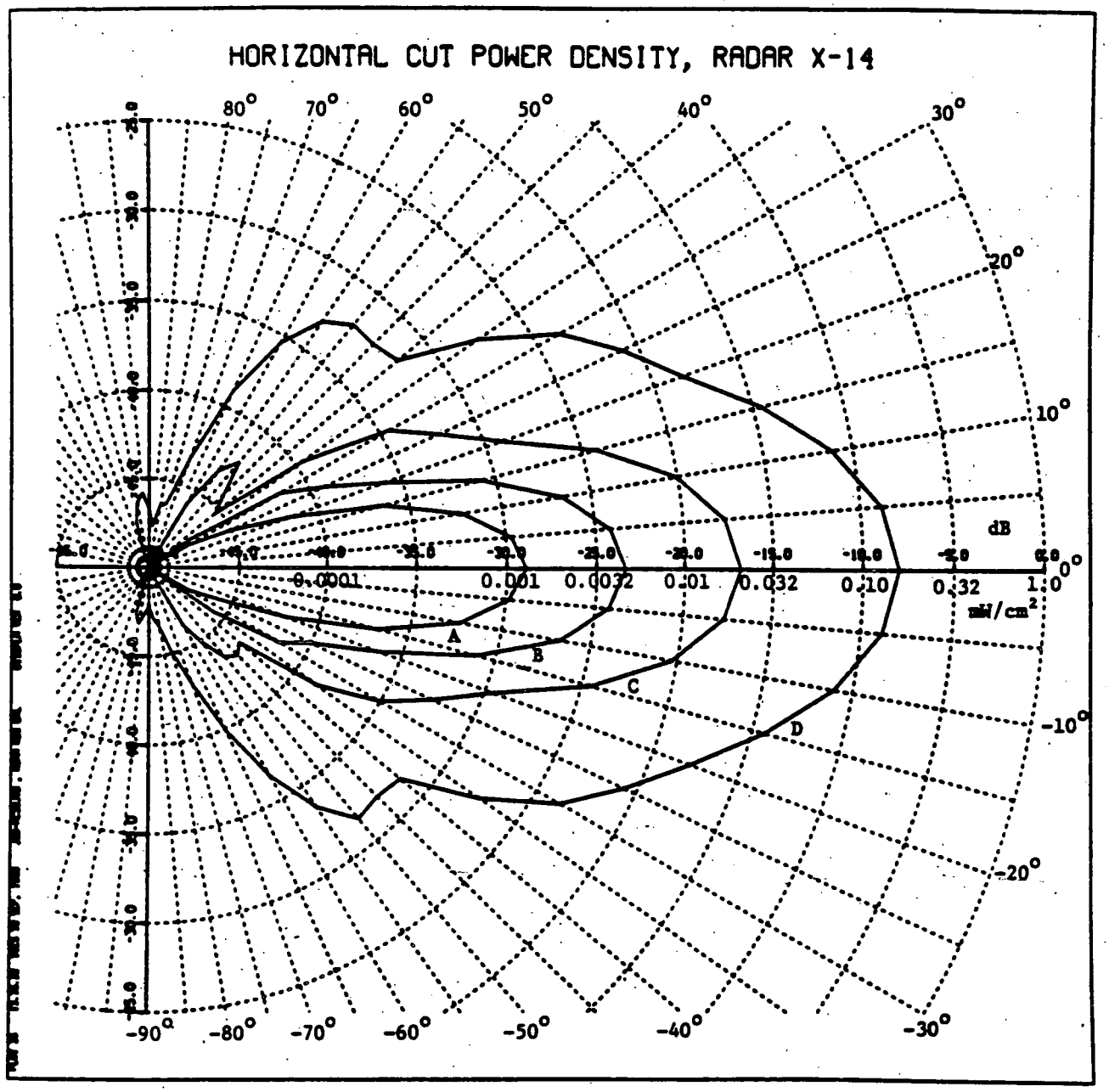
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 33. Horizontal power patterns for Radar X-13 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



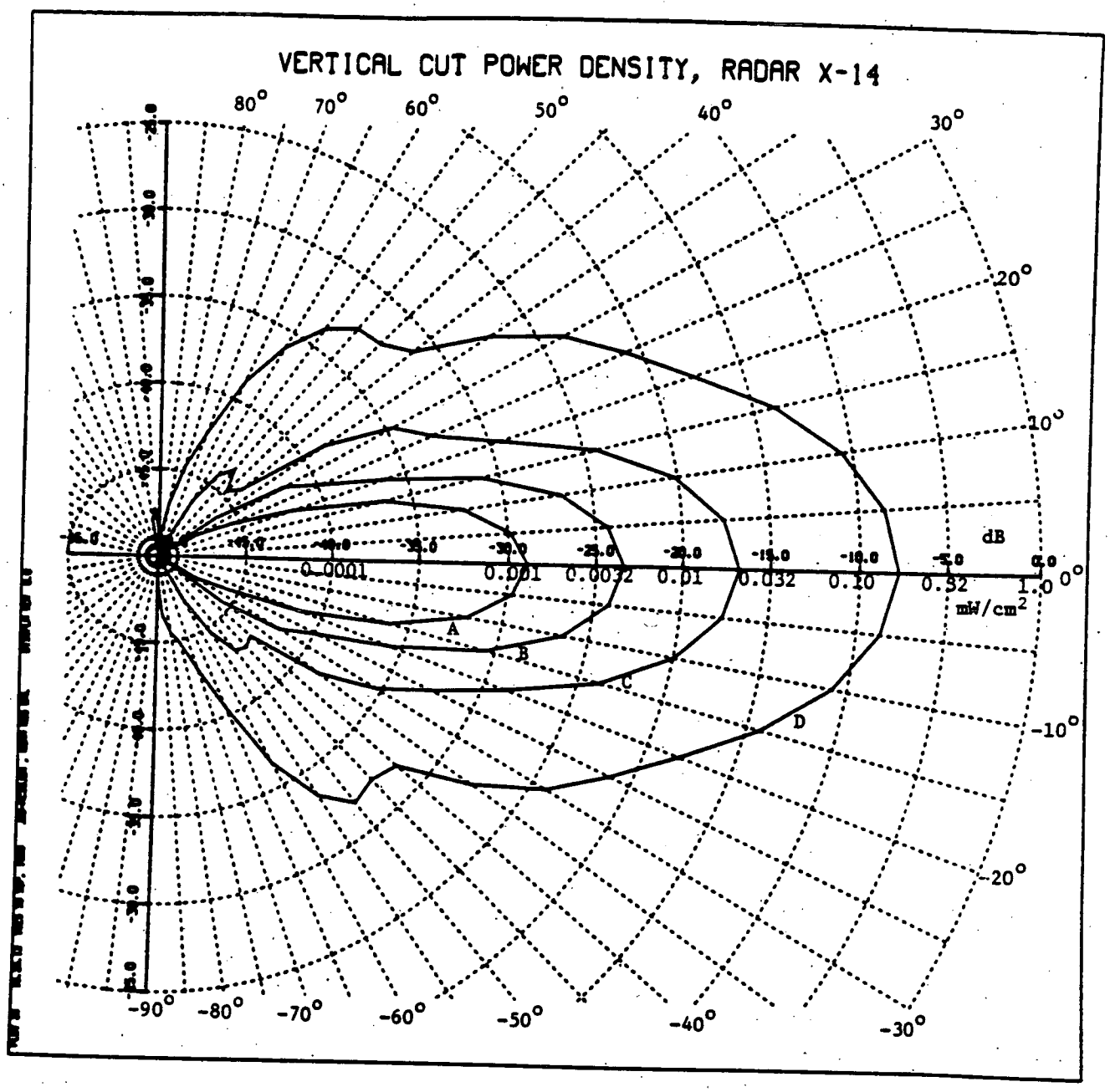
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 34. Vertical power patterns for Radar X-13 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction in the main beam.



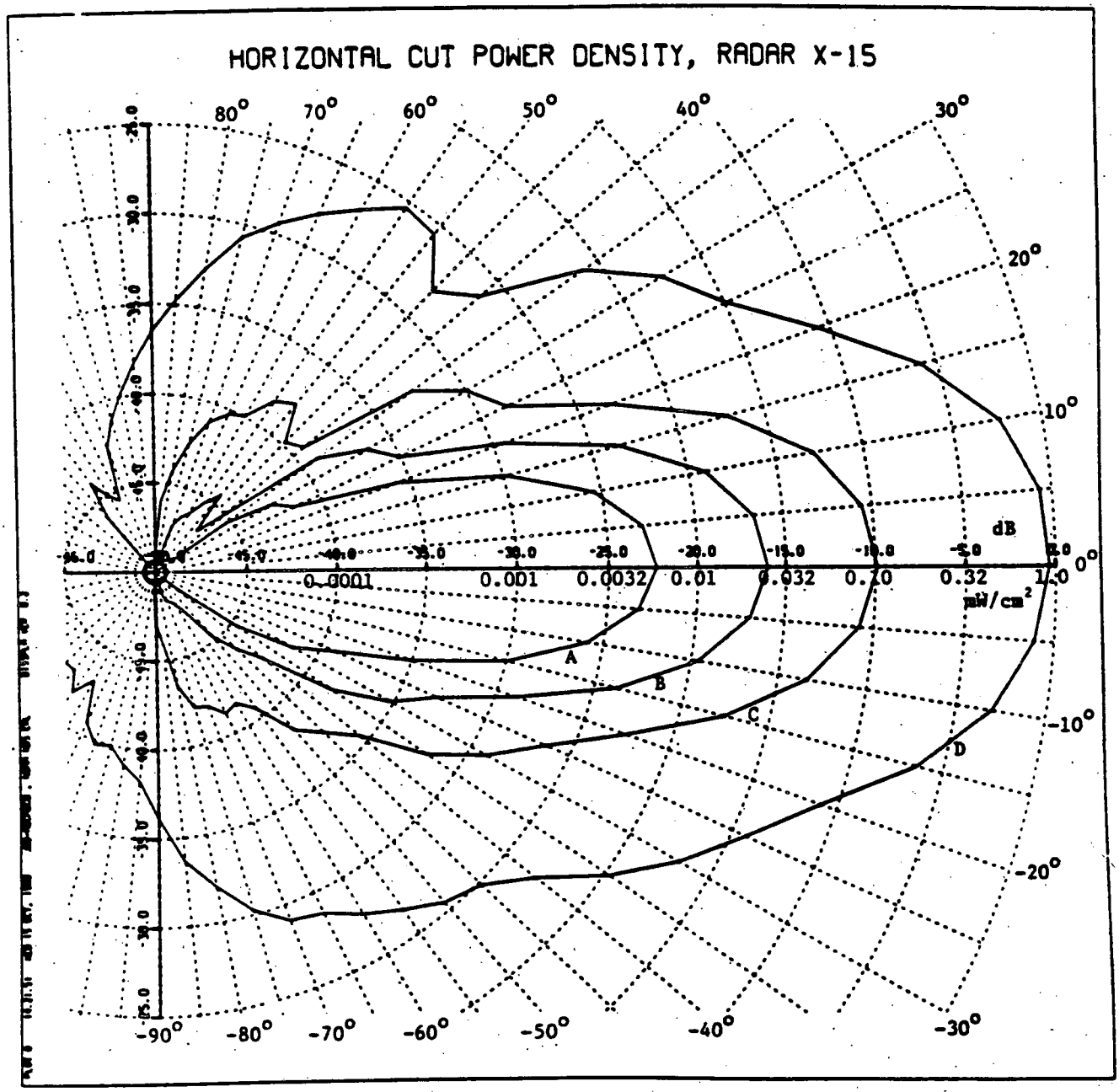
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 35. Horizontal power patterns for Radar X-14 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



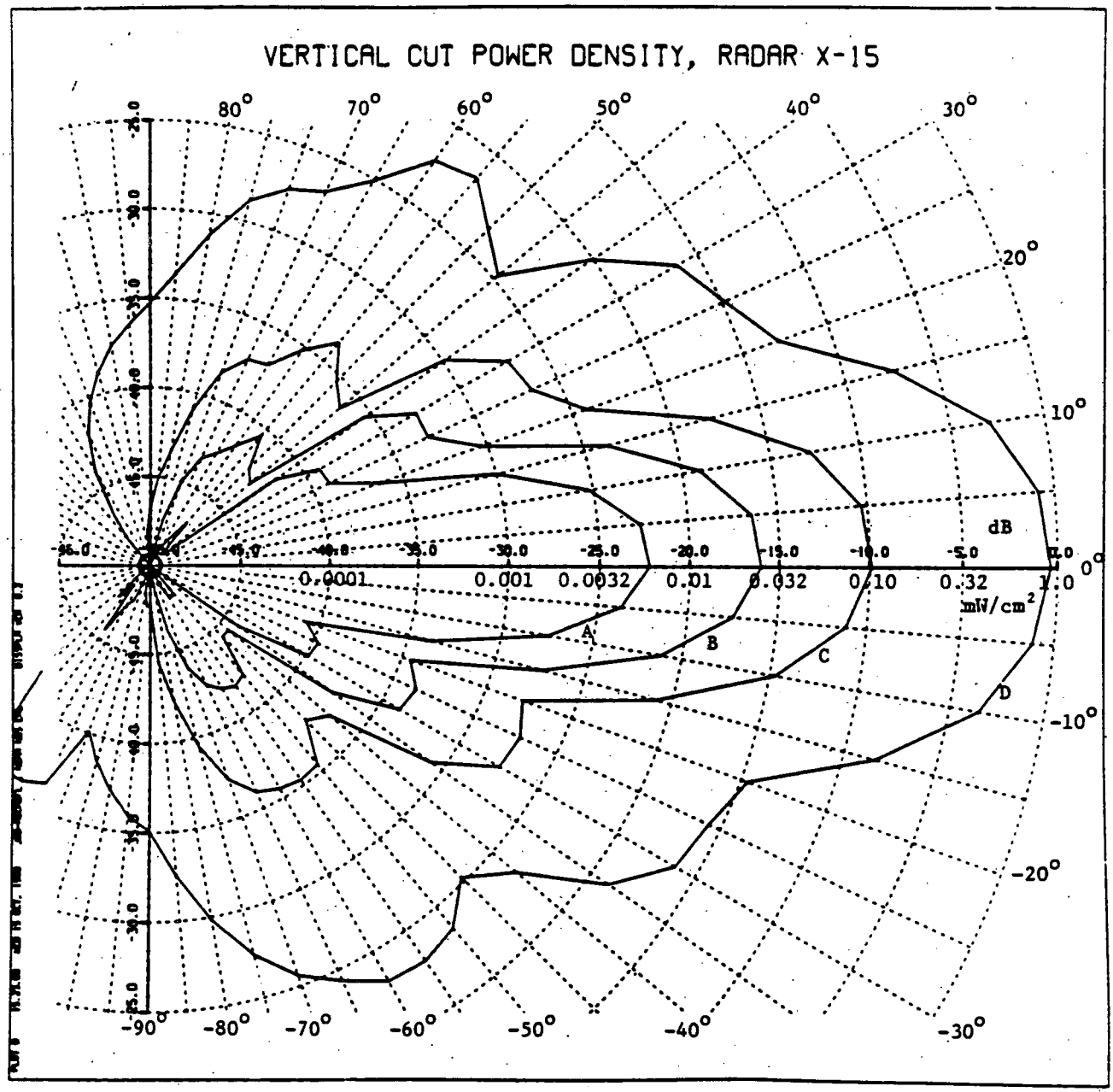
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 36. Vertical power patterns for Radar X-14 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



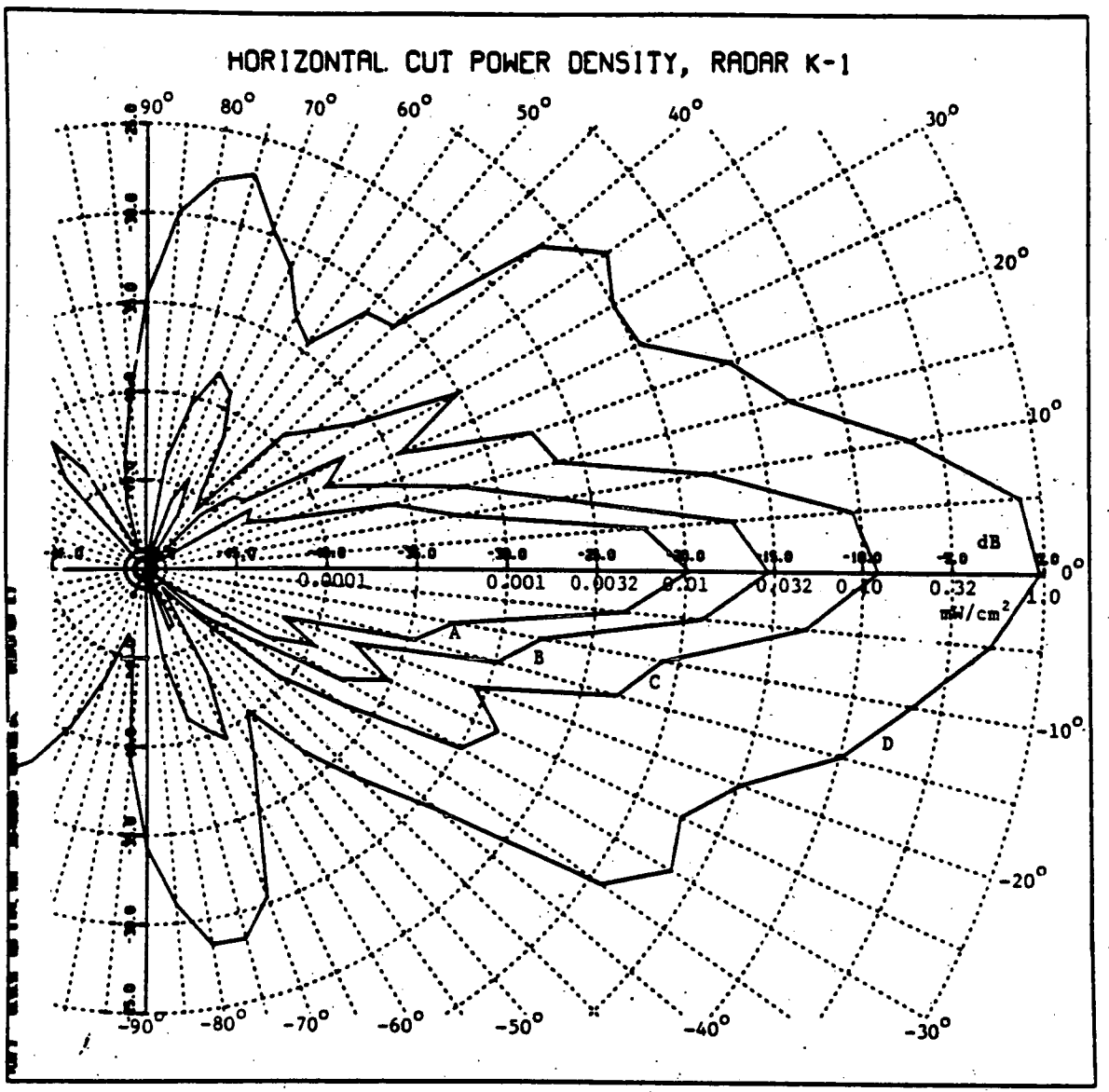
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 37. Horizontal power patterns for Radar X-15 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



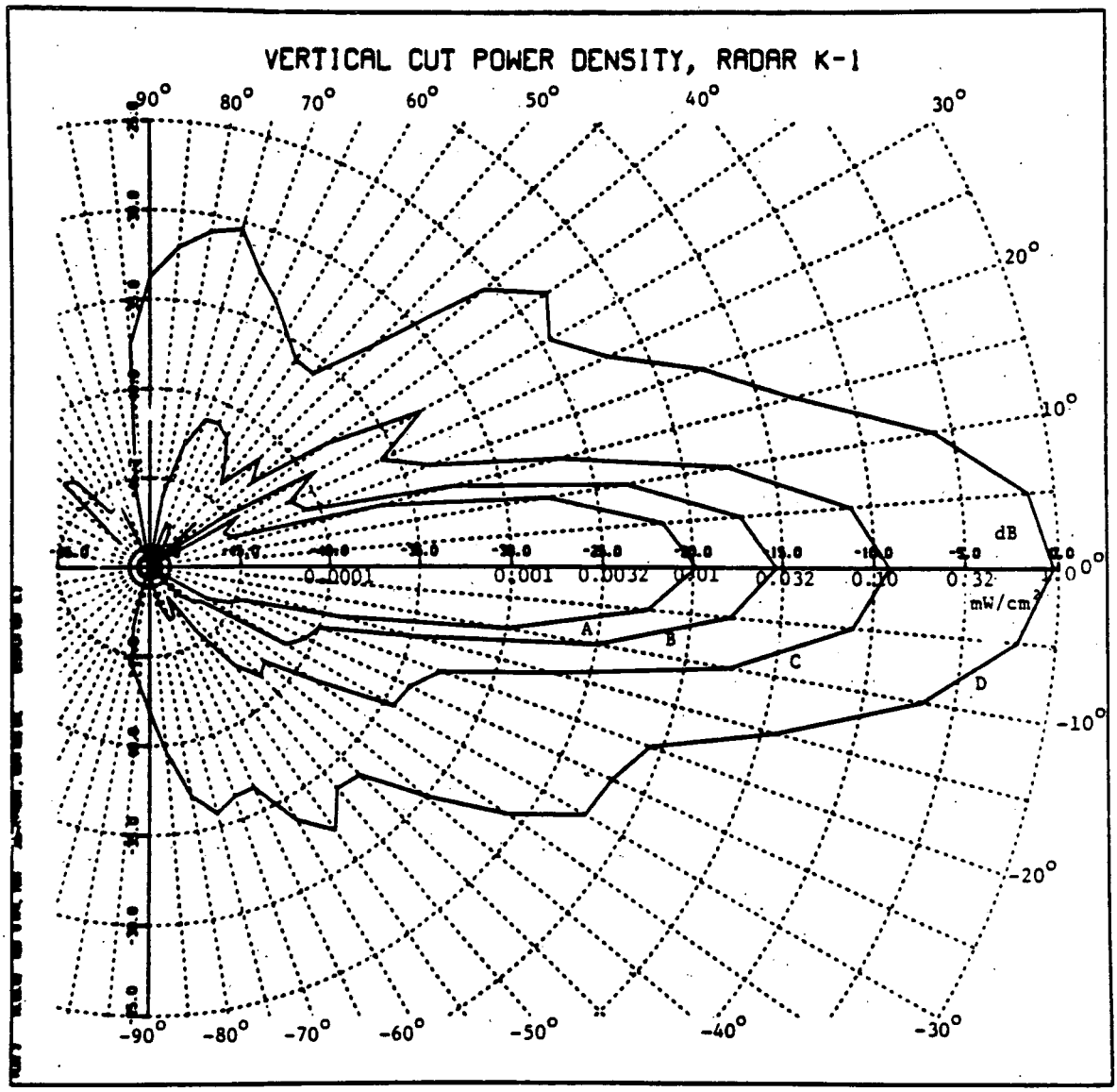
Distances From Radar (Inches): A = 144, B = 72, C = 36, D = 12

Figure 38. Vertical power patterns for Radar X-15 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 39. Horizontal power patterns for Radar K-1 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 40. Vertical power patterns for Radar K-1 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.

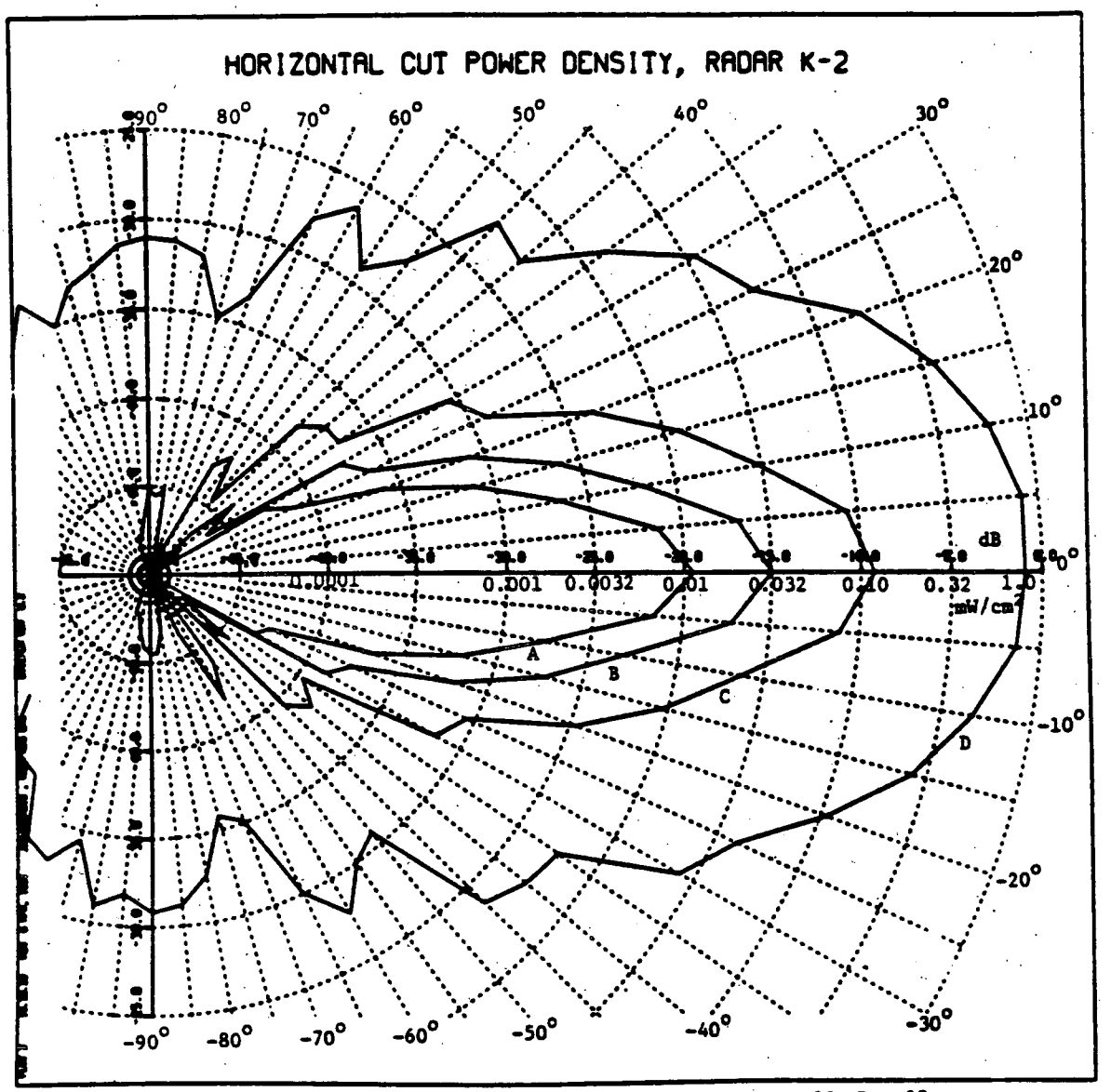
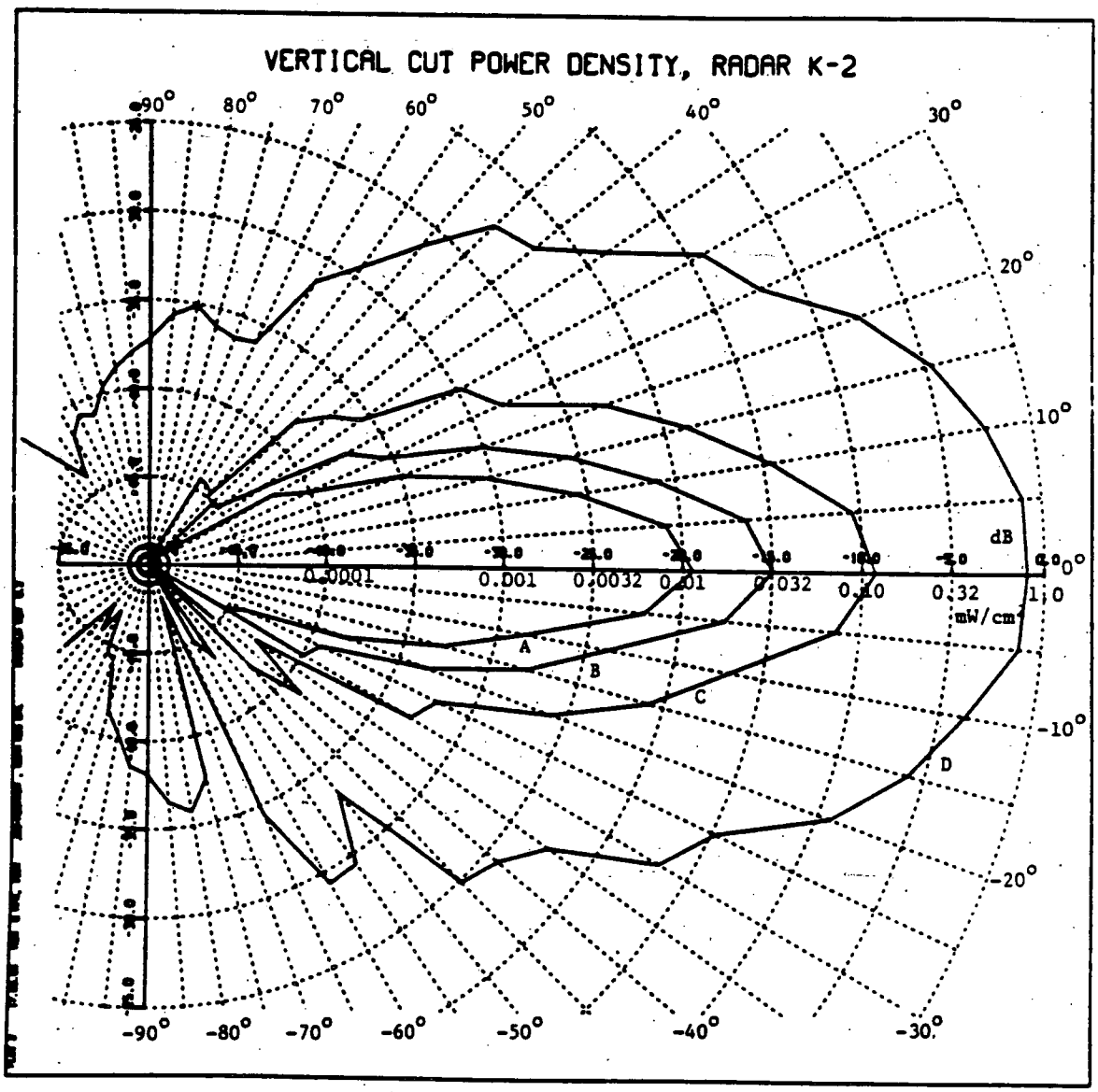


Figure 41. Horizontal power patterns for Radar K-2 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 42. Vertical power patterns for Radar K-2 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.

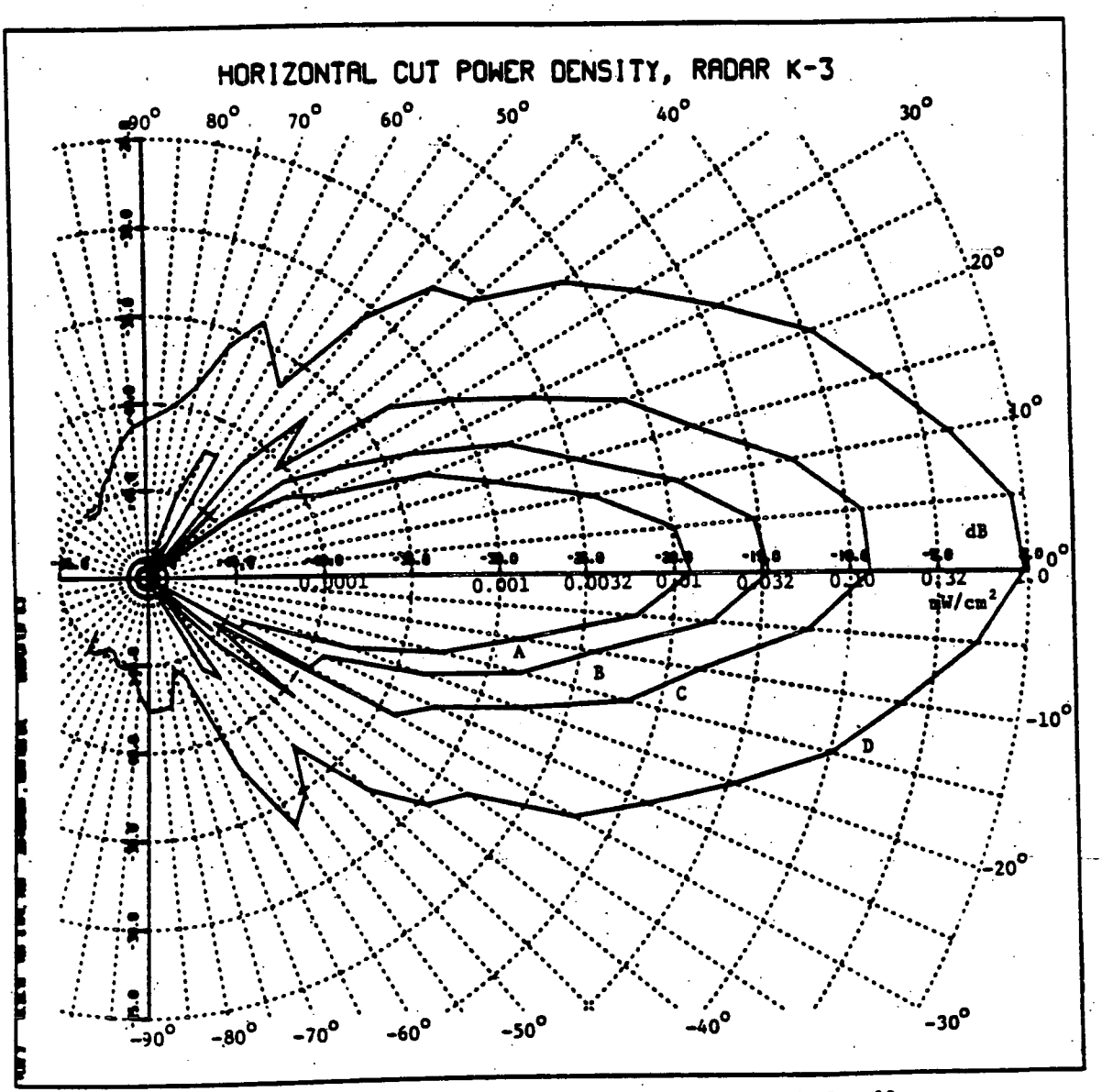
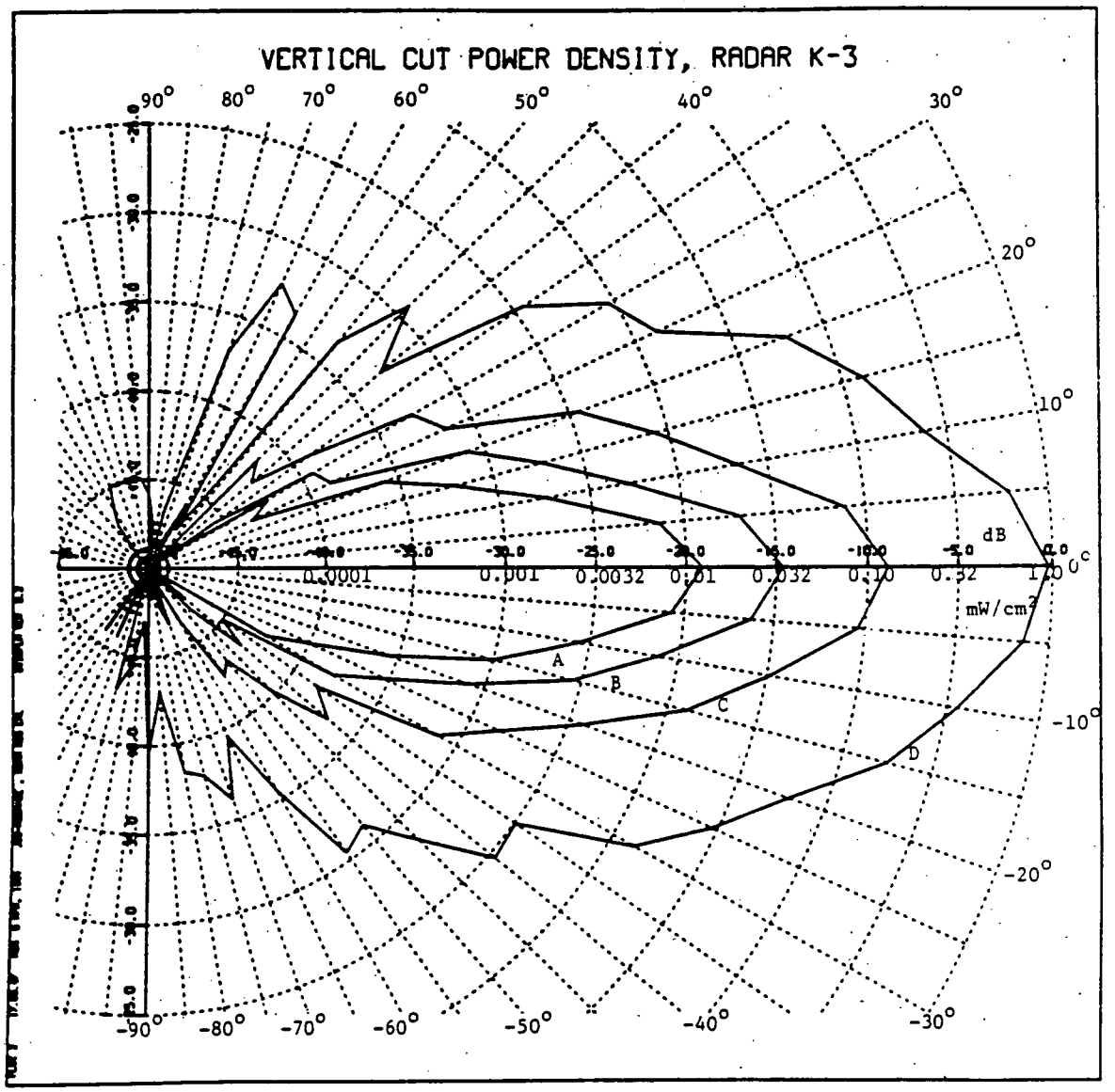
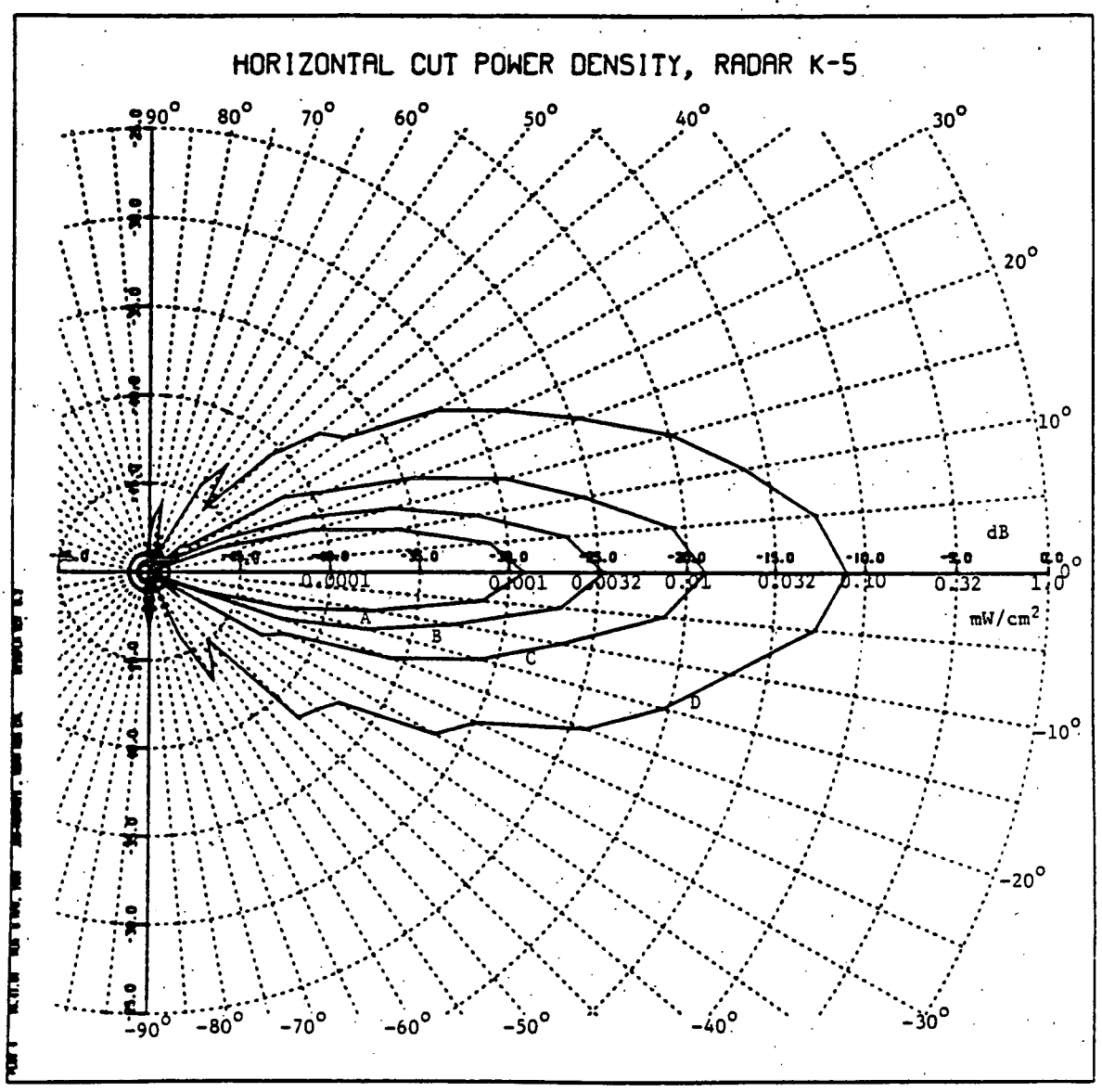


Figure 43. Horizontal power patterns for Radar K-3 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



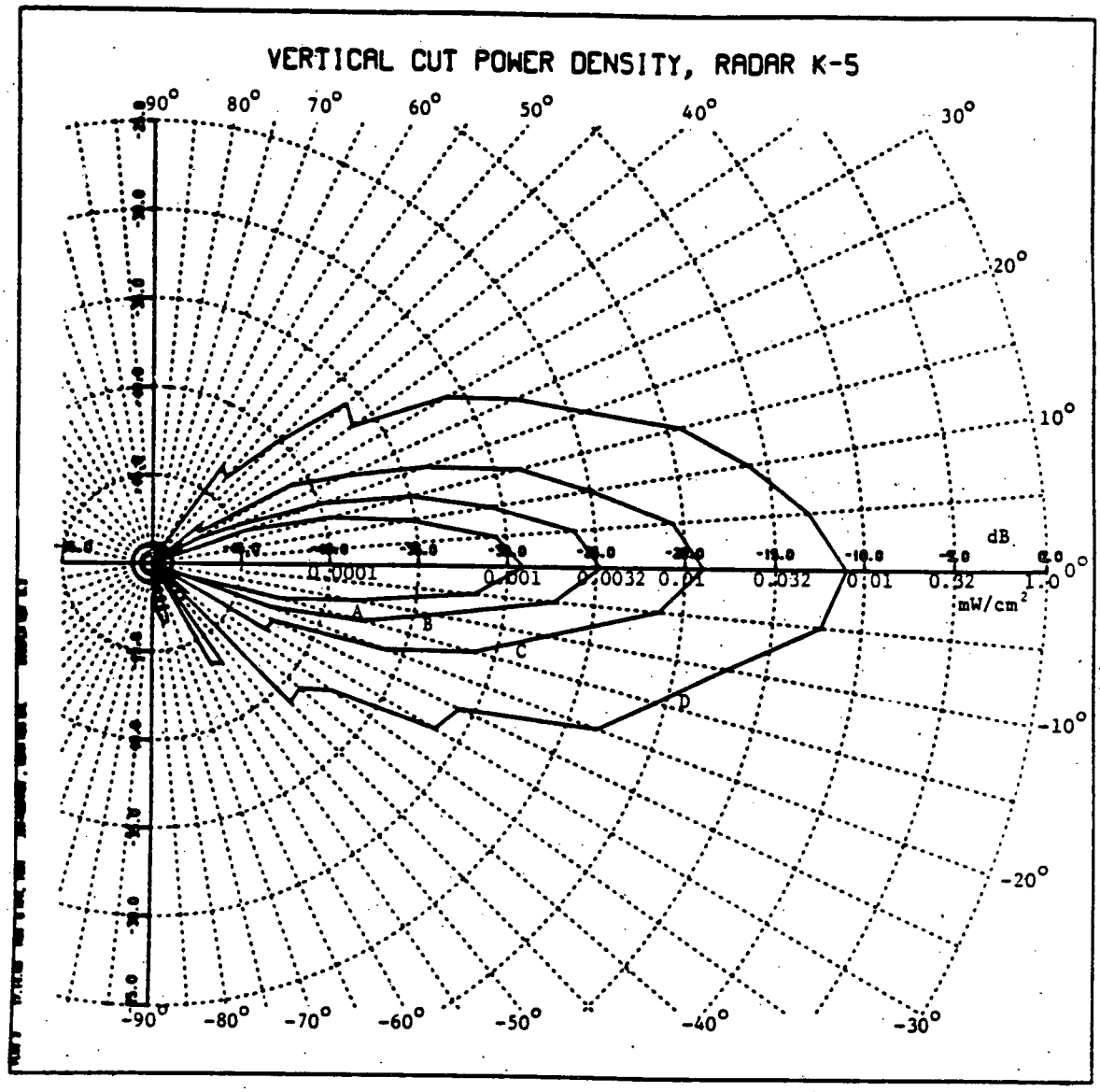
Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 44. Vertical power patterns for Radar K-3 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 45. Horizontal power patterns for Radar K-5 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 46. Vertical power patterns for Radar K-5 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.

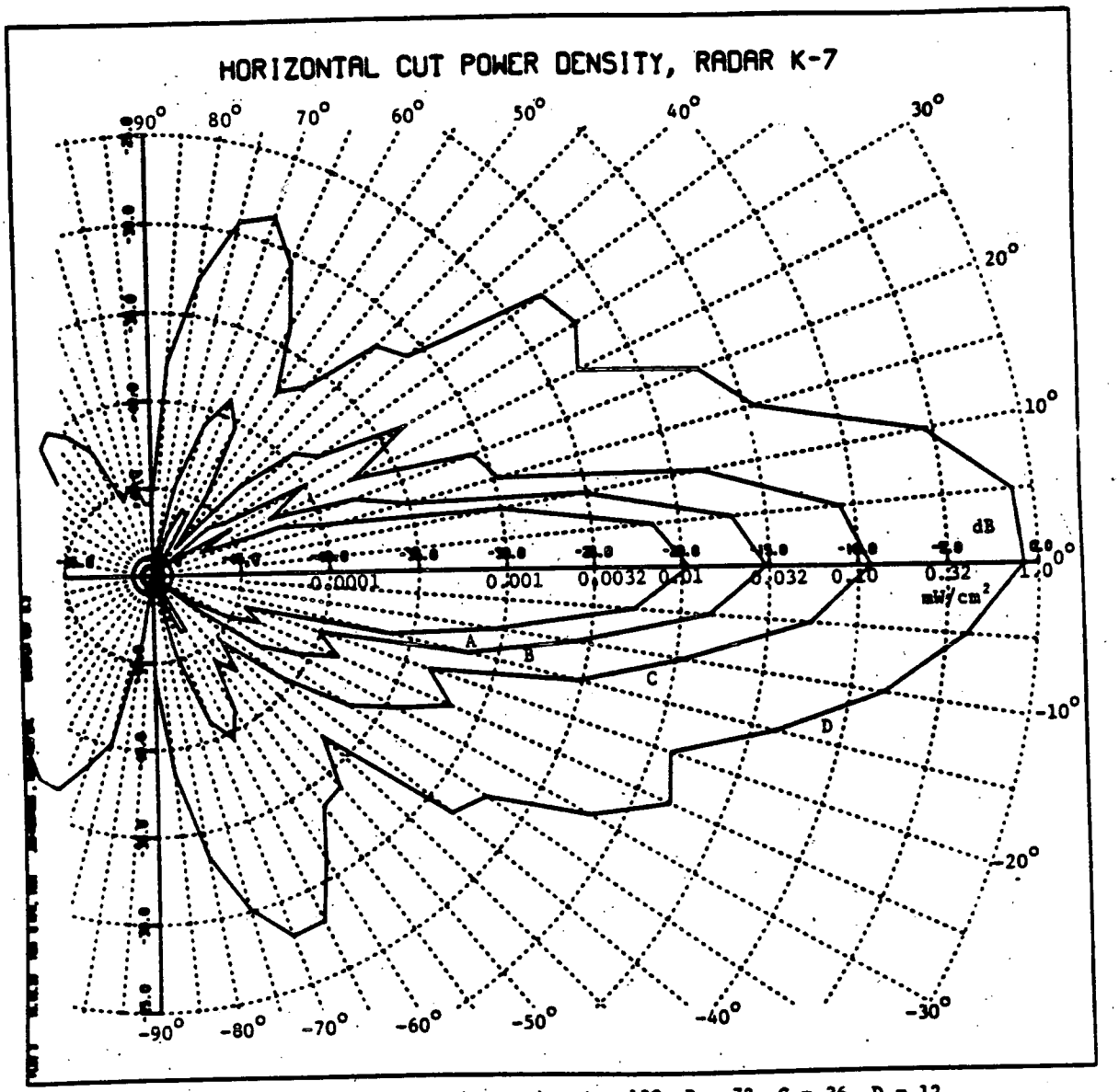


Figure 47. Horizontal power patterns for Radar K-7 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.

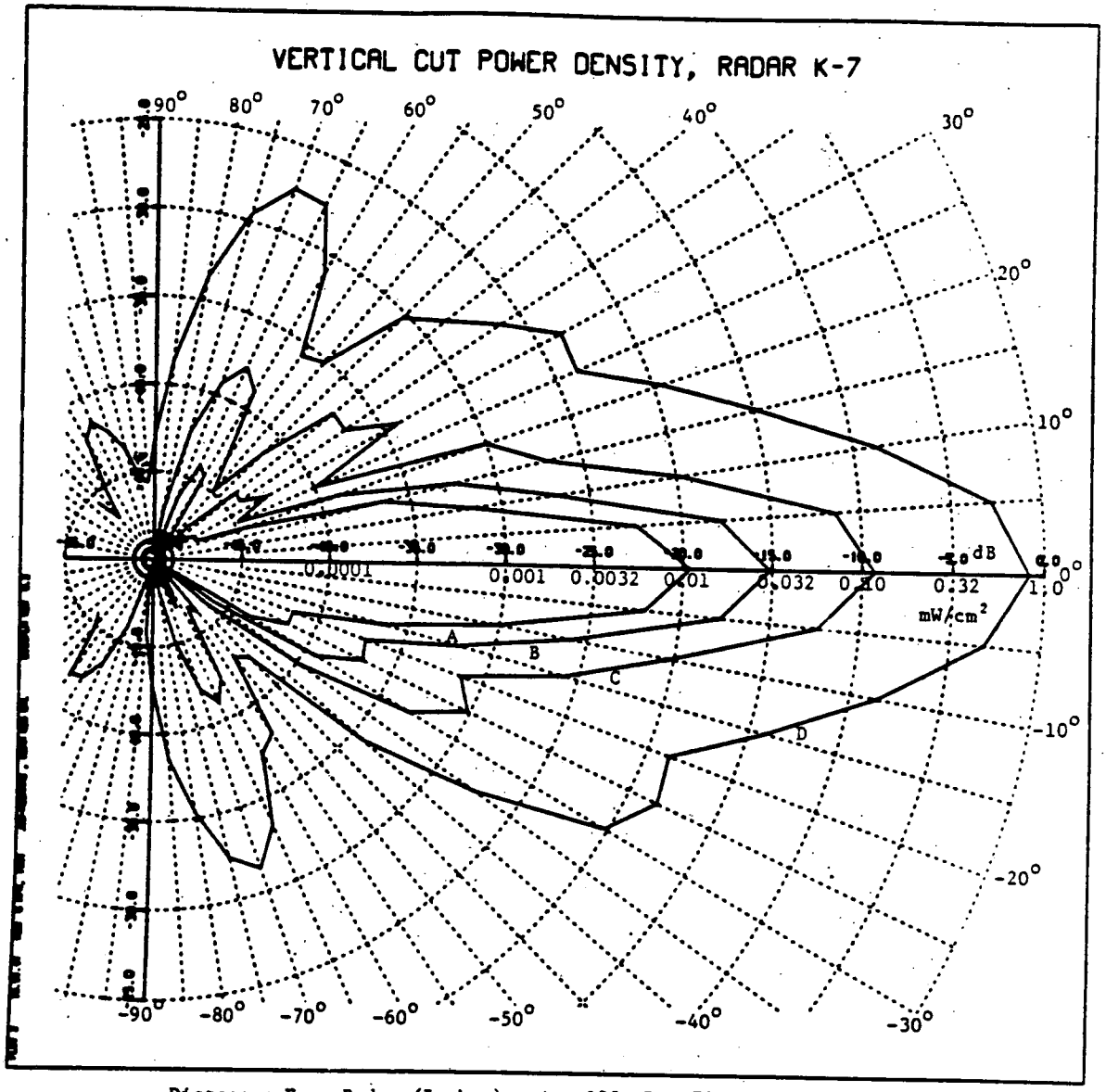
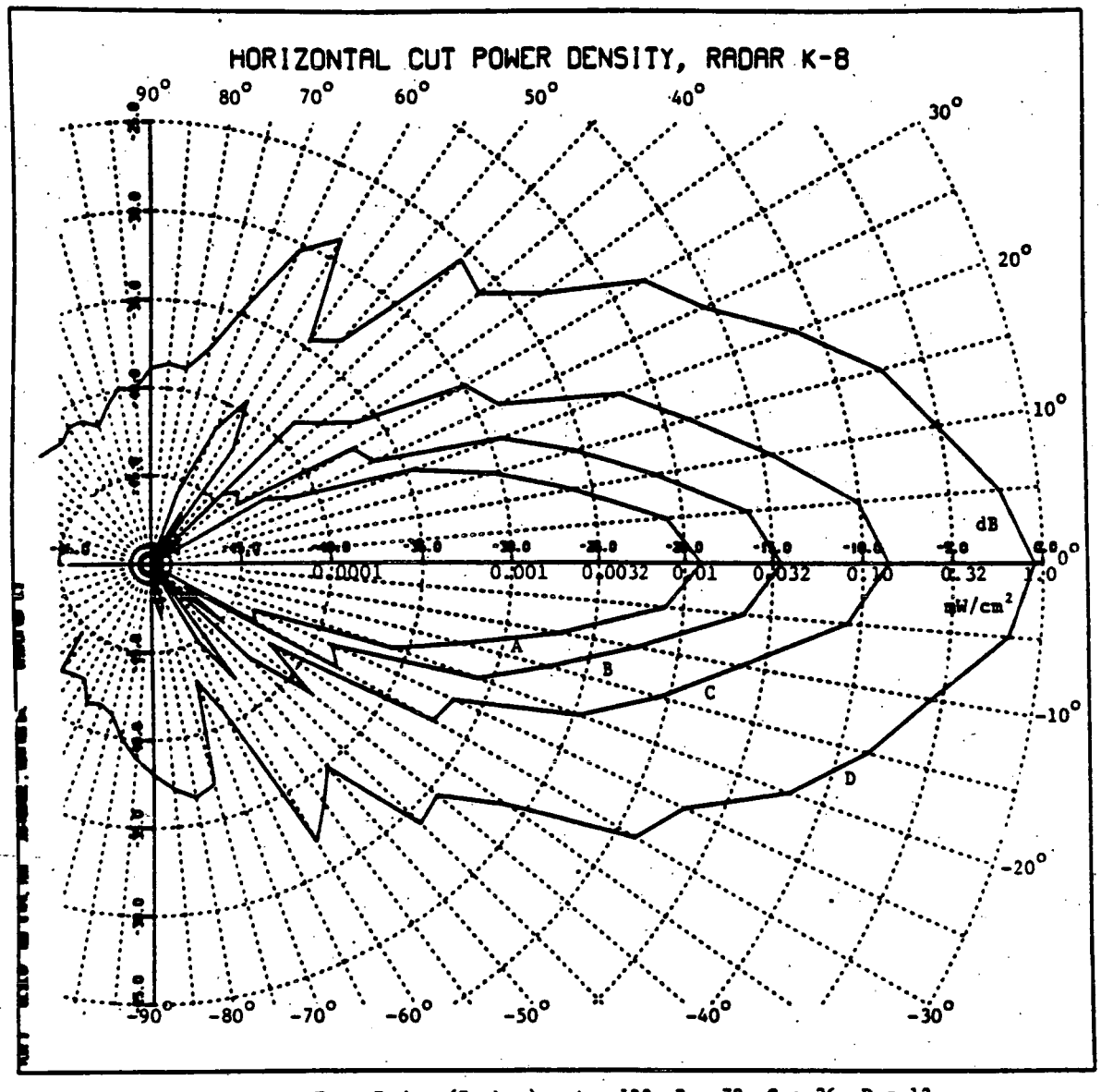
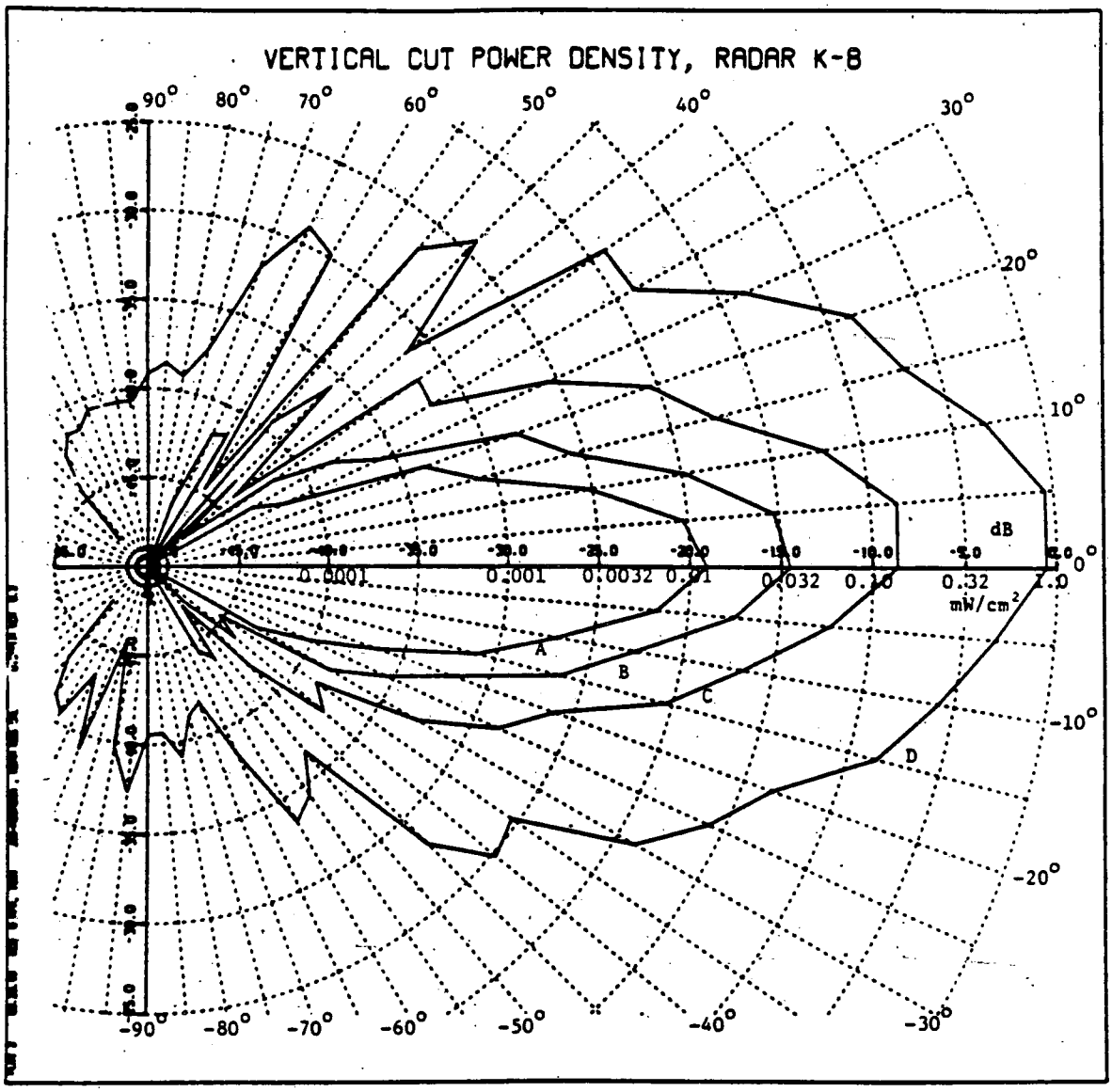


Figure 48. Vertical power patterns for Radar K-7 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.



Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 49. Horizontal power patterns for Radar K-8 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the azimuth angle in degrees. Zero degrees coincides with the direction of the main beam.



Distances From Radar (Inches): A = 120, B = 72, C = 36, D = 12

Figure 50. Vertical power patterns for Radar K-8 obtained at four distances from the aperture. Each curve displays the total power density in mW/cm^2, or dB with respect to 1 mW/cm^2, as a function of the elevation angle in degrees. Zero degrees coincides with the direction of the main beam.

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10. SUPPLEMENTARY NOTES

☐ Document describes a computer program; SF-185, FIPS Software Summary, is attached.

11. ABSTRACT (A 200-word or less factual summary of most significant information. If document includes a significant bibliography or literature survey, mention it here)

The objective of this project was to measure the microwave radiation emitted by speed measuring radar units to obtain a data base for evaluating the potential radiation hazards of these devices. Measurements were taken both in free-space and with the radar units mounted in typical operating positions inside or attached to a four-door sedan. The free-space measurements were made at four different distances to determine the field strength as a function of distance from the radar units. Calibrated radiation level probes were used to measure the field strength inside the automobile and scan the interior volume of the four-door sedan with particular attention to the driver and passenger locations. Twenty-two radar units were involved, and the data are presented in a power density format.

12. KEY WORDS (Six to twelve entries; alphabetical order; capitalize only proper names; and separate key words by semicolons)

Doppler radar; field intensity; hazard; microwave radiation; power density; radar unit; radiation level; speed measuring radar.

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Brain Tumor Mortality Risk Among Men With Electrical and Electronics Jobs: A Case-Control Study [1]

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Elizabeth T. H. Fontham, *Dr.P.H.*, [7] **Margit L. Bleecker**, *M.D., Ph.D.*, [3,8]
Patricia A. Stewart, *M.S.*, [2] *and* **Robert N. Hoover**, *M.D., Sc.D.* [2,9]

ABSTRACT—Brain tumor risk associated with electrical and electronics jobs and with occupational exposure to microwave and radiofrequency (MW/RF) electromagnetic radiation was evaluated with the use of data from a death certificate-based case-control study of brain tumors and occupational risk factors in northern New Jersey, Philadelphia, PA, and southern Louisiana. Next-of-kin of 435 white men who died of a primary brain tumor and of 386 controls who died from other causes were interviewed to obtain information on lifetime occupational history and other factors that might be related to excess brain tumor risk. The relative risk (RR) for all brain tumors was elevated among men exposed to MW/RF radiation [RR=1.6; 95% confidence interval (CI)=1.0, 2.4] and was significantly elevated among men exposed for 20 or more years. All of the excess risk for MW/RF radiation-exposed subjects was derived from jobs that involved the design, manufacture, repair, or installation of electrical or electronic equipment (RR=2.3; 95% CI=1.3, 4.2), while risk of brain tumors among MW/RF radiation-exposed subjects who never worked in electrical or electronics jobs was not elevated (RR=1.0; 95% CI=0.5, 1.9). Furthermore, risk was elevated for electronics workers who were considered to have no exposure to MW/RF radiation. Among electrical and electronics workers, risk was highest for engineers, teachers, technicians, repairers, and assemblers combined (RR=3.9; 95% CI=1.6, 9.9) and was limited to excess risk from astrocytic tumors (RR=4.6; 95% CI=1.9, 12.2). Risk of astrocytic tumors among these electronics manufacture and repair workers increased with duration of exposure to tenfold among those employed for 20 or more years. Among electricians and power and telephone linemen combined (electrical tradesmen), the RR for astrocytic tumors was slightly elevated, but not statistically significant (RR=1.8), and showed no consistent evidence of a duration-response relationship. Electrical tradesmen are exposed to extremely low frequency electromagnetic radiation, while men in some jobs associated with electronics manufacture and repair are exposed to electromagnetic radiation in the very high frequency and ultra-high frequency ranges and also may be exposed to soldering fumes, solvents, and a variety of other chemicals.—JNCI 1987; 79:233-238.

Two recent investigations suggested that individuals occupationally exposed to MW/RF electromagnetic radiation have an elevated risk of brain tumors (*1, 2*). In mortality surveys of occupations listed on death certificates, excess brain tumor deaths were observed among electrical engineers (*1, 3, 4*), electronics technicians (*2*), electricians (*1, 2, 5-7*), power station operators (*2*), and telegraph, telephone, and power linemen and servicemen (*1, 3*). Although these occupations might involve exposure to MW/RF radiation, some of them also might involve contact with lead, fumes from soldering, solvents, and a variety of other chemicals. These reports prompted an analysis of brain tumor mortality risk associated with MW/RF electromagnetic radiation exposure and with jobs involving electronics and electrical work using lifetime work histories obtained from next-of-kin in an investigation conducted in Louisiana, New Jersey, and Pennsylvania to study brain tumors in the petrochemical industry. Other occupational exposures considered in the present analyses were lead and soldering fumes.

SUBJECTS AND METHODS

Study population.—Cases and controls were selected from death certificates of usual residents of northern New Jersey; Philadelphia, PA, and surrounding counties; and the gulf coast of Louisiana. Cases were white men age 30 years or older who died of brain or other central nervous system tumors between January 1, 1979, and December 31, 1981 (January 1, 1978, and June 30, 1980, in Louisiana). Brain tumor diagnoses were verified through a hospital record review, and men whose underlying cause of death had been misclassified were elimi-

ABBREVIATIONS USED: CI=confidence interval; ELF=extremely low frequency; MW/RF=microwave and radiofrequency; RR=relative risk; SIC=Standard Industrial Classification; UHF=ultra-high frequency; VHF=very high frequency.

[1] Received September 9, 1986; revised February 19, 1987; accepted February 27, 1987.

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[9] We thank Ms. Diane Stoner for her excellent work in all aspects of the data-collection phases and Dr. Mustafa Dosemeci for his scientific input.

TABLE 2.—*Brain tumor risk among study subjects whose jobs ever involved exposure to MW/RF radiation by duration exposed*

Exposure and/or occupation	Total duration, in yr of MW/RF radiation exposure[a]			
	Unexposed[b]	<5	5-19	≥20
Ever exposed to MW/RF radiation				
Cases	359	18	18	29
Controls	341	17	8	15
RR[c]	1.0	1.0	2.3	2.0[d,e]
Ever exposed to MW/RF radiation in an electrical or electronics job				
Cases	359	8	11	22
Controls	341	8	3	7
RR[c]	1.0	1.1	3.7	3.1[d,e]
Exposed to MW/RF radiation but never worked in electrical or electronics job				
Cases	359	10	7	7
Controls	341	9	5	8
RR[c]	1.0	1.0	1.5	1.0

[a] "Exposed" subjects with unknown duration employed are excluded from these analyses.
[b] The unexposed referent excludes subjects exposed to MW/RF radiation by method 1.
[c] RR = maximum likelihood estimate of the RR adjusted for educational class.
[d] 95% CI does not include 1.0.
[e] Chi-square test for linear trend statistically significant at the .05 level.

cally significant twofold elevated brain tumor mortality among those exposed to MW/RF radiation for 5 or more years, although there was no consistent duration-response relationship with longer exposure (table 2). Once again, this overall excess was due to a threefold elevated risk among MW/RF radiation-exposed subjects who had an electrical or electronics job for 5 or more years (table 2).

The method 2 classification resulted in a similar finding for MW/RF radiation field exposure (RR=1.7; 95% CI=1.1, 2.7). When men who also had exposure to soldering fumes (primarily in electronics jobs) or lead were removed from the MW/RF radiation-"exposed" group, the RR for brain tumor mortality was 1.4 (95% CI=0.7, 3.1). Removing men who might have had occupational exposure to organic solvents from both the MW/RF radiation-exposed group and the unexposed referent further reduced the risk of brain tumors to 0.4 (2 cases; 5 controls).

Because excess brain tumor risk among MW/RF radiation-exposed men was restricted to those who held electronics and electrical jobs, these occupations were examined in more detail (table 3). There is overlap between the "exposed" subjects in table 3 and those in tables 1 and 2; however, electronics jobs not involving exposures to MW/RF radiation (e.g., assemblers) were also included in table 3. Electronics and electrical jobs were divided into the following two categories based on their potential exposures: 1) jobs associated with the manufacture and repair of electronics equipment, including engineers, teachers, technicians, repairers, and assemblers who could have exposure to VHF and UHF electromagnetic radiation and also to solvents and fumes from soldering (*12*); and 2) electrical trades jobs, including electricians, power linemen and servicemen, and telephone linemen and servicemen who would be exposed to ELF electromagnetic radiation (*12*). A statistically significant threefold excess risk of brain tumors occurred among men who had worked in jobs associated with the manufacture and repair of electronics. Brain tumor mortality risk estimates were elevated for each of the individual jobs included in this category: electrical engineers (cases=3; controls=1; RR=2.2; 95% CI=0.2, 55.8), electronics teachers (cases=4; controls=0), electronics technicians (cases=4; controls=1; RR=4.1; 95% CI=0.4, 96.3), electronics equipment repairers (cases=18; controls=4; RR=4.6; 95% CI=1.4, 16.4), and electronics assemblers (cases=6; controls=1; RR=5.6; 95% CI=0.7, 124.9). Some study subjects were counted more than once when the data were analyzed in this manner

TABLE 3.—*Brain tumor risk among study subjects ever employed in an electronics or electrical job*[a]

Occupation (Census codes)	All brain tumor cases	Exposed controls			Astrocytic tumor cases			Other brain tumor cases		
		No.	RR[b]	95% CI	No.	RR[b]	95% CI	No.	RR[b]	95% CI
Electronics manufacture and repair workers (055, 148, 213, 523-526, 528-533, 683)	28	7	3.9	1.6, 9.9	25	4.6	1.9, 12.2	3	1.4	0.3, 6.4
Electrical tradesmen (527, 575-577)	28	15	1.9	0.9, 3.8	18	1.8	0.8, 3.9	10	2.1	0.8, 5.2

[a] The unexposed referent excludes subjects exposed to MW/RF radiation by methods 1 and 2. Five cases and 2 controls had jobs in both occupation categories.
[b] RR = maximum likelihood estimate of the RR adjusted for educational class.

excess brain tumor risk. Elevated brain tumor mortality appeared to be due primarily to excess astrocytic tumor risk, which increased by duration employed in electronics manufacture and repair jobs. Results of the present study are consistent with those of earlier studies that found excess brain tumor risk among men in electronics jobs (*1-4*). Studies of electronics workers in Sweden did not find elevated risk for brain tumors (*13-15*).

The pattern of excess brain tumor risk among electrical and electronics workers, and not among others exposed to MW/RF radiation, suggests that simple exposure to MW/RF radiation is not the responsible agent. Modifying factors might include the type of MW/RF radiation or the presence of other exposures. Electrical tradesmen are exposed to ELF radiofrequencies associated with electric power lines, while some electronics manufacture and repair workers are exposed to UHF and VHF frequencies (*12*). However, MW/RF radiation exposure in electronics jobs is probably intermittent and may be accompanied by exposures to lead, solder fluxes, solvents, and other chemicals.

Solders used in the electronics industry are usually a combination of lead and tin, but they may also contain cadmium or zinc (*12, 16*). The most common solder flux, the coating around the soldering wire, is pine resin (colophony), which is associated with occupational asthma (*16*). When colophony is heated, by-products include aliphatic aldehydes like formaldehyde (*16*) and many other substances. Fluoride compounds are also used as solder fluxes (*16*). Lead is known to be neurotoxic (*17*), but its role as a carcinogen is questionable. Several laboratory studies have produced renal tumors in rodents exposed to inorganic lead (*18*), and gliomas were induced in rats fed a diet of lead subacetate (*19*). A case report described 2 children who showed clinical symptoms of lead poisoning and subsequently developed astrocytomas (*20*). However, numerous studies have been conducted of workers exposed to lead in battery plants and smelters, and none have indicated an excess risk of brain cancer (*18, 21-25*). Our data showed an elevated brain tumor mortality risk among men occupationally exposed to soldering fumes, but almost all of the "exposed" were electronics workers; thus the effects of soldering fumes could not be evaluated separately from solvents, MW/RF radiation, and other exposures associated with these jobs. There was no increased brain tumor mortality risk among men who presumably had occupational exposure to lead.

Numerous solvents including 1,1,1-trichloroethane (methylchloroform), trichloroethylene, tetrachloroethylene, and methyl ethyl ketone (*16*) used throughout the electrical and electronics industry are known neurotoxins, causing peripheral neuropathy, central nervous system depression, and neurobehavioral dysfunction (*17*). A common acute effect of exposure to halogenated hydrocarbon solvents like methylchloroform, trichloroethylene, and tetrachloroethylene is anesthesia (*17*). Experimental studies have shown that trichloroethylene and tetrachloroethylene cause liver tumors in animals (*17, 26*), suggesting that they could be carcinogenic in humans. There have been insufficient experimental data to evaluate the carcinogenicity of methylchloroform (*26, 27*); however, a recent study reported evidence of astrogliosis in gerbils exposed to methylchloroform by inhalation (*28*).

In summary, our data suggest that certain jobs involving the design, manufacture, installation, or maintenance of electronics or electrical equipment involve exposures that are related to excess risk of astrocytic brain tumors. Because these jobs may involve a wide variety of exposures (*16*), a specific etiologic agent cannot be identified from the present data. Results should be interpreted with some degree of caution, because when risks were calculated for specific occupations and for individual strata by duration employed, numbers in single cells were very small; therefore, the magnitude of some RR estimates may be overstated or understated by these analyses. Despite this limitation, our findings suggest that further investigations of electronics jobs should be conducted, with particular attention to exposures to MW/RF radiation, soldering fumes, and solvents.

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Suppression of T-Lymphocyte Cytotoxicity Following Exposure to Sinusoidally Amplitude-Modulated Fields

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Significant inhibition of allogeneic cytotoxicity of the target cell MPC-11 by the murine cytotoxic T-lymphocyte line CTLL-1 was observed when the 4-h cytotoxicity assay was conducted in the presence of a 450-MHz field sinusoidally amplitude-modulated at 60 Hz. Exposure of the effector cells to the field prior to adding them to the target cells in the cytolytic assay resulted in a similar inhibition, suggesting a direct interaction of the field with the cytolytic T lymphocyte. The inhibition was preferentially expressed during the early allogeneic recognition phase. Field-exposed cytolytic cells recovered their full cytolytic capacity in 12.5 h. A differential susceptibility was observed with modulation frequencies from 0 to 100 Hz. Peak suppression occurred at 60 Hz modulation, with progressively smaller effects at 40, 16, and 3 Hz. The unmodulated carrier wave did not affect the cytotoxicity. Effects with 80- and 100-Hz modulation were smaller than at 60 Hz. These results demonstrate an inhibitory but recoverable effect by certain amplitude modulations of weak nonionizing radiation upon the cell-mediated cytolytic immune response.

Key words: microwaves, amplitude modulation, murine allogeneic cytotoxicity, T lymphocytes

INTRODUCTION

Exposure of laboratory animals to nonionizing radiation can produce both general and specific changes in immune competence. Cases reported involving perturbation of the general hematopoietic system include: decreased total cell volume of bone marrow and spleen in mice [Rotkovska and Vacek, 1975], decreased leukocyte counts in hamsters [Lappenbush et al, 1973], and decreased circulating lymphocytes with concomitant increasing circulating neutrophils in mice [Liburdy, 1977]. Specific changes in immune response observed after exposure of animals for various lengths of time to nonionizing radiation include increased lymphoblastoid transformation of cultured peripheral blood lymphocytes from rabbits [Czerski, 1975], decreased phytohemagglutinin (PHA)-induced mitosis of cultured lymphocytes from hamsters [Lysina, 1965], and enhanced PHA-

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induced mitosis of cultured lymphocytes from rhesus monkeys [Prince et al, 1972]. Other specific immune changes reported include an increase in complement-receptor positive lymphocytes in the spleens of mice [Schlagel et al, 1980], decrease of the B-cell primary immune response to sheep red blood cells in immunized mice [Wiktor-Jedrzejczak et al, 1977], increase in response of cultured lymphocytes of rats to both T- and B-cell mitogens [Smialowicz, 1979], and increased antibody titer to *Streptococcus pneumoniae* in mice [Liddle et al, 1980]. Field strengths were clearly at levels, in some of these studies, that would produce raised temperatures in animal test subjects in the range of 0.5 °C or more, and in others marginal increases may have occurred. Direct exposure of leukocytes in vitro to nonionizing radiation where thermal effects were carefully excluded has also been demonstrated to be capable of altering immune competence, as in the decreased viability of rabbit granulocytes [Szmigielski, 1975], and a reduction in the numbers of granulocyte and macrophage colony-forming units from preparations of mouse bone marrow [Lin et al, 1979].

This report describes a reproducible inhibitory effect of 450-MHz fields, sinusoidally modulated at frequencies from 0 to 100 Hz, on the allogeneic cytotoxicity reaction conducted in vitro by CTLL-1 cytotoxic T lymphocytes. All radiation exposures were in an anechoic horn chamber at an incident field intensity of 1.50 mW/cm^2. The experiments reported here describe: (a) the magnitude of suppression of cytotoxic killing, (b) the cell population affected by the radiation (target cell or T-cytotoxic cell), (c) the phase of the cytotoxic event that is affected (recognition and induction or cytolysis), (d) reversibility of the suppressive effects, and (e) differential sensitivity to amplitude modulation of the carrier wave at frequencies between 0 and 100 Hz.

MATERIALS AND METHODS

Maintenance of the T Lymphocyte Line

The "CTLL-1" T-cytotoxic cell line was obtained from Dr. James Watson (University of Adelaide, Australia), and was characterized by Gillis and Smith [1977]. Log-growth phase CTLL-1 cells were cultured at an initial density of 1×10^4 cells/ml and subcultured when the density reached a maximum of 2×10^5 cells/ml. These cells that require T-cell growth factor [Schreier et al, 1980] were supplemented with new media every 48 h.

Preparation of T-Cell Growth Factor-Containing Medium

Sprague-Dawley rat spleens were disaggregated to a single cell suspension by teasing with sterile forceps, counted, and resuspended in 100-ml volumes in glass culture flasks at a final density of 1×10^8 cells/flask. The culture medium was RPMI 1640 with 10% fetal calf serum, 2 mM glutamine, antibiotics (100 μg/ml streptomycin, 100 U/ml penicillin, and 2.5 μg/ml fungizone), 5×10^{-5} M β-mercaptoethanol, and 5 μg/ml concanavalin A. Following 48 h of incubation at 37 °C the T-cell growth-factor (TCGF) containing supernatants were centrifuged, pooled, and sterile-filtered. Aliquots were frozen at −20 °C until needed. Before use, the TCGF medium was mixed with an equal volume of fresh medium and used to culture the CTLL-1 cell line. This medium was also used in the cytotoxicity assay.

Labeling of Allogeneic Target Cells

The tumor target cells used for the cytolytic assay were a H-2^d B lymphoma MPC-11 [Laskov and Scharff, 1970]. Log-phase target cells at a density of 1×10^7 cells/0.5

ml of RPMI-1640 were incubated with 100 μCi $Na^{51}Cr$ for 40 min at 37 °C. Labeled cells were washed three times in 50-ml volumes of Hank's balanced salt solution, then a fourth time immediately before being dispersed into the assay system. Labeling of target cells routinely produced 3,000–8,000 disintegrations per minute per 5×10^4 cells.

Cytotoxicity Assay

Chromium-labeled MPC-11 target cells were suspended in TCGF media and placed into microtiter wells (Linbro) at a cell density of 5×10^4 cells/well (0.1-ml volumes). CTLL-1 allogeneic cytolytic cells were then added to these wells in 0.1-ml volumes to give a final volume of 0.2 ml/well. Following 4 h of incubation at 37 °C, 0.1 ml was carefully removed from the top of each well and the radioactivity measured with a gamma-counter (Beckman Gamma 4000). Spontaneous release of radioactivity by labeled cells was determined by culturing the cells in media only. Maximal release was determined by analysis of supernatants from freeze-thawed labeled cells. Viabilities of all cultures were 99% or greater both at dispersal of cells into the plates and after a 4-h field exposure. Viabilities have also been determined for longer field exposure, both for 26-h and 40-h exposures, and have been measured at 99 ± 1.096 for control and test cultures (450 MHz modulated at 60 Hz). Specific allogeneic cytoxicity was determined using the following formula:

$$\% \text{ specific cytotoxicity} = \frac{(\text{test counts} - \text{spontaneous counts}) \times 100}{(\text{maximum releasable counts} - \text{spontaneous counts})}$$

Three-times freeze-thawed labeled MPC target cells routinely released 70% of the total counts in each culture. The maximum-releasable counts for each experiment was determined by taking 70% of the total counts in each culture, giving one maximum release value for each experiment. These values were statistically identical with or without field exposure. Microwave-exposed cultures were tested in parallel to identical plates in the absence of microwaves. Control cultures were prepared identically to the field-exposed cultures, and consisted of a separate culture plate with six spontaneous-release wells (target cells only), and six test wells (target cells plus effector cells). The control culture was prepared at the same time as the field-exposed culture, using the same cell population, and was gassed with 5% CO_2 and immediately covered with a pressure-sensitive adherent plastic film. The plate was placed into an incubator monitored at the same temperature as the anechoic exposure system. The field-exposed plate was placed on a plastic stand slanted at a 70° angle facing the field. The control plate was also placed on a stand at a 70° angle.

Anechoic 450-MHz Exposure System

The irradiation facility was constructed as a horn radiator, with a plywood frame lined with copper screening. The four walls and the plywood surface closing the wide end of the horn were lined with radiofrequency absorbing material (Echosorb). The absorber on the endwall had a surface geometry designed to maximize absorption at 450 MHz. The exposure area was located near the broad end of the horn 3 m from the point of excitation. The feed system used a terminated wave guide, and this wave guide could be rotated through 90° to allow either vertical or horizontal polarization of the E-vector. All materials inside the chamber were plywood, plastic, or paper. With an overall length of 4 m, the chamber was approximately 7 wavelengths long and was excited in the TE (transverse electric) mode. The chamber has a forced-air environmental control system

that recirculates heated air and ensures a uniform temperature throughout the chamber. This system was activated 2 h before exposure of cell cultures and maintained a temperature of 35 ± 1 °C at a constant relative humidity of 40% for all experiments.

A volume, in the exposure area near the broad end of the horn, in excess of 1.0 m^3 has an SWR of 1.2:1 or less, when unperturbed by biological preparations. The electrical characteristics of the chamber have been calibrated by H. Bassen (Bureau of Radiological Health, Rockville, MD). The field generating system was comprised of a low-frequency waveform generated with sine, square, and triangular wave outputs over the spectrum from 0.01 Hz to 100 kHz (Wavetek Model 159). The modulating signal was applied through a PIN diode modulator to the output of a 450-MHz phase-locked loop-controlled signal generator (Wavetek Model 3000). This generator drove a broadband linear power amplifier with a maximum power output of 80 W (Ailtech Model 35512). Depth of sinusoidal amplitude modulation was monitored with an oscilloscope and with an inline modulation meter/forward-reflected power meter (Bird Model 5483). Modulation depth was maintained at 75–85 percent. An input power to the chamber of 10 W produced a measured field intensity of 10 mW/cm^2 in the 1 m^3 at the exposure site described above, with less than 5% variation over the entire volume.

Incident field level at the exposure location was measured in two ways: with a commercial field intensity probe (Narda Model 8300); and with an experimental triple dipole probe provided for collaborative studies in tissue dosimetry (H. Bassen, Bureau of Radiological Health). A relationship between the sensitivity of this triple probe and an implantable tissue probe was established by exposing both to the same 450-MHz fields in a Crawford cell [Adey et al, 1981]. However, attempts to use the implantable tissue probe for direct dosimetry measurements in individual culture wells were unsuccessful, due to the small dielectric volume in the well (0.2 ml) with respect to the wave length of the incident field (67 cm). Each well plate has 96 wells uniformly spaced in a 12 × 8 matrix. Spacing between well centers was 9 mm. Each plate was 13 × 9 cm. During exposure, plates were tilted to an angle of 70° above the horizontal, with the wells facing the incident field. As many as three plates were exposed at one time. They were then placed vertically one above the other within the low SWR exposure site described above. Incident field levels were adjusted to 1.5 mW/cm^2 in all experiments. Harmonic and spurious content of the radiofrequency (RF) signal was periodically measured with a spectrum analyzer (Tektronix model 7L13) as a check against partial failure of the power amplifier output transistors, with possible contamination of the output spectrum by these components.

A further control on the cytotoxic sensitivities to modulated microwave fields reported here has been made in a Crawford cell exposure system at 37.0 ± 0.1 °C. With a 60-Hz modulated 450-MHz field (1.5 mW/cm^2 incident power), observed inhibition of cytotoxicity in four separate experiments was at the same 20% level noted in the horn chamber data reported here. Controls were placed adjacent to the Crawford cell in the same large incubator that housed it.

Pre- and postexposure temperature measurements were made in the horn exposure chamber, in wells on tissue plates in the exposure chamber, and in the control incubator. A digital thermistor thermometer was used with a resolution of 0.1 °C. The exposed thermistor tip diameter was 1.0 mm and its plastic sheath diameter was 3.5 mm. Well temperatures did not differ significantly from the air temperature of 35 °C in the exposure chamber at commencement and termination of field exposure, nor from incubator air temperatures at those same times for control preparations. Chamber temperature was

35 ± 1 °C. No significant increase in exposure chamber temperature occurred over the 4-h exposure epoch.

Statistical Analysis

The statistical significance of percent reductions in cytotoxicity were determined for each individual experiment, as indicated by the P values in Tables 1–5. The P values were determined using Student's t-test. Arcsin transformations of the six replicate decimal numbers from which the mean percent test cytotoxicity was determined were compared using Student's t-test to the arcsin transformations of the six replicate decimal numbers from which the mean percent control cytotoxicity was determined. Each decimal number was obtained by subtracting the appropriate spontaneous release mean from the individual counts per minute released in the assay and dividing that by the mean of the maximum releasable counts from which the spontaneous mean had been subtracted.

RESULTS

Exposure to the 450-MHz field sinusoidally modulated at 60 Hz during the 4-h allogeneic cytotoxicity assay resulted in a 20% inhibition of the observed cytotoxicity (Table 1). Similar suppression was observed in experiments in which the effector cells were exposed to microwave radiation for a 4-h period before the target cells were added for the 4-h cytotoxicity assay which was conducted in the absence of the field (Table 2). Since the target myeloma cells (MPC-11) were never field-exposed in the latter experiment, observed cytotoxicity changes are attributed to field actions on the effector T lymphocytes.

To evaluate the time-course of the cytotoxic suppression over the 4 h of field exposure, the cytotoxic cells were exposed to microwaves for the first 2 h only, for the last 2 h only, and for the entire 4 h of the cytotoxicity test (Table 3). Exposure to the field during only the first 2 h of the assay resulted in inhibition at levels similar to those noted from a full 4-h exposure to the field. In contrast, inhibition produced by exposure confined to the last 2 h of the assay produced only partial inhibition when compared with that elicited by the continuous 4-h exposure. This suggests a preferential effect upon the recognition phase of cytotoxicity.

TABLE 1. Inhibition of CTLL-1 H-2^d-Directed Cytotoxicity by Exposure During the 4-h Assay to a 450-MHz Field at 1.50 mW/cm^2, Sinusoidally Amplitude-Modulated at 60 Hz*

Experiment		Percent specific cytotoxicity	CPM during cytotoxicity	Spontaneous CPM	Maximum releasable counts	Percent inhibition of cytotoxicity	P value
1	Field	36	2,550 ± 115	1,309 ± 76	4,759 ± 101	20	<.05
	Control	45	2,903 ± 356	1,411 ± 222			
2	Field	58	4,959 ± 718	2,511 ± 796	6,759 ± 247	20	<.05
	Control	72	5,582 ± 278	2,580 ± 801			
3	Field	76	2,870 ± 368	861 ± 64	4,137 ± 216	24	<.05
	Control	81	3,500 ± 595	786 ± 16			
4	Field	25	1,839 ± 15	808 ± 31	5,009 ± 190	17	<.0001
	Control	30	2,125 ± 36	893 ± 58			
5	Field	30	885 ± 22	355 ± 24	2,121 ± 105	17	<.0005
	Control	36	1,015 ± 39	383 ± 18			

*These data represent five experiments, each with six replicates.

TABLE 2. Inhibition of CTLL-1 H-2^d-Directed Cytotoxicity by Exposure of the T Lymphocytes to a 450-MHz Field Modulated at 60 Hz at 1.50 mW/cm^2, for 4 h Prior to Cytotoxicity Assay*

Experiment		Percent specific cytotoxicity	CPM during cytotoxicity assay	Spontaneous CPM	Maximum releasable counts	Percent inhibition of cytotoxicity	P value
1	Field	19	2,410 ± 173	—[a]	7,587 ± 460	24	<.025
	Control	25	2,810 ± 307	1,223 ± 47			
2	Field	53	1,521 ± 145	—[a]	2,270 ± 51	15	<.025
	Control	62	1,663 ± 45	670 ± 36			
3	Field	47	3,524 ± 542	—[a]	6,141 ± 350	15	<.025
	Control	55	3,945 ± 350	1,232 ± 90			
4	Field	35	1,502 ± 84	—[a]	3,172 ± 306	17	<.025
	Control	42	1,676 ± 135	610 ± 56			
5	Field	12	1,334 ± 58	—[a]	5,226 ± 147	25	<.01
	Control	16	1,542 ± 104	857 ± 21			

*These data represent five experiments, each with six replicates.
[a]This table describes the effect on cytotoxicity when the T lymphocytes are irradiated before the cytotoxicity assay is begun. Target cells were added to wells containing CTLL-1 cells after the CTLL-1 had been exposed to the field.

TABLE 3. Inhibition of CTLL-1 H-2^d-Directed Cytotoxicity by Exposure to a 450-MHz Field, 1.50 mW/cm^2, Modulated at 60 Hz, During the Entire Assay, First 2 h, or Last 2 h of the 4-h Assay*

Field on during cytotoxicity test	Percent specific cytotoxicity	CPM during cytotoxicity	Spontaneous CPM	Percent inhibition of cytotoxicity	P value
Experiment 1					
All 4 h	58	4,959 ± 718	2,511 ± 796	19	<.05
First half	60	4,861 ± 263	1,992 ± 443	17	<.005
Last half	66	5,288 ± 207	2,449 ± 580	8	<.05
Control	72	5,582 ± 287	2,580 ± 80	—	—
Experiment 2					
All 4 h	30	885 ± 22	355 ± 24	17	<.0005
First half	32	977 ± 26	441 ± 61	11	<.01
Last half	36	990 ± 19	364 ± 16	0	>.25
Control	36	1,015 ± 39	383 ± 18	—	—

*These data are from two experiments, each with six replicates. (Maximum release counts: experiment 1, 759 ± 247; experiment 2, 121 ± 105.)

The effects of prior exposure of the cytotoxic T lymphocytes to the electromagnetic field at varying intervals prior to the cytotoxicity assay were also investigated. Field exposure was delivered 1, 4, 9, and 12.5 hours prior to the 4-h cytotoxicity assay. A 20% inhibition of cytotoxicity was observed in the assay 1 h post-field exposure (Fig. 1, Table 4). The inhibition of cytotoxicity had decreased to 13% and 12% at 4 h and 9 h post-field exposure, respectively. Cytotoxic inhibition was no longer observed after 12.5 h post-field exposure. As in the experiments in Table 2, target myeloma cells were not

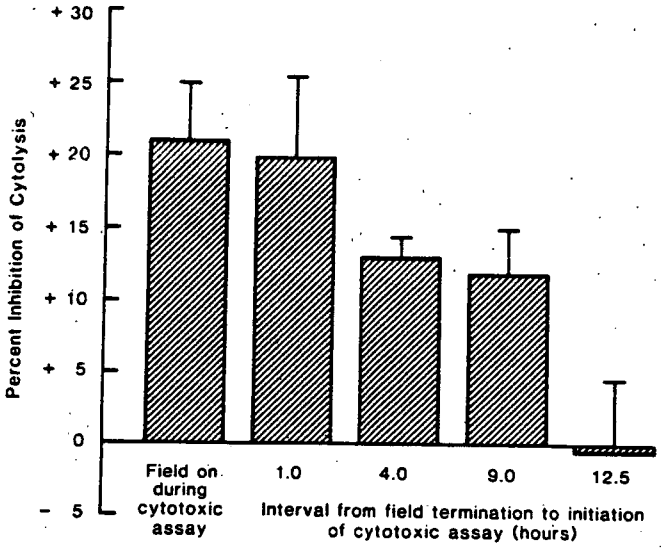

Fig. 1. Recovery of CTLL-1 H-2^d-directed cytotoxicity inhibited by a 4-h exposure of the T lymphocytes to a 450-MHz field, 1.50 mW/cm^2, 60-Hz modulation. Two experiments are averaged here. Final values for each experiment are the average of six replicate cultures exposed in parallel.

field-exposed, and altered cytotoxicity is attributed to modified effector action by field-exposed T lymphocytes. The cultures were supplemented with fresh medium every 2 h by replacement of 0.1 ml of liquid in each well. Cell viabilities at the end of the assay were the same for both control and test wells (99%).

The possibility of differential effects with amplitude modulation frequencies from 0 to 100 Hz was then examined (Fig. 2; Tables 5 and 6). The unmodulated carrier wave did not influence cytotoxicity, and 3-Hz modulation produced only an insignificant suppression. Differential suppression at higher frequencies was seen, with 16, 40, 80, and 100 Hz all producing suppression (9%, 11%, 10%, and 13% respectively), but at lower levels than that seen at 60 Hz (20% suppression).

DISCUSSION

A significant reproducible inhibition (20%) of allogeneic cytotoxicity of the TCGF-maintained murine lymphocyte CTLL-1 was observed when the 4-h cytotoxicity assay was conducted in the presence of a 450-MHz field sinusoidally amplitude-modulated at 60 Hz. This inhibition decreased sharply at higher and lower frequencies, with less suppression (9%, 11%, 10%, and 13% respectively) observed at 16, 40, 80, and 100 Hz. Reduction of allogeneic cytotoxicity was not seen at 3 Hz or in the presence of an unmodulated field. The suppression observed at 60 Hz was transient in nature. Complete recovery was observed 12.5 h following exposure to the microwave field.

The modulated field could inhibit T-cell mediated cytotoxicity by at least two potential mechanisms: (a) interaction with glycoprotein target cell receptor molecules in the membrane of the cytotoxic T lymphocytes; and (b) modulation of critical calcium ion flux. Field interactions with cell membrane surface glycoproteins may in itself involve

TABLE 4. Recovery of CTLL-1 H-2^d-Directed Cytotoxicity Inhibited by a 4-h Exposure of the T Lymphocytes to a 450-MHz Field, 1.50 mW/cm^2, 60-Hz Modulation

Interval from field termination to assay		Percent specific cytotoxicity	CPM during cytotoxicity	Spontaneous CPM	Maximum releasable counts	Percent inhibition of cytotoxicity	P value
Experiment 1							
During assay	Field-exposed	76	2,870 ± 368	861 ± 64	4,137 ± 216	24	<.05
	Control	81	3,500 ± 595	786 ± 16			
1 h	Field-exposed	35	1,502 ± 84	—[a]	3,172 ± 306	17	<.025
	Control	42	1,676 ± 135	610 ± 56			
4 h	Field-exposed	37	1,929 ± 150	—[a]	3,584 ± 69	14	<.025
	Control	43	2,143 ± 183	611 ± 48			
9 h	Field-exposed	36	1,816 ± 219	—[a]	3,980 ± 126	14	<.05
	Control	42	2,015 ± 111	597 ± 59			
12.5 h	Field-exposed	34	1,848 ± 124	—[a]	4,419 ± 102	−4	>.25
	Control	32	1,800 ± 337	513 ± 27			
Experiment 2							
During assay	Field-exposed	25	1,839 ± 15	808 ± 31	5,009 ± 190	17	<.0001
	Control	30	2,125 ± 36	893 ± 58			
1 h	Field-exposed	12	1,376 ± 58	—[a]	5,226 ± 147	25	<.01
	Control	16	1,542 ± 104	851 ± 21			
4 h	Field-exposed	15	1,286 ± 64	—[a]	4,199 ± 78	12	<.10
	Control	17	1,360 ± 94	787 ± 49			
9 h	Field-exposed	19	1,271 ± 43	—[a]	3,826 ± 132	10	<.10
	Control	21	1,340 ± 92	673 ± 9			
12.5 h	Field-exposed	32	1,282 ± 55	—[a]	2,699 ± 79	3	>.10
	Control	33	1,299 ± 32	608 ± 31			

[a]This table describes the recovery of cytotoxicity to control levels as measured by cytotoxicity assays initiated at different times after the CTLL-1 cells were exposed to the field.

modified calcium binding. It has been shown that weak electromagnetic fields can influence the aggregation of acetylcholine receptors on embryonic muscle fibers [Orida and Poo, 1978], and the aggregation of concanavalin A receptors on lymphocytes [Poo and Robinson, 1977]. Evidence has also been accumulated using a clonal line of osteoblast cells, as well as cultured bone preparations, that weak electromagnetic fields can alter the rate of formation of adenylcyclase in response to parathyroid hormone [Luben et al, 1980; Norton et al, 1980]. Adherence to the effector cytotoxic T lymphocyte via specific membrane receptor molecules to the target cell has been shown to be a necessary prerequisite to lysis of the target cell [Abler et al, 1970]. Furthermore, the target cell receptor has been implicated in directing or triggering the T cell cytotoxicity mechanism [Kuppers and Henney, 1976; Fishelson and Berke, 1978].

Although the mechanism of T-cell mediated lympholysis has not yet been agreed upon, considerable evidence supports the delivery of lytic molecules of lymphotoxin to the adhered target cell membrane as directed by specific target cell receptor molecules [Ware and Granger, 1981]. According to Ware and Granger's model of T-cell mediated lympholysis, lymphotoxin precursor subcomponents form part of a macromolecular complex upon the surface of the cell membrane. The complex is thought to have three essential

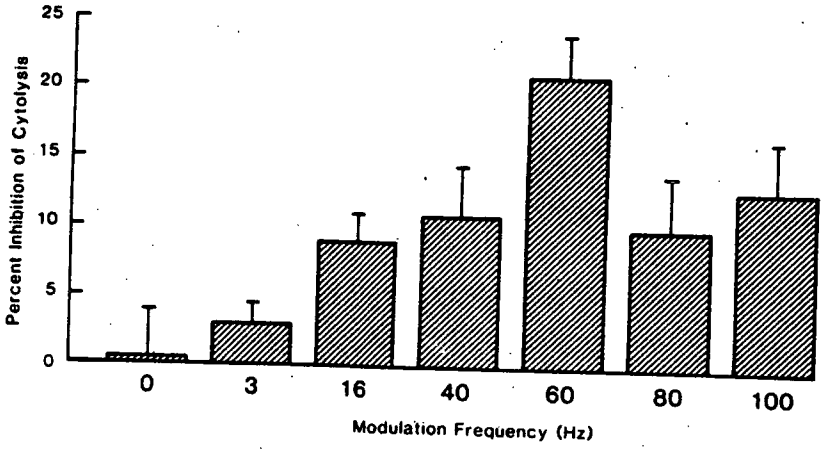

Fig. 2. Inhibition of CTLL-1 H-2^d-directed cytotoxicity by exposure to a 450-MHz field, 1.50 mW/cm^2, sinusoidally amplitude-modulated between 0 and 100 Hz. The inhibitions represented at 0, 3, 16, 40, and 80 Hz are the average of two experiments each. The inhibition at 100 Hz is the average of three experiments. The inhibition at 60 Hz is from the same five experiments of Table 1. Final values for each experiment are the average of six replicate cultures.

TABLE 5. Inhibition of H-2^d-Directed Cytotoxicity by Exposure to a 450-MHz Field, 1.50 mW/cm^2, Sinusoidally Amplitude-Modulated at 0, 3, and 16 Hz

Modulation frequency	Experiment		Percent specific cytotoxicity	CPM during cytotoxicity	Spontaneous CPM	Maximum releasable counts	Percent inhibition of cytotoxicity	P value
0	1	Field	50	3,355 ± 321	1,378 ± 124	5,299 ± 144	4	<.25
		Control	52	3,308 ± 138	1,160 ± 53			
	2	Field	34	2,038 ± 31	914 ± 80	4,270 ± 132	−3	>.25
		Control	33	1,998 ± 104	892 ± 84			
3 Hz	1	Field	73	2,269 ± 66	846 ± 18	2,787 ± 73	4	<.25
		Control	76	2,323 ± 102	884 ± 47			
	2	Field	49	1,521 ± 76	432 ± 44	2,644 ± 154	2	<.25
		Control	50	1,519 ± 163	380 ± 17			
16 Hz	1	Field	36	3,576 ± 194	1,079 ± 77	8,017 ± 450	10	<.01
		Control	40	3,875 ± 145	1,121 ± 77			
	2	Field	54	1,669 ± 58	432 ± 20	2,711 ± 86	8	<.10
		Control	59	1,784 ± 105	391 ± 24			

subunits: (a) the antigen-specific receptor, (b) a catalytic unit composed of inactive lymphotoxin subcomponents, and (c) an activation unit. Upon binding to antigen or lectin, two signals would occur: one to instruct the cytotoxic T lymphocyte to redistribute additional complexes to the area of effector-target cell membrane contact, and the other signal prompting the activation and delivery of the lymphotoxin unit to the target membrane surface. Following this "programming-for-lysis," the presence of the effector lym-

TABLE 6. Inhibition of CTLL-1 H-2^d-Directed Cytotoxicity by Exposure to a 450-MHz Field, 1.50 mW/cm^2, Sinusoidally Amplitude-Modulated at 40, 80, and 100 Hz

Modulation frequency	Experiment		Percent specific cytotoxicity	CPM during cytotoxicity	Spontaneous CPM	Maximum releasable counts	Percent inhibition of cytotoxicity	P value
40 Hz	1	Field	61	965 ± 50	231 ± 16	1,436 ± 41	9	<.025
		Control	67	1,045 ± 36	258 ± 14			
	2	Field	40	984 ± 73	306 ± 16	1,995 ± 40	13	<.05
		Control	46	1,069 ± 57	284 ± 22			
80 Hz	1	Field	22	1,656 ± 72	793 ± 17	4,768 ± 301	12	<.005
		Control	25	1,866 ± 51	907 ± 27			
	2	Field	47	1,344 ± 71	332 ± 19	2,471 ± 86	8	<.05
		Control	51	1,416 ± 49	328 ± 19			
100 Hz	1	Field	19	2,648 ± 74	1,201 ± 63	8,850 ± 259	9	<.10
		Control	21	2,953 ± 216	1,415 ± 62			
	2	Field	43	1,348 ± 52	378 ± 20	2,661 ± 116	14	<.0025
		Control	50	1,493 ± 80	338 ± 26			
	3	Field	61	1,062 ± 22	247 ± 32	1,590 ± 31	15	<.001
		Control	72	1,208 ± 66	245 ± 21			

phocyte is no longer necessary for subsequent lysis of the target cell, which may take up to several hours to occur [Martz and Benacerraf, 1973].

We observed that the murine allogeneic cytotoxicity reaction is preferentially inhibited by field exposure during the initial phase of the assay (first 2 h), in which programming-for-lysis occurs. The modulated field could be affecting any or all of the postulated functions of the target cell receptor which are crucial to the cytolytic mechanism. These sites for field-perturbation are: (a) interaction of the receptor with specific target cell antigen, (b) receptor-directed redistribution of additional macromolecular complexes to the area of lymphocyte-target cell membrane contact, or (c) receptor-mediated delivery of activated lymphotoxin units to the target cell membrane.

Another early requirement for lysis of the target cell is the presence of calcium [Goldstein, 1975]. The programming-for-lysis stage will not occur if Ca^{2+} is absent from the culture medium [Gately and Martz, 1979]. It has been established that calcium efflux from nervous tissue can be modulated by weak electromagnetic fields [Bawin et al, 1978]. Furthermore, calcium efflux from chick brain tissue was shown to be sensitive to amplitude modulation frequency, with a peak effect occurring at 16 Hz [Bawin et al, 1975; Blackman et al, 1979]. Exposure of chick cerebral hemispheres to 450-MHz fields amplitude-modulated at 16 Hz resulted in an increased calcium efflux of 10% or more at field intensity of 0.1–1.0 mW/cm^2. A similar effect, but with different field parameters, might be occurring during the calcium-dependent programming-for-lysis stage of T-cell mediated lympholysis.

Field-perturbation of critical calcium ion flux or membrane receptor function are thus possible explanations for the suppressive effect of weak nonionizing radiation on cell-mediated cytotoxicity described here. These observations add new evidence that the

functional capacity of the T lymphocyte in cell-mediated immune response is sensitive to weak microwave fields amplitude-modulated at low frequency, and that there is a differential sensitivity to the modulation frequency, with maximum effect at 60 Hz.

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evolved from my clinical research (4). In a group of scientists and technicians working with prototype masers and lasers, abiotic melanomata began to appear in the eyes of a small, but significant, number of individuals. The effects were not associated with injuries from direct ocular exposure, but appeared to be the result of repeated ambient exposures to coherent irradiations at specific nonionizing radiation wavelengths from maser/laser systems. These findings suggested that the biological reaction was mediated via alteration of wavelength-specific sites in receptor molecules such as, for example, rearrangement of chemical bonds resonating at the same frequency as the incident irradiation. By instituting protective measures, the abnormal trend in abiotic effects was apparently reversed for many of the individuals under my surveillance.

Regardless of the characteristic frequency of the incident energy, irradiation of tissue results in a change in the energy state of many of its atomic and molecular components. For example, electrons can be pumped to higher energy levels. The return of the molecules to their normal energy levels is accompanied by secondary re-radiation, some of which may occur at radiofrequencies characteristic of specific atomic elements or chemically combined components of the molecules. Thus, from such re-radiated secondary frequencies, or from a primary radiofrequency irradiation itself, as from an external source, if such frequencies are in resonance with an inherent frequency of a chemical bond in a genetic material molecule, then, by resonance interference or augmentation, some alteration of chemical bonds can occur in the genetic material.

Should the affected genes be so altered and then replicated as modified recombinant DNA or RNA, the resultant altered molecule may affect the action of proto-oncogene and thus serve as the mechanism for the cloning of mutated cells. Should proto-organogene be so affected, this could result in teratism. Should the effect be limited to genetic encoding sites, other physiological dysfunction, including tumor formation, could ensue.

CONCLUSIONS

The role of the resonance-frequency hypothesis in explaining how radiofrequency irradiation produces genetic alterations is presently speculative, but it seems worthy of further investigation. Far more imperative, however, is the immediate need to apply appropriate diagnostic procedures to individuals exposed to radiofrequency energy, and to provide the best protection possible for the population groups at risk.

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patient had a long history of systemic occupational exposure to electromagnetic energy. The associated pathology observed in this case, an insulinoma, has previously been seen in occupationally radiation-exposed individuals. No other factor associated with pancreatic cancer was present in the case. My clinical conclusion, therefore, was that the pancreatic tumor and the cataract in the patient's left eye were both caused and/or aggravated by his occupational exposure to nonionizing radiation.

Differential diagnosis depends upon the presence or absence of common factors. Above, we considered three cases of pancreatic tumor (one exocrine and two endocrine) which, in common, occurred in radio-frequency technicians. That is strongly suggestive that chronic radiation exposure could have been a causal or contributory etiological factor.

However, when one considers that, in the two cases of insulinoma discussed above, both of them exhibited, in addition, ocular evidence of radio-frequency injury, then the connection between insulinoma and exposure to injurious levels of radio-frequency irradiation becomes compelling. The rationale is straightforward because insulinoma is so rare that there is no meaningful statistic for its prevalence so that most physicians have never encountered a solitary case throughout their entire professional careers. Thus, one clinician finding two cases of insulinoma in his own practice is noteworthy. The fact that both cases occurred in radio-frequency technicians indicates the possibility that the condition was caused, at least in part, by the occupational environment. When it is noted further that both cases also had evidence of having been exposed to levels of irradiation capable of producing pathological tissue change, namely radio-frequency cataractogenesis, then that corroborating feature of the differential diagnosis establishes radio-frequency radiational injury as the probable cause for the insulinoma.

Although small in number, these two cases nevertheless represent a cluster. And, because insulinoma is such a rare medical curiosity, the finding of two cases cannot be explained away due to chance, especially in

such a relatively small population group as the approximately 500 cases of nonionizing radiation cataract in my collection.

I have found at least one other case of insulinoma in my collection. This patient, a white male about 30 years old when I examined him in 1960, exhibited radio-frequency cataractogenesis. In view of the unusual aspects of his case (it was amongst the first cataract cases that I found in a radio-frequency technician), I decided to re-examine him after a short period of time, three months instead of the ordinary 12 month interval. However, that was not possible because he died during the interim with a presumptive diagnosis of insulinoma.

These three cases of insulinoma and radio-frequency cataract do not prove a cause-and-effect relationship involving radiofrequency exposure with scientific certitude. They do represent a cause-and-effect relationship with exposure in terms of medical probability. The two levels of certainty signify different aspects and stages of science. As such, they should stimulate the search for a better understanding of how the different sciences may be correlated.

THE RESONANCE FREQUENCY HYPOTHESIS

By what mechanism can radiofrequency energy induce genetic damage? Slight chemical aberrations, even involving as few as two or three atoms, when located at critical loci within organic macromolecules may result in altered biological effects. Should such biochemical transformations occur in genetic material such as proto-organogenes, proto-oncogenes or encoded genetic compounds acting as master switches, altered chemical activity may occur, resulting in major aberrations such as tumor (insulinoma), teratism (Down's symdrome) and cancer (leukemia), all of which have been reported in association with chronic exposure to radiofrequency energy.

The concept of resonance-frequency interference altering normal biochemical action, as mediated via nonionizing electromagnetic radiation,

Under what conditions can human pathology -- other than the signature disease of capsular cataract -- be justifiably attributed to radiofrequency exposure? The question is of increasing importance because of the proliferation of radiofrequency-emitting devices, and the apparent increase in the incidence of disease with which they are associated. In attempting to resolve that question the epidemiologist is concerned with disease correlations exhibited by groups of individuals, and the basic scientist studies cause-and-effect relationships in animal populations that are under his complete control.

In contrast, the clinician deals with individual human beings, and he must make rational decisions about many aspects of the course of treatment, as well as about the etiology of the disease presented by specific patients. I have monitored a large number of radiofrequency scientists and technicians over long periods, up to 25 years in duration for some patients. Based on this experience, I have evolved a set of restrictive differential diagnostic criteria in an attempt to formulate a rational methodology for clinical research involving the relationship between exposure to radiofrequency energy and pathological effects, including mutagenic effects, in humans. These criteria are described below, and their application is illustrated in the case report of a pancreatic tumor.

DIFFERENTIAL DIAGNOSTIC CRITERIA

Consider a clinical condition that satisfies the following four criteria: (1) the patient exhibits a signature radiation cataract; (2) the radiofrequency exposure consisted of partial-body or total-body irradiation, and was not limited to the eye itself; (3) the additional pathology exhibited has previously been reported in association with radiofrequency-energy exposure; (4) no other known or suspected factor capable of causing or facilitating the observed pathology is found in association with this specific patient.

I submit that when these criteria are satisfied it is justifiable to conclude, within the bounds of medical reasonableness, that the observed pathology was caused or aggravated by the exposure to the radiofrequency energy. Thus, the operant test for humans is not scientific certitude but, instead, it is clinical credibility.

APPLICATION OF DIFFERENTIAL DIAGNOSTIC CRITERIA

In 1975, two radiofrequency-equipment repairmen (ages 31 and 34), who were coworkers for approximately 10 years, both contracted pancreatic tumors. One who had an exocrine pancreatic cancer died from metastatic disease shortly after his illness was discovered and before I could examine him; the other underwent apparently successful surgical extirpation of the tumor, which was a rare form of the disease (insulinoma). However, I had the opportunity to examine the second patient before his surgery, and found that he exhibited an early stage of radiation cataracts. In this context, consider the following case report which is about a different additional patient, the third case in my recent personal experience of pancreatic tumor in a radiofrequency repairman.

A Case Report

The patient was a well-developed, well nourished Hispanic male who, at age 63, expired on March 2, 1983. His death resulted from post-operative complications of unsuccessful therapy for an insulinoma. From 1939 to 1982, the patient worked as an electronics technician, primarily as a radiofrequency-equipment repairman. In 1974-1982, he repaired equipment that operated at 115 MHz and 470 MHz, and he was repeatedly irradiated in at least three ways. He received generalized whole-body exposure due to the fluctuating ambient levels in his occupational environment. Additionally, this left-handed patient used a walkie-talkie in its normal communication modality, with the antenna close to his left eye, and during transmission, that eye was directly irradiated. Finally, the patient frequently used a belt-mounted antenna, and it resulted in direct radiation of his abdomen.

The two important medical findings were signature radiofrequency cataract of the left eye, and insulinoma.

The patient exhibited the signature radiation cataract, which is indicative of a history of chronic exposure to electromagnetic energy. The

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RADIOFREQUENCY IRRADIATION AS A FACTOR IN HUMAN TUMORS, TERATISM AND CANCER: THE RESONANCE FREQUENCY HYPOTHESIS

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ABSTRACT

A rationalized methodology for clinical research is described that permits investigation of the relationship between nonionizing radiation exposure and mutagenic effects in humans. A hypothesis is proposed that explains a possible mechanism whereby radiofrequency irradiation can directly alter the chemical activity of genetic molecules -- the resonance frequency hypothesis.

INTRODUCTION

The fact that acquired capsular cataract could serve as a signature of injury due to exposure to radiofrequency energy was first described by me in 19[illegible] (1). This finding was subsequently verified independently (2,3), thereby confirming that radiofrequency lens injury was indeed a nosological entity.

In 1974 I presented a series of select case reports of radiofrequency lens injuries in which each case exhibited an additional pathology. At that time it was not possible nor intended to establish a strict cause-and-effect relationship between the radiofrequency exposure and the observed additional pathology. Indeed, this etiologic connection was strongly suggested in the group of otherwise healthy individuals who acquired capsular cataract in one eye following repeated or chronic radiofrequency irradiation. That report raised an interesting question.

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Alterations in Protein Kinase Activity Following Exposure of Cultured Human Lymphocytes to Modulated Microwave Fields

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Cultures of human tonsil lymphocytes were exposed in a Crawford cell to a 450-MHz field (peak envelope intensity 1.0 mW/cm^2), sinusoidally amplitude modulated (depth 80%) at frequencies between 3 and 100 Hz for periods up to 60 min. The Crawford cell was housed in a temperature-controlled chamber (35 °C) and control cultures were placed in the same chamber. Activity of cAMP-dependent protein kinase relative to controls remained unaltered by fields modulated at 16 or 60 Hz with exposures of 15, 30, and 60 min. By contrast, total non-cAMP-dependent kinase activity fell to less than 50% of unexposed control levels after 15 and 30 min exposures, but, despite continuing field exposure, returned to control or preexposure levels by 45 and 60 min. A smaller reduction (20–25%) also occurred with 60-Hz modulation and was also restricted to exposure durations of 15 and 30 min. CW 450-MHz fields were without effect. Reduced enzyme activity occurred with 16-, 40-, and 60-Hz modulation frequencies, but not with 3-, 6-, 80-, or 100-Hz modulation. The specific identity of this kinase is unknown. This rapid but transient reduction in lymphocyte protein kinase activity restricted to modulation frequencies between 16 and 60 Hz and to less than 30 min exposure is consistent with "windowing" with respect to modulation frequency and exposure duration.

Key words: microwaves, amplitude modulation, human lymphocytes, protein kinase

INTRODUCTION

During the past ten years, a considerable number of reports have dealt with effects of nonionizing electromagnetic fields on the immune system. Many of these studies have investigated alterations in immune cell functions following exposure of animals or lymphoid cells in vitro to microwave fields of sufficient intensity to increase body or cellular temperatures [Smialowicz, 1976, 1979; Sultan et al, 1983; Szmigielski et al, 1978; Yang et al, 1983; Rama Rao et al, 1983]. By contrast other studies have found changes in a variety of parameters related to immune function following exposure to microwave and other radiofrequency fields at "nonthermal" intensities [Baranski, 1971,1972; Czerski, 1975; Deichman et al, 1964; Djordjevic

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and Kolak, 1973; Huang, 1980; Lyle et al, 1983; Mayers and Habeshaw, 1973; Stodolnik-Baranska, 1974a,b]. Also, in some studies no measurable effects of low energy fields were observed in general hematopoietic functions [Smialowicz, 1976; Yang et al, 1983; Roberts et al, 1983]. Our studies have been concerned with these nonthermal effects of modulated microwave fields of low energy on cultured lymphocytes.

We have measured the activity of protein kinases in cultured lymphocytes exposed to modulated microwave fields. Protein kinases are enzymes with the general property of phosphorylating other enzymes or proteins. This covalent modification, ie, the addition of a phosphate group to a serine, threonine, or tyrosine residue of the protein, alters the function of the protein phosphorylated. If the phosphorylated protein is an enzyme, for example, activity of the enzyme is modulated following phosphorylation. Protein kinases have been implicated in regulation of many intracellular processes, including glucose and lipid metabolism, protein synthesis, membrane permeability, enzyme induction, viral transformation, and many other functions [see Rosen and Krebs, 1981].

In terms of immune system function, we have reported previously that the activation of a specific protein kinase, cyclic AMP-dependent protein kinase, may be important in mediating action of mitogens on cultured human peripheral blood lymphocytes [Byus et al, 1977, 1978; Klimpel et al, 1979]. The activation of cAMP-dependent protein kinase occurred within an hour of the addition of various mitogens to the cultured lymphocytes, well before any increase in DNA synthesis. For this reason, we examined effects of modulated microwave fields of low energy upon activity of protein kinase(s) in tonsil lymphocytes under conditions in which the temperature elevation at the end of field exposure was less than 0.1 °C.

We observed a marked reduction in tonsil lymphocyte protein kinase activity within 15–30 min of onset of field exposure. The decrease in kinase activity was transient in that after a 60-min period in the field activity had returned to control (nonfield) levels. The reduced enzyme activity resulting from field exposure was dependent on the frequency at which the field was sinusoidally amplitude modulated.

The protein kinase that was affected by the field proved not to be cAMP-dependent protein kinase and as yet remains to be fully characterized. The possible identity of the field-sensitive protein kinase as well as the significance of both the frequency and time dependence of these effects are discussed. It is emphasized that agents such as mitogens and hormones commonly used to alter protein kinase activity were not used in these experiments, except for those serum factors present in the lymphocyte culture medium.

MATERIALS AND METHODS

Lymphocyte Culture

Human tonsils were placed on ice in sterile culture medium (see below) immediately following removal and were routinely placed in culture 3–4 h postoperatively. The tonsils were rinsed in Roswell Park Memorial Institute (RPMI) medium containing 10% fetal bovine serum (FBS), and 100 U/ml penicillin and 100 μg/ml streptomycin and finely minced with a scalpel under sterile conditions. The dispersed tonsil was suspended in RPMI + 10% FBS, pipetted repeatedly through a 25-ml pipette; and centrifuged at approximately 100 × g to form a pellet of lymphocytes. The

pelleted cells were washed twice in the same culture medium and resuspended in medium at a density of 5×10^6 cells/ml. Lymphocyte cultures used for all experiments always contained greater than 77% viable cells as determined by trypan-blue exclusion. Viability did not change following a 12-h culture period or following exposure to any of the fields. Two milliliters of medium containing the lymphocytes (10^7 cells) were placed in sterile 15-ml plastic culture tubes and stored in a CO_2 incubator at 35 °C for 1–3 h until exposure to the field (see below). These are standard procedures for culturing tonsil lymphocytes and yield a human lymphocyte population that is approximately 50% T cells and 50% B cells.

Field Exposure

The Crawford cell exposure system was operated in the same general configuration as in previous studies [Lyle et al, 1983]. The cell (Instruments for Industry Model 3000) was designed to operate as a coaxial transmission line with a characteristic impedance of 50 ohms over the spectrum DC to 520 MHz. With the biological test specimens in place and a 50-ohm noninductive termination, the standing-wave-ratio (SWR) did not exceed 1.17:1 at the operating frequency of 450 MHz. Measurements with a field intensity probe (Narda Model 8300) and with an experimental triple dipole probe reported elsewhere [Adey et al, 1981; Lawrence and Adey, 1982] showed reasonable isotropy of electric field distribution, except near the side walls of the cell (where the field was lower than in the central zone where the cell cultures were always placed). The viability of the cultures was not altered by exposure to any of the fields used in the sudies presented here.

The field generating system comprised a low-frequency waveform generator (Wavetek Model 159) as a modulating signal source. This signal was applied through a PIN diode modulator to the output of a 450 MHz phase-locked-loop-controlled signal generator (Wavetek Model 3000). This generator drove a broadband linear power amplifier with a maximum power output of 20 W (Ailtech Model 35512). Depth of sinusoidal amplitude modulation was monitored with an oscilloscope and with an in-line modulation meter/forward-reflected power meter (Bird Model 5483). Modulation depth was maintained at 75–85%. An input of 1.7-W peak envelope power (PEP) to the cell produced a peak field intensity of 1.0 mW/cm^2. PEP levels were therefore adjusted to this level with the carrier wave modulated to a depth of 75–85% in all experiments.

The long axis of the Crawford cell was oriented vertically inside a large incubator maintained at 35 ± 0.5 °C. These were the largest perturbations observed and related to prolonged opening of the chamber door. Temperature changes after equilibration and during field exposure were typically around 0.1 °C. Tubes containing the cell cultures (volume 2.0 ml) were placed in styrene foam racks supported on a lucite stand within 1–3 h of being placed in culture. Diameter of the culture tubes was 1.0 cm and they were arranged in a checkerboard pattern with intertube distances of not less than 2.0 cm nor more than 3.0 cm. Control cultures were placed in the same incubator in metal wire racks alongside the Crawford cell. A Narda probe disclosed no measurable field leakage on the exterior of the Crawford cell.

No changes in temperature of the culture medium between the beginning and end of the exposure were detected, but the question of a temperature rise in the culture tubes as a result of field exposure has been examined in detail in this laboratory by Dr. B. Vasquez and Dr. G. Kamin (personal communication). They used a

Crawford cell identical to that in these culture experiments. It is equipped with a heating and insulating system that maintains a temperature stability of ±. 0.2 °C. A small thermistor probe was placed on a thin layer of styrofoam® on the central platform conductor of the Crawford cell. The thermistor was sandwiched between the styrofoam® layer and the base of the culture tube containing 1.0 ml Kreb's solution. The base of the culture tube and the thermistor were coated with heatsink compound to ensure good thermal contact. Temperature resolution of the thermistor probe was 0.05 °C. Measurements were first made at 15 min intervals using a 450-MHz CW field, average power input of 1.7 W, producing an average incident field intensity of 1.0 mW/cm^2 at the exposure site. No temperature rise was noted over a 1-h exposure period. At 10 times this power input (average incident field 10 mW/cm^2), the temperature in the fluid increased by only 0.12 ± 0.05 °C after 1 h.

Protein Kinase Activity

The activity of the supernatant protein kinases was determined essentially as described previously [Byus et al, 1977, 1978; Byus and Fletcher, 1982] for cAMP-dependent protein kinase in human peripheral blood lymphocytes. The lymphocytes (10^7) in culture medium were centrifuged at 1000 × g for 5 min. The culture medium was rapidly decanted, and the pelleted cells were suspended in 350 μl of 0.05 mM potassium phosphate (pH 6.8), 0.5 mM isobutylmethylxanthine, 5 mM EDTA, 20 mM NaF, 125 mM KCl, and 1 mM dithiothretol. The lymphocytes were sonicated for 5 s at 0 °C using an E/MC sonicator (Kontes, Berkeley, CA) fitted with a 5-inch microprobe at a power setting of 6. The sonicated extract was rapidly transferred to a minicentrifuge tube and centrifuged at 4 °C for 30 s (Beckman model B Microfuge). Then 25-μl aliquots of the supernatants were assayed for protein kinase activity in the presence of saturating amounts of cyclic AMP (5 μM) in a total volume of 75 μl of the same buffer with 25 mM magnesium acetate, 200 μg of mixed calf thymus histone (Sigma Chemical Co., St. Louis, MO), and 0.5–1.0 μCi of γ-^{32}P ATP (2000 Ci/mmole; Amersham/Searle Corp., Arlington Heights, IL), plus sufficient nonradioactive ATP to bring the total substrate concentration to 0.1 mM. The assay was initiated by the addition of the supernatant and allowed to proceed for 5 min at 30 °C, and the reaction mixture was spotted on Whatman No. 3 MM paper filters. The filters were air dried and washed for 20 min in ice-cold 15% trichloracetic acid, followed by three 15-min washes in 5% trichloracetic acid and a 2-min wash in 95% ethanol. The discs were then air-dried and counted in 5 ml of a toluene-based scintillation fluid. Under these conditions the assay was linear for 5 min in the presence of cyclic AMP. In order to determine the relative amount of the total histone kinase activity that was due specifically to the cAMP-dependent protein kinase the lymphocyte supernatants were also assayed in the presence of the specific protein inhibitor (I) of this enzyme [see Byus et al, 1983; Byus and Fletcher, 1982; Ashby and Walsh, 1972, for details]. This protein inhibitor inhibits specifically the catalytic subunit of cAMP-dependent protein kinase. When saturating amounts of inhibitor are added to the supernatant assay only the cAMP-dependent kinase is inhibited. When the assays are performed in this manner the residual histone kinase activity is due to other than the cAMP-dependent enzyme kinases (see Discussion). In this manner replicate kinase assays were performed in the presence of saturating amounts of exogenous inhibitor and the activity due to cAMP-dependent protein kinase and other histone kinases was determined (see Results).

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RESULTS

Effect of 450-MHz Field Modulated at 16 Hz on cAMP-Dependent Protein Kinase Activity in Cultured Lymphocytes

Since our previous studies with cultured lymphocytes suggested a role for cAMP-dependent protein kinase in mediating the action of a variety of known mitogens [Byus et al, 1977, 1978; Klimpel et al, 1979] we first measured the alteration in total activity of this enzyme following exposure to a microwave field modulated at 16 Hz at a field intensity of 1.7 mW/cm^2 peak envelope power. The activity of cAMP-dependent protein kinase was measured in supernatants prepared from lymphocyte cultures exposed for up to 60 min (Fig. 1). These values were compared to those in control cultures placed outside the Crawford cell yet inside the same large incubator maintained at 35 ± 0.5 °C. Exposure for up to 60 min resulted in no significant alteration in specific activity of the cAMP-dependent protein kinase relative to the activity measured in unexposed cultures (Fig. 1).

Effect of 450-MHz Field Modulated at 16 Hz on cAMP-Independent Histone Kinase Activity in Cultured Lymphocytes

Activity of cAMP-dependent protein kinase was assayed in supernatant preparations using exogenous histone as a substrate as described in the "Methods" sections. However, there are other less well defined protein kinases in cells that will phosphorylate histones under standard conditions used for assay of cAMP-dependent kinase. When activity of cAMP-independent histone kinase(s) was determined in field-exposed cultures by assaying in the presence of cAMP-dependent protein kinase inhibitor (see Materials and Methods), a marked time-dependent reduction in kinase activity was observed (Fig. 2). While activity of this histone kinase(s) remained unchanged in control cultures, the enzymes measured in lymphocytes exposed to the microwave

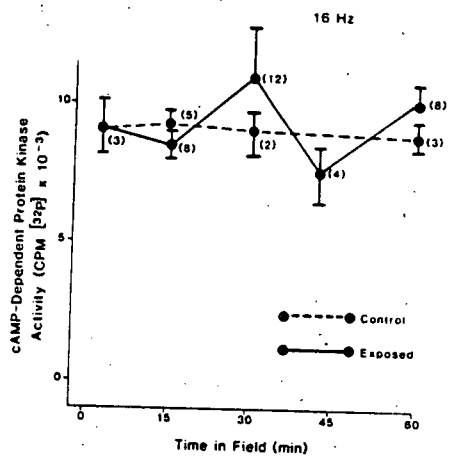

Fig. 1. Time-dependent alterations in cAMP-dependent protein kinase in cultured tonsil lymphocytes exposed to a 450-MHz field (peak intensity 1.0 mW/cm^2) modulated at 16 Hz. Control cultures were placed alongside the Crawford cell inside the temperature-controlled chamber for equivalent periods of time. The cultures were assayed for cAMP-dependent protein kinase activity, which is illustrated as the mean ± SEM for control (● - - - ●) and field exposed (●——●) cultures. Numbers in parenthesis indicate numbers of cultures assayed at each point in time.

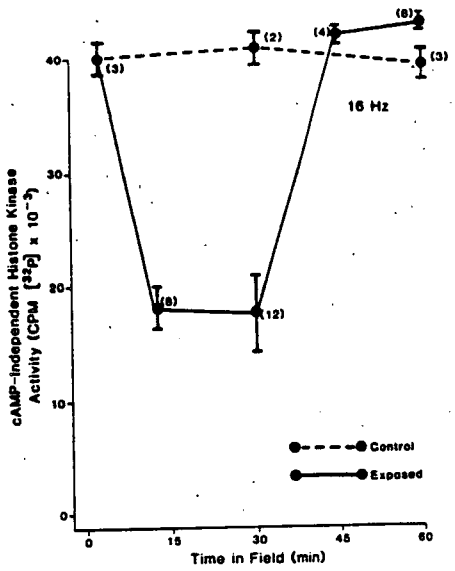

Fig. 2. Time-dependent alterations in cAMP-independent histone kinase activity in cultured tonsil lymphocytes exposed to a 450-MHz field (peak intensity 1.0 mW/cm^2) modulated at 16 Hz. Kinase activity shown is the total cAMP-*independent* protein kinase (ie, that kinase activity not inhibited by the protein inhibitor of the cAMP-dependent kinase). Data are presented as mean ± SEM for control (● - - - ●) and exposed (●—●) cultures. Numbers in parenthesis indicate numbers of cultures assayed at each point in time.

field modulated at 16 Hz decreased within 15 min of onset of field exposure (Fig. 2). The cAMP-independent histone kinase activity was reduced by 50–55% in the epoch from 15 to 30 min from onset of exposure to the field. However, in cultures exposed to the field for 45 or 60 min, activity of the histone kinase remained unchanged in comparison to the values in control or unexposed lymphocytes. Data in Figure 2 indicate that this amplitude-modulated microwave field induced a transient but major decrease in histone kinase activity that rapidly returned to control levels within 45 min of onset of continuous exposure.

Effect of 450-MHz Field Modulated at 60 Hz on cAMP-Independent Histone Kinase Activity in Cultured Lymphocytes

We monitored the time-dependent reduction in histone kinase activity in cultures exposed to a microwave field at the same intensity as that used in the previous experiment but modulated at 60 Hz. The 60-Hz field also caused a significant decrease in histone kinase activity in relation to the control cultures within 15 and 30 min of field exposure (Fig. 3). Reduction in kinase activity with 60-Hz modulation was not as large as in the 16-Hz field (Fig. 2), but still resulted in a 15% and 35% decrease in activity at 15 and 30 min, respectively (Fig. 3), in a manner similar to that observed in the 16-Hz field. However, the reduction in histone kinase activity by the 60-Hz field was transient, returning to control levels by 60 min.

We did observe some variation in the cAMP-independent protein kinase activity among different tonsils cultured and assayed by techniques described in the Materials and Methods section (Table 1). However, the variation in this activity from control or unexposed lymphocytes within a single tonsil preparation was relatively small. In

Fig. 3. Time-dependen
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TABL

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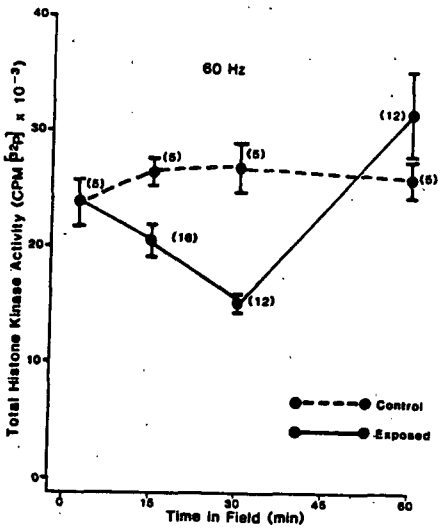

Fig. 3. Time-dependent alterations in histone kinase activity in cultured tonsil lymphocytes exposed to a 450-MHz field (peak intensity 1.0 mW/cm^2) modulated at 60 Hz. Lymphocyte cultures were assayed for protein kinase activity as described in Materials and Methods. The cAMP-independent histone kinase activity was assayed at indicated times as described previously. Data are presented as mean ± SEM for control (● - - - ●) and exposed (●——●) cultures. Numbers in parenthesis show numbers of cultures assayed at each time.

TABLE 1. cAMP-Independent Histone Kinase Activity*

Tonsil	Kinase (CPM ^{32}[P] × 10^{-3})	Activity (% of control)
1	41 ± 2 (8)	100 ± 5
2	26 ± 3 (20)	100 ± 11
3	34 ± 2 (5)	100 ± 6
4	39 ± 3 (12)	100 ± 8
5	37 ± 4 (6)	100 ± 10
6	35 ± 3 (8)	100 ± 9

*Variation in basal protein kinase activity among unexposed lymphocyte cultures from individual tonsils, showing cAMP-independent protein kinase activity obtained from six different tonsils. The data show means ± SEM of protein kinase activity from control unexposed lymphocyte cultures (number of cultures assayed in parentheses). Lymphocytes remained in culture from 1–4 h prior to assay. Protein kinase activity is also shown as ± SEM relative to 100% control value for each tonsil.

Figure 4 (see next section) we illustrate the frequency-dependent alterations in histone kinase activity relative to control (unexposed) values from a series of different experiments, ie, different tonsils.

Frequency Dependence of Amplitude Modulation on Field-Induced Effects on Histone Kinase Activity

Frequencies of amplitude modulation of the 450-MHz field in the range 0–100 Hz were tested for their ability to alter histone kinase activity of cultured lymphocytes

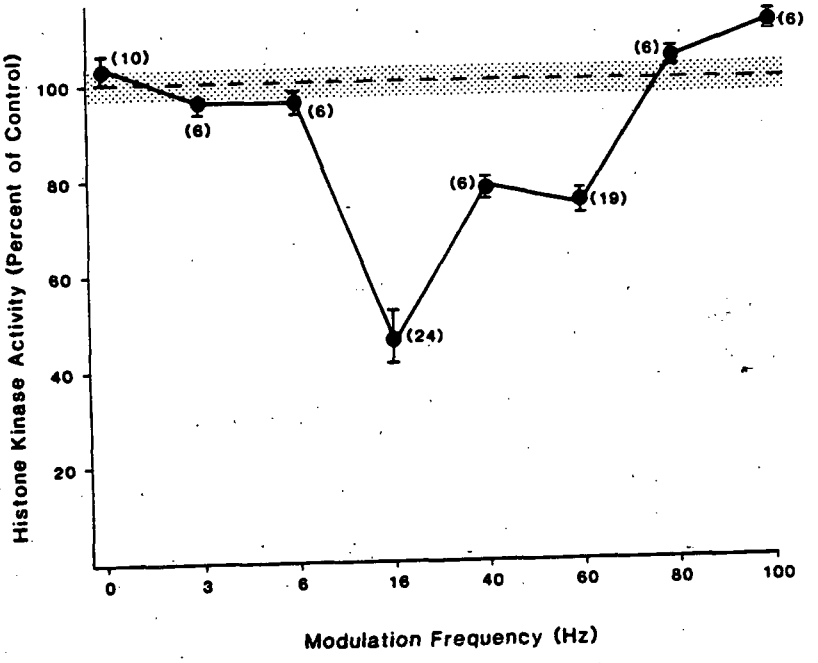

Fig. 4. Modulation–frequency-dependent alterations in protein kinase activity in cultured tonsil lymphocytes exposed to a 450-MHz field (peak intensity 1.0 mW/cm^2) sinusoidally modulated at 3–100 Hz (modulation depth 75–80%). All cultures were exposed for 30 min at specified modulation frequencies and assayed for histone kinase activity (see Methods). Control (unexposed) lymphocyte cultures were placed in the temperature-controlled chamber alongside the Crawford cell. Altered histone kinase activity at various modulation frequencies is shown relative to control (unexposed) values. Data are presented as mean ± SEM for control (shaded area) and exposed (●——●) cultures. Numbers in parenthesis show numbers of exposed cultures assayed.

(Fig. 4). Specifically, an unmodulated 450-MHz carrier with the same incident energy as the modulated fields produced no significant change in enzyme activity relative to the paired-unexposed cell cultures (Fig. 4). Modulation frequencies of 3, 6, 80, and 100 Hz were equally ineffective in altering histone kinase activity. Reduction of protein kinase activity in the lymphocytes exhibited a "windowed" frequency dependence with only 16-, 40-, and 60-Hz modulation frequencies reducing the activity of the enzyme (Fig. 4). The largest response occurred with 16-Hz modulation. Even though we did observe some variation in basal protein kinase among different tonsil preparations (Table 1), the 16-, 40-, and 60-Hz modulated fields consistently reduced the kinase activity relative to the control (unexposed) values within individual tonsils.

DISCUSSION

We have observed a large reduction in histone kinase activity in lymphocytes during exposure of the cells to relatively low levels of modulated microwave radiation. It is necessary to elucidate the "windowed" character of this response with respect to low modulation frequencies [Adey, 1980]. The finding of a transient

sensitivity of this protein kinase activity to continuing microwave field exposure suggests a "window in time" and merits further study.

This study has disclosed major microwave effects on cAMP-independent protein kinases, but no alterations specifically in the cAMP-dependent kinase. Thus, the activation pathway does not appear to involve adenylate cyclase and its action in ATP in production of cAMP. This finding contrasts with low-frequency pulsed magnetic field effects on bone cells, where adenylate cyclase activation by parathyroid hormone (PTH) was inhibited by the field [Luben et al, 1982]. Kostyuk [1983] has reported a role for protein kinases in calcium gating currents of muscle cells, also concluding that cAMP-dependent kinases are involved. A full knowledge of the broad range of possible transmembrane coupling systems remains for future research.

Maximum sensitivity to the microwave field occurred with 16-Hz modulation, coinciding with the frequency-dependent responses in brain tissue Ca^{2+} efflux both for low-frequency fields and for radio-frequency fields modulated at low frequencies [Bawin and Adey, 1976; Bawin et al, 1975; Blackman et al, 1979]. In contrast, allogeneic T lymphocytes showed maximum sensitivity in cytolytic activity when exposed to fields similar to those used here modulated at 60 Hz. This highly differential frequency sensitivity in the low-frequency range has been modeled in terms of quasiparticle behavior in linear macromolecules, such as transmembrane lipoproteins [Lawrence and Adey, 1982].

Disclosure of a "time window" in protein kinase sensitivity to field exposure suggests transient induction of a persisting but reversible molecular state in elements of the transductive system. There is evidence that these field-induced changes involve the cell membrane. Cytolytic activity of allogeneic T lymphocytes modified by a modulated 450-MHz field slowly reverses over a 12-h period [Lyle et al, 1983]. Electrophoretic displacement of concanavalin-A receptors on embryonic muscle cells is also reversible [Poo et al, 1978]. However, it is not clear from these studies why the response seen here should be "windowed," reversing in the face of continued field stimulation. Phase transition models of fixed-charge organization on membrane surface glycoproteins have been suggested as a basis for low-frequency sensitivities and persisting molecular states in field interactions [Grodsky, 1976; Lawrence and Adey, 1982], or they may arise in Volterra transitions in population charge states [Fröhlich, 1977] or limit cycle phenomena [Kaczmarek, 1976].

Since the decrease in histone kinase activity was observed to be both frequency- and time-dependent in the lymphocytes, this transient loss in the activity of an enzyme may serve as a measure of the ability of an electromagnetic field to affect a eucaryotic cell. Such observations might be important in defining limits of specific field and exposure parameters determining interactions with a biological system. These studies might then be important in controlling potential hazards in field exposure.

At present, our data offer no insight into the real biological effect of these fields upon lymphocyte function in particular, or upon the general state of the immune system. We cannot say at this time whether this transient decrease in protein kinase alters the ability of lymphocytes to perform any specific function. For this reason it is difficult to make any statement concerning potential damage to the cell based upon these data. A number of studies have reported altered ability of lymphocyte or immune systems to function in a "normal" manner following exposure to particular fields. A recent study [Roberts et al, 1983] has concluded that existing U.S. safety standards for general population exposure to microwave fields are adequate. Their

study was based on CW field absorption rates up to 4 mW/ml in lymphocyte culture medium, ignoring possible effects of low-frequency amplitude modulation. Findings here and in other studies of lymphocyte immune functions [Lyle et al, 1983] indicate that need for a more cautious assessment, with due consideration for bioeffects of amplitude modulation found in virtually all domestic, industrial, and military microwave fields.

We intend to further define and identify the specific histone/protein kinase that is affected by the 16–60-Hz modulated fields and thus to understand what functional changes, if any, may have occurred in the lymphocytes during field exposure. Though cAMP-dependent protein kinase activity did not appear altered in the exposed lymphocytes (Fig. 1), other known kinases would lead to the phosphorylation of mixed calf-thymus histone under the reaction conditions employed. These include calcium–calmodulin-activated protein kinase [Moore and Dedman, 1982] and the phospholipid–calcium-activated kinase (protein kinase c) described by Nishizuka and his colleagues [Ogawa et al, 1981].

ACKNOWLEDGMENTS

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The Microwave Problem

Is exposure to low levels of microwaves a hazard? How strict should exposure limits be? These issues remain in dispute in part because some findings on the biological effects of microwaves are ambiguous

by Kenneth R. Foster and Arthur W. Guy

CHMN, ANSI C95.1 SUBCOMMITTEE 1970-1982. VICE CHMN 1982 ON.

Air-traffic-control systems, police and military radar, earth-to-satellite television broadcast systems, long-distance telephone equipment, medical diathermy devices and microwave ovens all generate microwaves. Except when microwaves are deliberately applied to the body for therapy, little of this invisible energy ever reaches the public. Is exposure to such low levels of microwave energy hazardous to human health?

This question is easier to ask than to answer. The interaction of microwaves and living organisms has been studied much more extensively than most other potential environmental hazards have, and yet the result has been continuing public and scientific controversy.

Communications equipment is a source of widespread—albeit extremely weak—public exposure to microwaves. A typical dispute arises when a company petitions a county zoning board for permission to install a microwave communications facility. The company's spokesmen might explain that the intensities, or power levels, of transmission will result in peak exposure levels that are some thousandths or millionths as high as allowable limits. These limits in turn are considerably below the levels known to produce biological damage.

Opponents argue that low levels of microwave energy may pose some as yet unproved danger and that their safety is still to be demonstrated. They point to the many biological effects that have been suggested at one time or another (by animal studies) to be related to exposure to low-level microwave energy, such as changes in immune-system functions, altered behavior, changes in the permeability of the brain to molecules carried in the blood, damage to chromosomes and the development of cancer.

Controversy continues despite the data provided by approximately 6,000 studies in the 40 years since microwave technology was introduced are inconsistent and inconclusive. Whereas it is known that exposure to high levels of microwaves can burn human tissues or cause heat stress, no clear-cut damage to human beings from low-level radiation has been demonstrated. On the other hand, exposure to low levels of microwaves cannot be proved free of hazards. This lack of consensus does not result from flawed research alone. The cause is more fundamental: the normal process of risk assessment yields data that can be subject to different interpretations and can generate controversy, whether or not a hazard is ever demonstrated.

Given the inconclusive state of the published evidence, we do not argue here that exposure to low-level microwave energy is either hazardous or safe. Rather, we shall describe the process by which known hazards have been quantified. We shall also review the development of the first major American standard for limiting exposure to microwaves and the rationale behind the standard. We shall then review several case histories that indicate why assessing the possible hazards of microwaves—and low-level environmental agents of all kinds—is so challenging.

Concern over the biological effects of microwaves must be viewed in the context of experience with lower-frequency electric fields, which were exploited for technology before microwaves were. Microwaves are a part of the electromagnetic spectrum, and their band extends from 300 megahertz to 300 gigahertz; that is, it includes waves with oscillation frequencies ranging from 300 million hertz, or cycles per second, to 300 billion hertz. Those frequencies are higher (and the wavelengths corresponding to the frequencies are shorter) than those of standard radio and television signals. Above the microwave band there are, in order of increasing frequency, infrared radiation, visible light and "ionizing" radiation: ultraviolet radiation, X rays and gamma rays.

X-RAY ETC

We should point out that ionizing radiation is qualitatively very different from microwave energy in its effects on biological systems. As one photon, or energy packet, of an ionizing ray passes through a substance, the photon breaks chemical bonds (even in the absence of any appreciable heating) and causes neutral molecules to become charged. Such ionization can damage tissues. In contrast the energy of a photon of a one-gigahertz microwave is only one six-thousandth of the kinetic energy possessed by a molecule in the body owing to normal thermal agitation, even less than the energy needed to break the weakest chemical bond. This does not rule out the possibility that weak microwave energy can directly alter molecules of tissue, but it does not suggest a mechanism by which significant changes could occur.

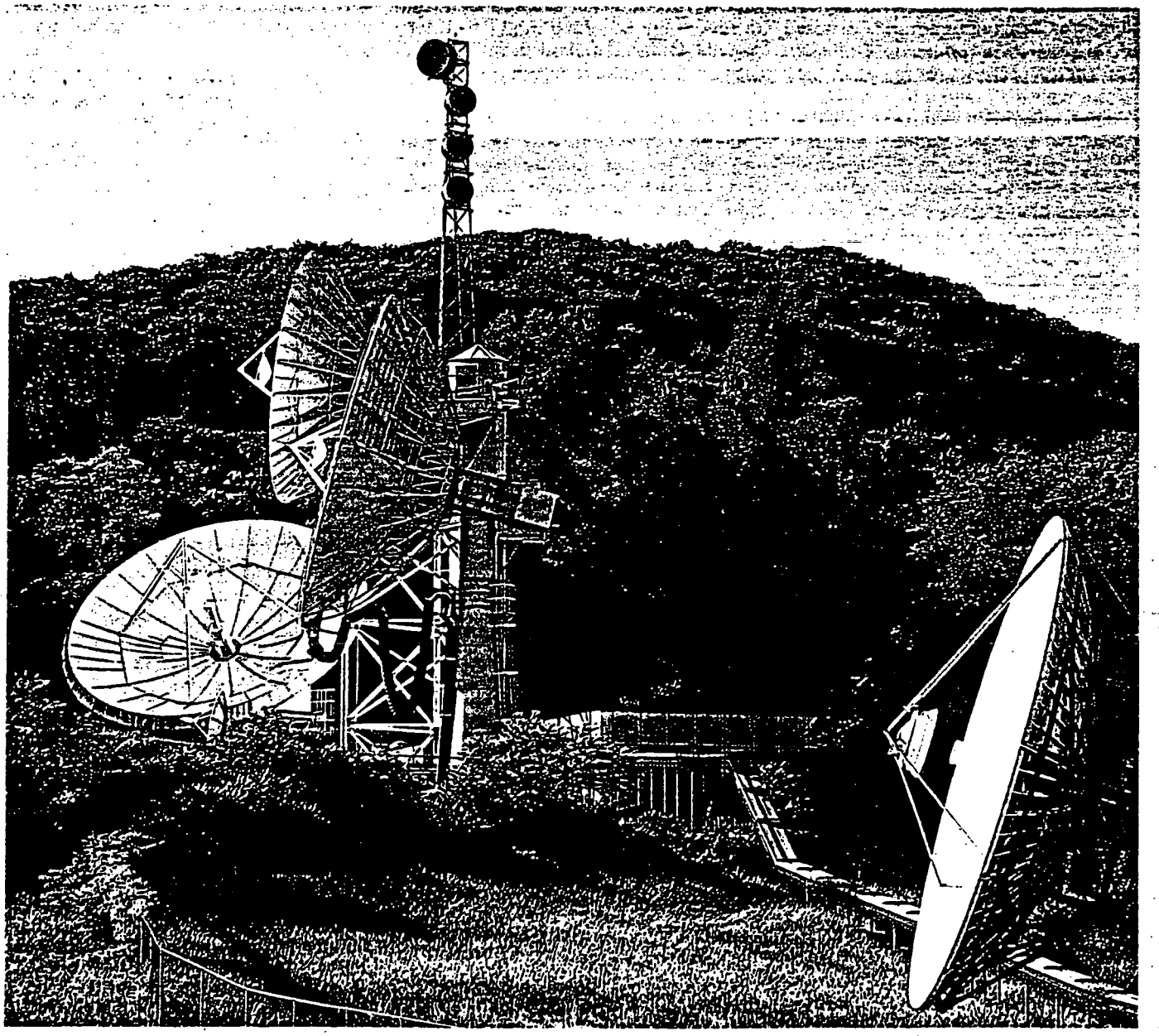
By the time microwave technology was introduced, during World War II (when radar changed the course of the

war), electromagnetic fields at lower frequencies had been employed for therapeutic heating for most of the century. Although the value of therapeutic heating was not disputed, the one obvious potential hazard was that of burns or other damage resulting from excessive tissue heating. In addition controversy raged among investigators over claims that high-frequency electromagnetic energy produces biological effects in various organisms, effects that some believed did not arise from simple heating alone.

In the decade after World War II microwave technology continued to develop rapidly, and with this progress came widespread human exposure to the energy. Although few injuries of any kind were reported, the possibility of hazard became increasingly disturbing. Because of the military's extensive application of relatively high-powered radar equipment, the services took a lead in investigating microwaves, establishing research programs in the early 1950's that continue in expanded form today. Also in the early 1950's, many investigators and Government officials began to consider setting exposure guidelines.

One of those people was Herman P. Schwan of the University of Pennsylvania. Based on theoretical estimates of tissue heating, he recommended in 1953 that human exposure to microwave energy be limited to a maximum average "power density" of 100 watts per square meter. = 10mw/cm²

Schwan's calculations showed that exposure to this incident power level should raise the temperature of any region of the body by one degree Celsius or less, and add heat at a rate comparable to that generated by the body's normal physiological processes. The limit was roughly one-tenth as intense as bright sunlight and perhaps one-fiftieth as intense as the power levels of diathermy equipment. Schwan



ANTENNAS (*large "dishes"*) at a communications station in Vernon, N.J., transmit microwave signals to satellites; other antennas (*smaller disks*) relay energy to receivers on the earth. The signals are put to a variety of purposes, including satellite television broadcasts. Transmissions from these antennas, and those from the many others that make Vernon a hub of microwave satellite communications, result in exposure levels to the public that are well below the safety limits imposed by the state. (Backyard dishes that merely receive transmissions are not a source of exposure.) Vernon is one of several places in the U.S. where citizens' groups have protested the installation of microwave generators, fearing that exposure to even low levels of microwave energy may pose health hazards.

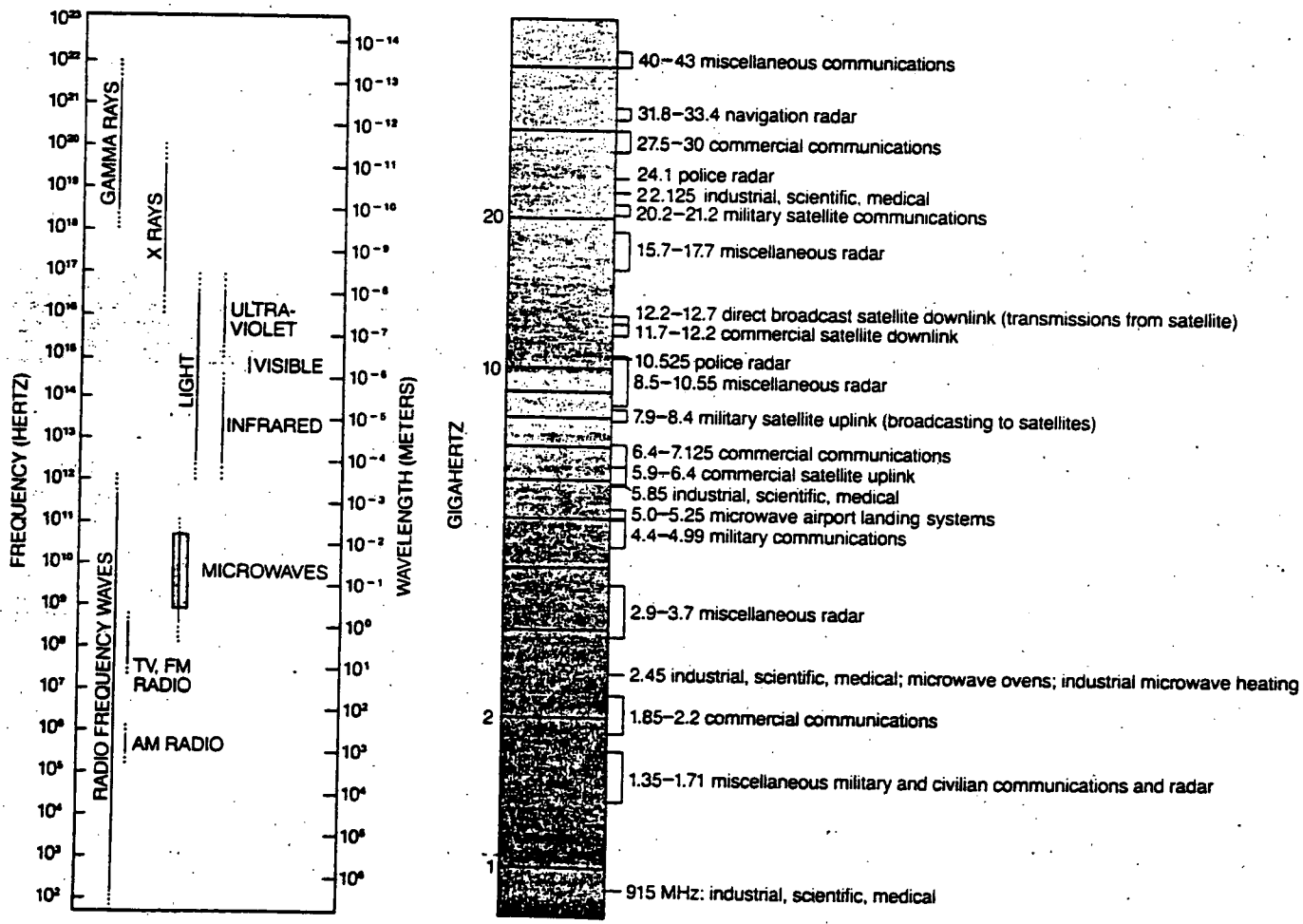
also calculated that power densities of about 1,000 watts per square meter might produce heat damage to the body in some circumstances. The proposed limit, then, allowed a safety margin of roughly 10.

Schwan directed his advice to the U.S. Navy, which had expressed concern about microwave safety, and his proposal was eventually embodied in a formal standard by the United States of America Standards Institute, later renamed the American National Standards Institute (ANSI). ANSI, a private organization that recommends safety guidelines of many kinds for different industries, adopted the standard in 1966 after reviewing the scientific literature and finding no convincing evidence for damage in animals exposed to microwaves at power levels below 100 watts per square meter. Many Western nations soon adopted comparable standards, and the ANSI guideline (formally known as ANSI Standard C95.1) became the most influential standard for occupational and public exposure to microwaves in the U.S.

The original ANSI standard, which covered frequencies between 10 megahertz and 100 gigahertz, remained essentially the same for many years, but by 1982 ANSI's periodic reexamination of the guideline suggested a change was in order. By that time too the safety of exposure to low-level microwave fields—ones transmitted at power levels too low to cause significant heating—had become a subject of popular, political and scientific debate.

Public concern had been heightened by several factors. Between the 1940's and the 1970's many studies had addressed the biological effects of microwave energy. Most of the reported effects occurred at power densities above 100 watts per square meter, but a few, some of which might be interpreted as harmful, had been reported at power densities substantially below the ANSI guideline. (One investigator, for example, reported in 1968 that pulsed microwave energy beamed at an average power density less than one ten-thousandth of the ANSI guideline could change the beating rate of isolated frog hearts, sometimes stopping a heart entirely. That finding could not be confirmed by other investigators.) People had also heard news reports that the Soviet Union had for many years beamed low levels of microwave energy at the American Embassy in Moscow, reports that often included speculations about health effects. In addition the Soviet Union and the Warsaw Pact countries had set exposure limits for the general population at levels 100 to 1,000 times lower than any American standard.

The major impetus for ANSI's revision of the guideline was data from studies employing improved methods of dosimetry, or measurement of absorbed energy. The absorption of energy by an animal or a human being depends in a complex way on such variables as the frequency of the energy, the subject's size and orientation to the waves, and the type of transmitting antenna used. Exposed to identical energy, a rat and a human being will absorb vastly different amounts of energy per unit of body weight. This



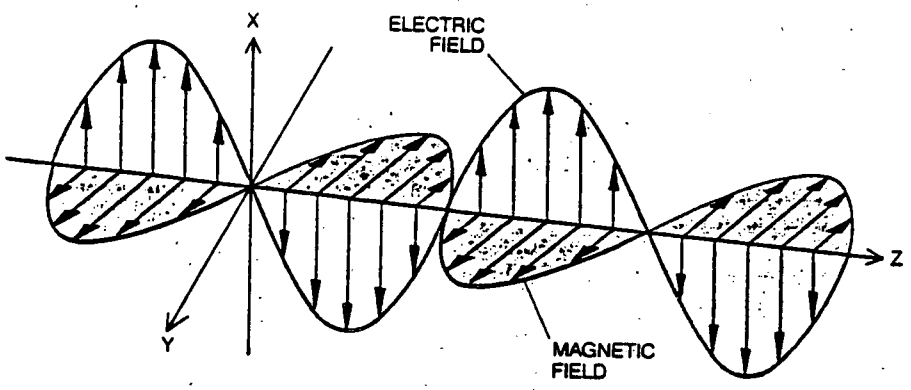
MICROWAVE BAND of the electromagnetic spectrum (*left*) extends from approximately 300 million to approximately 300 billion hertz. Microwaves are exploited for an array of applications, only a few of which are listed at the right. Military and civilian radar and communications functions are concentrated in the range of .3 to 40 gigahertz (billions of hertz). Medical, industrial and scientific technologies employ several frequencies, but the ones most commonly applied are 915 megahertz (millions of hertz) and 2.45 gigahertz.

complexity had long been recognized, but before the late 1960's few investigators carefully measured the amount of energy absorbed by their subjects. Beginning in the 1960's engineers undertook studies that led to a fuller understanding of energy absorption and to better experimental techniques for measuring it in animals.

Several methods developed in the past two decades calculate absorbed energy by measuring the temperature rise in models of living subjects. One of us (Guy) introduced the "split phantom" technique in 1968. Workers shape plastic foam into a hollow model of an animal, fill it with a gel that has electrical properties resembling those of living tissue and briefly irradiate it. Then they split the model open and photograph it with an infrared camera to record temperature increases. Workers can also determine energy-absorption patterns by computer simulation. The animal or exposed person is modeled as a simple ellipsoid or cylinder. More elaborate mathematical models, consisting of block figures, have also been studied.

By 1982 all the new methods agreed that the amount of energy absorbed varies widely with the frequency of the radiation and the size of the body. Under unfavorable circumstances a person might absorb up to 10 times as much energy at frequencies between 70 and 100 megahertz as at higher frequencies, depending on the individual's orientation to the waves. Electromagnetic waves consist of electric and magnetic fields that are perpendicular to each other and to the direction in which the waves are traveling [*see top illustration on this page*]. Maximum absorption occurs when waves impinge on the body from the side, where the electric field is parallel to the body's long axis and the magnetic field is perpendicular to the front of the subject. The human body is an efficient antenna for waves in the 70-to-100-megahertz range; it is said to "resonate" with the fields at those frequencies.

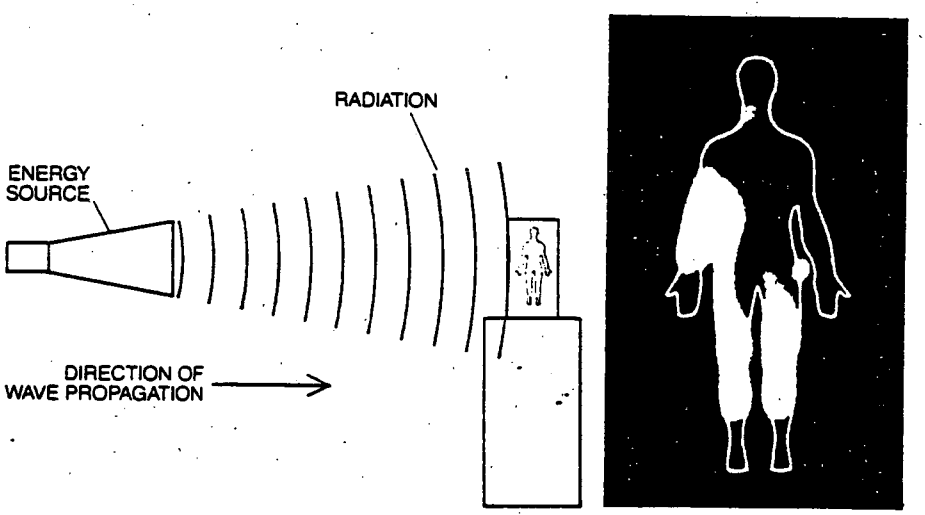
To adjust for these resonance effects, in 1982 an ANSI committee reviewing the standard decided to make power-density limits dependent on frequency, with the overall goal being to limit the energy absorbed by the body. Whereas the old guideline had specified only the intensity of the energy incident on the body, the new one attempted to limit average entire-body absorption levels to .4 watt per kilogram of body weight. (Somewhat higher peak absorption levels might be allowed for partial-body exposure.) When the body is at rest, it normally generates heat at twice that rate, and it generates much more heat during moderate exercise. Compared with the previous guideline, the 1982 standard called for significantly reduced power densities at resonance frequencies, and it applied to a wider range of frequencies, from 300 kilohertz to 100 gigahertz [*see top illustration on page 38*]. The incident power level was calculated by averaging power over six-minute periods; thus the standard allowed brief exposure to high power levels.



ELECTRIC AND MAGNETIC FIELDS of microwaves and other forms of electromagnetic radiation are perpendicular to each other. They are also perpendicular to the direction of wave propagation. When the radiation travels in the direction that is shown by the *z* axis, the electric field (*color*) is parallel to the *x* axis; the magnetic field is parallel to the *y* axis.

ANSI approved the 1982 standard only after its review of the scientific literature uncovered no convincing evidence for health-damaging effects in animals exposed to energy producing absorption rates below .4 watt per kilogram. The committee concluded that the threshold for possible hazards was an absorbed power level of roughly four watts per kilogram of body weight; in other words, the standard had a safety factor of about 10 built into it. The committee further concluded that the proposed standard would exclude heat stress and burns. It would also avoid other reported effects for whose existence and undesirability the evidence was considered reliable (such as serious disruption in the behavior of several kinds of animals at absorption levels of four to eight watts per kilogram).

The standard did not attempt to avoid all reported effects, because the

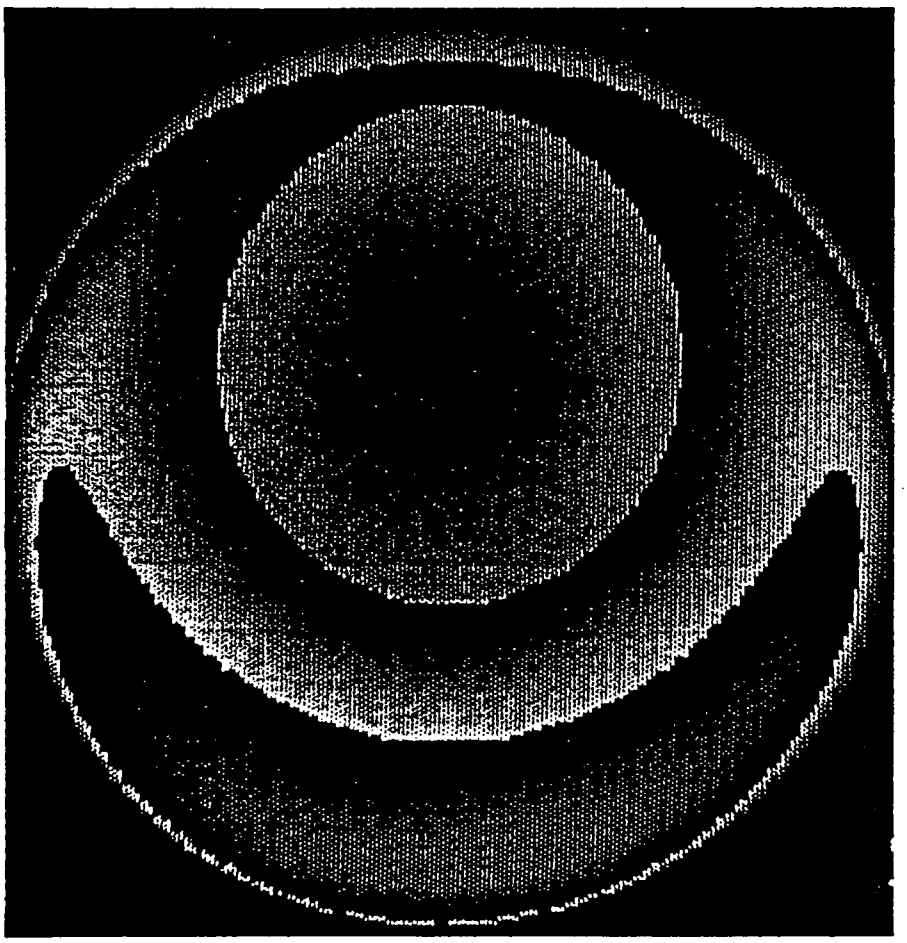


"SPLIT PHANTOM" TECHNIQUE for dosimetry measurement of absorbed energy was introduced by one of the authors (Guy) in 1968. The technique measures the temperature rise in models of human beings or animals and reveals the pattern, depth and amount of energy that would be absorbed by a living creature. Workers hollow out two halves of a Styrofoam block to form a mold of the body and then fill the mold with a gel that has electrical properties resembling those of living tissue. The model is briefly irradiated (*left*) and quickly split apart, and the interior is photographed with an infrared camera to record the amount of heating in various regions. The photograph at the right reveals the pattern of energy deposition produced in one such model, which, for practical reasons, is smaller than life size. The pattern is comparable to that produced by 79-megahertz waves (a frequency resulting in maximum absorption by humans) striking the side of a man weighing 70 kilograms and standing 1.74 meters tall. The bright areas indicate relatively high absorption.

literature relating to many of the effects was—and still is—problematic. Many studies have shown that exposure to high levels of microwaves is clearly hazardous, producing obvious heat stress in animals. Other studies, in which absorption levels are comparable to the rate of heat generated by the body, have observed changes that in part could be normal physiological responses to the added heat, although evidence for this inference is often less clear. Other effects have been reported at quite low power levels; no obvious explanation has been found.

There is also great diversity in the quality of the evidence. Although many studies reporting effects on living systems have apparently been well done, some have had obvious technical flaws (in particular, some have lacked adequate dosimetry) and others have been too briefly described to allow any judgment of their quality. Of the hundreds of biological effects of high or low levels of microwaves that have been reported, a surprising number are examples of the "Cheshire cat" phenomenon: they have not reappeared in follow-up studies.

Part of this disparity arises from the nature of the research itself. An investigator might report an "effect" on the basis of some difference found between control subjects and those exposed to microwaves. The reported effect might well arise from some specific biological activity of the energy. On the other hand, it might also result from a normal physiological response to added heat, from a statistical fluctuation or even from some experimental variable that was not adequately controlled by the investigators.



COMPUTER-GENERATED SPHERE can be employed to study energy-absorption patterns in tissues and is particularly valuable for determining how changes in frequency alter the distribution of energy within irradiated subjects. The sphere, which can represent a person or a part of the body, is evaluated as if it had electrical and heat-transporting properties resembling those of human tissues. With the computer an investigator calculates the energy-deposition patterns, assigning different colors to represent amounts of temperature increase. When a sphere with a 10-centimeter radius is irradiated at a frequency of 100 megahertz, the front surface absorbs the greatest amount of energy (*red*); the region just behind the surface absorbs a moderate amount (*green*) and the center of the sphere absorbs very little energy (*blue*). At an incident power level of 10 watts per square meter, the maximum calculated temperature rise in this sphere is minuscule: .003 degree Celsius. The image shown here was made by Haralambos N. Kritikos of the University of Pennsylvania.

To illustrate the ease with which data can be subject to various interpretations and produce apprehension, we have chosen three case studies. All the reported findings were observed at power levels that are within the ANSI guideline in effect before 1982, and all have played some role in the public controversy about possible hazards of low-level microwave energy.

The first case involves the possible influence of low-level microwave energy on brain function. In 1975 two American teams of workers reported that exposure to power levels from one-third to one-fiftieth of the ANSI standard could increase the rate at which tracer molecules enter the brain from the blood. Although the exact significance of this finding for human beings was difficult to judge, any such effect could be construed as disturbing the blood-brain barrier and would be cause for concern. Predictably, the report fueled the microwave-safety controversy. A dozen research groups followed up on this finding during the next decade. As studies continued and were progressively better controlled at low exposure levels, the effect went away. (Most investigators agree, however, that exposure to energy levels high enough to significantly heat the brain would produce substantial alterations in the barrier.)

The second case study involves the "microwave auditory effect," which has been known since 1947. When a subject's head is exposed to pulses of microwave energy, the person can often hear "clicks" in synchrony with the pulses; these clicks seem to originate from within the head. To be heard, the pulses must be relatively intense (on the order of 10,000 to 500,000 watts per square meter); they can be brief enough (microseconds), however, to result in a rate of energy absorption that, when averaged over time, falls well below maximum safety levels.

Results of one early study on the effect suggested that the center of the brain was the most sensitive region for producing the clicks. The alarming possibility thus arose that the pulses of microwave energy might somehow act directly on the brain. In 1974 one of us (Foster) proposed that the clicks might result from a benign physical effect accompanying the absorption of the energy by the head. The proposed mechanism was simple: the thermal expansion of tissue, caused by minuscule but abrupt heating (a few millionths of a degree following each pulse), launches sound waves that the subject perceives as clicks. Simple calculations and experiments using water-filled models showed that sound waves generated by

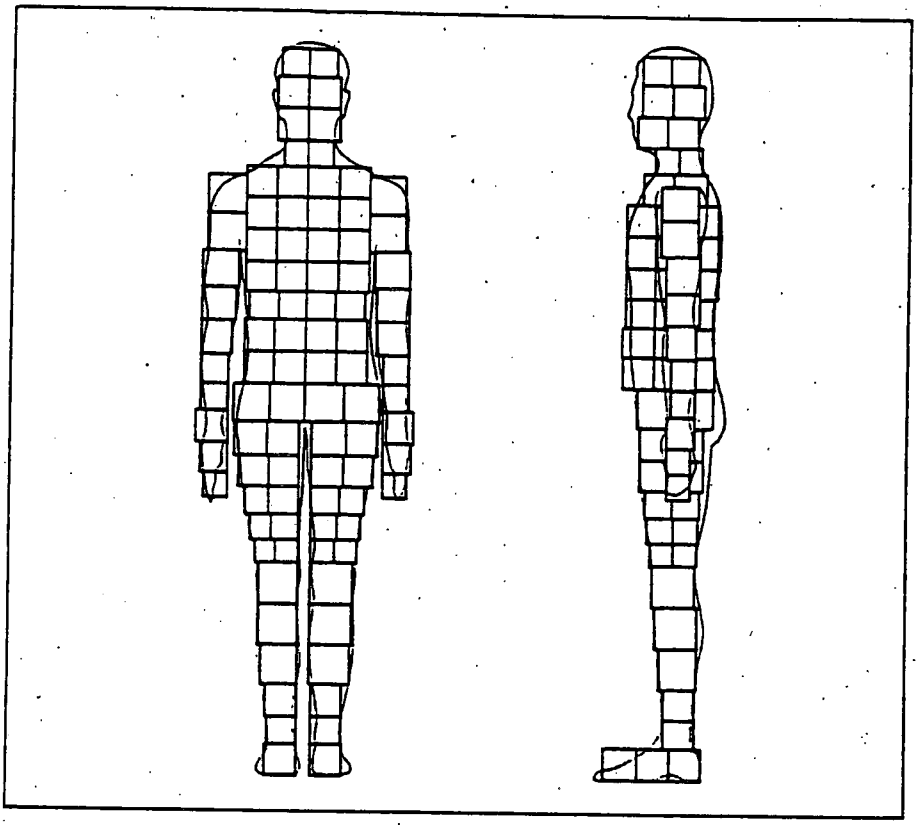
the microwave pulses should indeed be audible. Soon after, studies with animals confirmed the hypothesis. The microwave auditory effect is not now regarded as pointing to a hazard.

Our final illustration is an extended study conducted by one of us (Guy) with his colleagues at the University of Washington at Seattle. Its results reveal typical ambiguities that can result from screening studies, which are the source of many reports of microwave effects. The three-year study of the effects of long-term, low-level irradiation was funded by the U.S. Air Force School of Aerospace Medicine. It compared 100 rats that were irradiated for most of their lives with 100 rats that were not exposed to radiation but were otherwise treated identically. The radiation beamed at the experimental group had an average power level of five watts per square meter and a frequency of 2.45 gigahertz. The rats were exposed for 21 hours per day for 25 months. On the average, depending on age, they absorbed from .2 to .4 watt per kilogram of body weight, the latter being the current ANSI exposure limit for human beings.

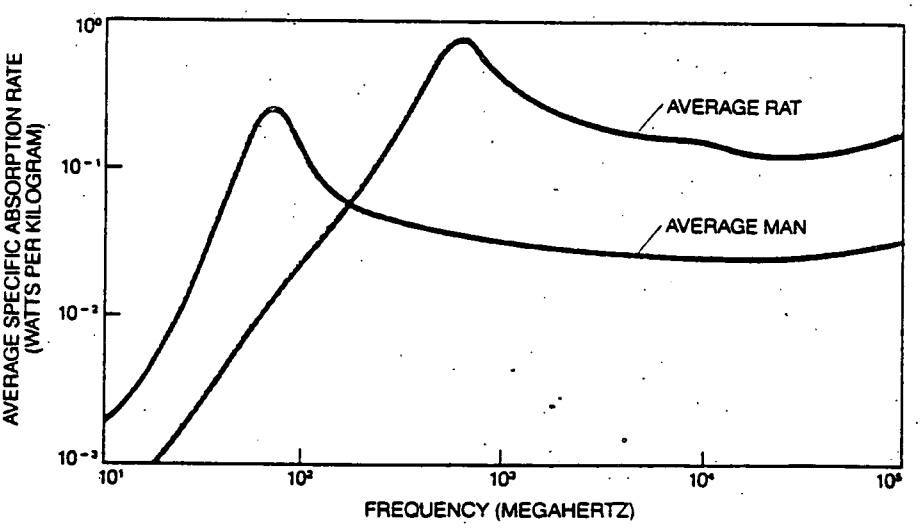
The investigators examined 155 different measures of health and behavior, including blood chemistry, body weight, daily food and water consumption, oxygen consumption, carbon dioxide production and activity level. The results revealed few differences between the exposed and the control rats, and those differences for the most part were either not statistically significant or came and went, suggesting that they might be due to chance. For example, plasma cortisone levels (which indicate the level of arousal) were higher for the exposed group during the first sampling session but were higher for the controls during the third session.

Earlier studies had suggested that microwaves might impair the immune system. The Seattle group therefore evaluated the function of lymphocytes, a cell type fundamental to the immune response. Some but not all of the immunological tests showed a difference between the exposed and the control animals after 13 months. After 25 months the differences were no longer discernible. The mean survival time of the exposed animals was slightly longer than that of the control animals: 688 days v. 663 days—a difference that was probably due to chance alone.

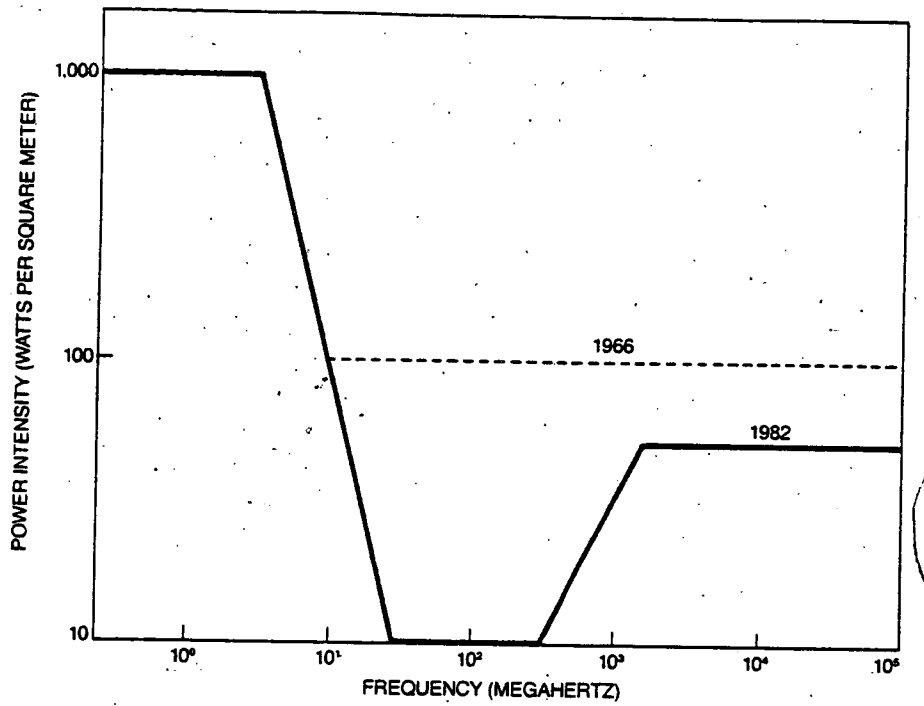
One difference was striking: primary malignant tumors developed in 18 of the exposed animals but in only five of the controls. The probability



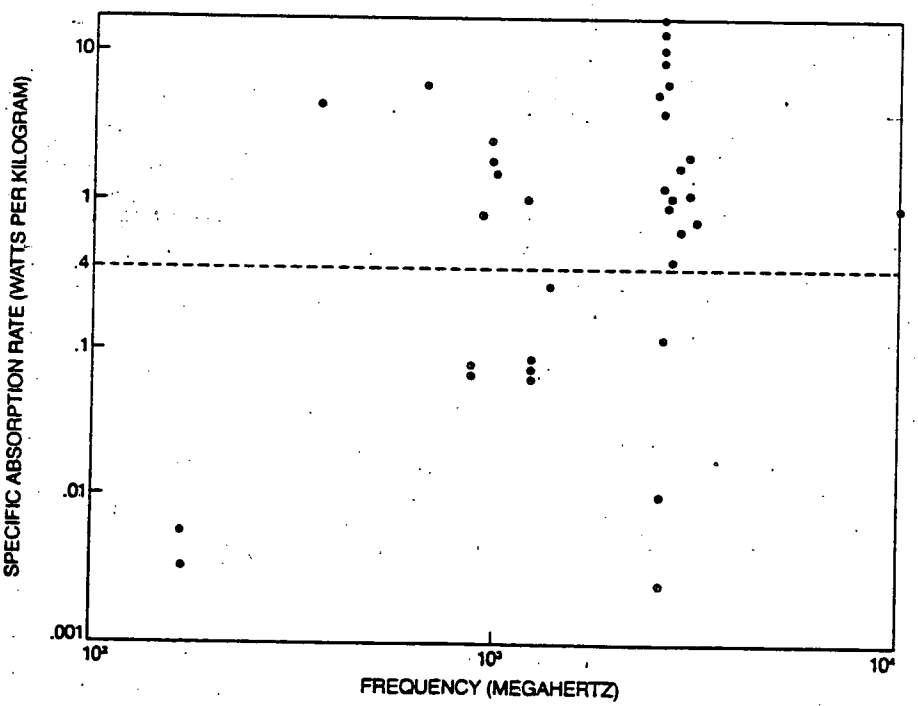
BLOCK FIGURES are the most complex of all theoretical models employed to calculate energy absorption. Workers load the coordinates of each block into a computer. Then, on the basis of presumed electrical properties of the tissues, radiation frequency and other factors, the computer calculates the specific absorption rate (SAR): the watts of energy absorbed per kilogram of weight when a subject is exposed to a power density of 10 watts per square meter. Other theoretical models (not shown) include ellipsoids that are assumed to be filled with homogeneous material representing human tissues and ellipsoids that are assumed to contain separate layers representing fat and muscle. Results from all these theoretical approaches agree well with one another and with data derived from experiments with irradiated models, increasing confidence in the accuracy of the various calculations.



RATES OF ENERGY ABSORPTION by an average-size rat and man differ at most frequencies and also peak at different frequencies, as is shown by comparisons of SAR's in ellipsoid models. Man has a pronounced increase in SAR at approximately 70 megahertz, the rat near 1,000 megahertz. The large differences in SAR's between the species arise from the differing properties of their bodies as antennas. Such differences, together with differing physiological responses to heat, are important sources of uncertainty when investigators attempt to extrapolate from animal data to determine a power threshold above which microwaves pose a hazard to human beings. The curves reflect absorption by subjects exposed to waves propagated toward the body at an angle that most promotes energy absorption. (The electric field is perpendicular to the long axis of the body; the magnetic field is perpendicular to the frontal plane.) The data are from Carl H. Durney of the University of Utah.



WIDELY RECOGNIZED STANDARD for maximum public and occupational exposure to microwaves was established in 1966 by the American National Standards Institute (ANSI) and was significantly revised in 1982. In 1966 the voluntary standard allowed the maximum incident power level to be 100 watts per square meter for frequencies ranging from 10 megahertz to 100 gigahertz (100,000 megahertz). An ANSI committee revised the limit in 1982 to reflect the finding that the human body absorbs more energy at some frequencies than it does at others. The 1982 version makes power densities dependent on frequency, lowering allowed power levels for frequencies in the neighborhood of 100 megahertz. The standard's goal is to limit absorption to .4 watt per kilogram, averaged over the entire body. If only part of the body is exposed, higher power intensities are sometimes permissible.



REPORTS OF BIOLOGICAL EFFECTS were plotted (*dots*) by the ANSI committee according to SAR and frequency before the organization revised its exposure standard in 1982. For effects reported to occur at SAR's above five watts per kilogram, different studies generally agreed on the nature of the effect and the SAR's that would reliably produce it. For effects reported at lower power levels, different studies showed much less agreement. Many of the effects reported at the lower levels were not considered indicative of a hazard. ANSI further determined that reported effects that could be considered hazardous occurred at an entire-body SAR of four watts per kilogram and above; the organization arrived at the 1982 SAR limit of .4 watt per kilogram (*broken line*) by then building in a safety factor of 10.

of such a difference occurring in two samples from an identical population of only 100 animals each is roughly .005, and so the difference is statistically highly significant.

At face value this last finding suggested that low levels of microwave radiation can cause cancer in mice (and by inference in humans). The finding was widely reported by the lay media in 1984 and has been frequently cited in public disputes over proposed microwave facilities. Nevertheless, various considerations militate against drawing a hasty conclusion.

For one thing, the total number of malignant tumors in the control animals was lower than the number expected for the particular strain of rat; the rate of malignancies in the exposed rats was about as expected. Thus the exposed animals had an excess of tumors only in comparison with the controls, not in comparison with the rate of tumor development generally observed in this strain of animal.

Other problems, of a statistical nature, also arose. So few malignancies were found that tumors of all kinds had to be grouped in the statistical analysis. No single type of tumor predominated, nor was the incidence of any single type of tumor unexpected on the basis of previous studies. If some specific type of tumor had predominated, that finding would have made a much stronger case for a carcinogenic effect from low levels of microwave energy.

The comparison of rates of malignancy was just one of 155 different comparisons made in the study. Given such a large number of comparisons, some striking differences would be likely to be found that are in fact merely chance occurrences. The cancer finding may be such a statistical anomaly. In short, the finding of excess cancer is provocative, but whether it reflects a biological activity of microwave radiation is not certain. To demonstrate reliably a connection between microwave irradiation and the development of any one kind of tumor might require a study hundreds of times larger and more expensive than the Seattle study—a size that might be infeasible. Our conclusion from these examples and from the large literature on microwaves is that although some hazard from weak microwave fields might be proved in the future, there is currently little evidence for the presence of such a hazard.

Considering that some uncertainty persists, how should future research proceed, and on what basis should any new standards be set? During the 1980's U.S. Government support of in-

vestigation into the possible biological effects of electromagnetic fields, including those at power-line and radio frequencies, has been at the level of about $10 million per year. The resulting work, taken together with earlier studies, has produced a consensus among most investigators that the only strong evidence for the hazards of microwaves is found at high levels of exposure. Beyond this, agreement is harder to come by. Scientists disagree over the meaning of effects that have been reported to occur in animals at exposure levels slightly below the ANSI standard. Such disagreements are not likely to result in drastic changes in exposure standards. Public debate, on the other hand, has often focused on the possibility of hazards at much lower levels of exposure and on the possible need for more stringent standards.

Better coordination of future research should reduce some of the scientific and public confusion. Many Government agencies, including the military, have funded investigations of the biological effects of microwaves. Lack of coordination among these agencies has unfortunately resulted in a scatter-gun approach in which many preliminary studies have been carried out but not followed up.

Before new studies are undertaken the Government should commit itself to supporting independent attempts at duplication and also supporting follow-up studies to explore the significance of new findings. Some agencies appear to be moving in this direction, with the Army, the Air Force and the Navy now funding follow-up studies on controversial recent reports. Conversely, some criteria must be developed for determining when to halt research on a given topic, open questions notwithstanding.

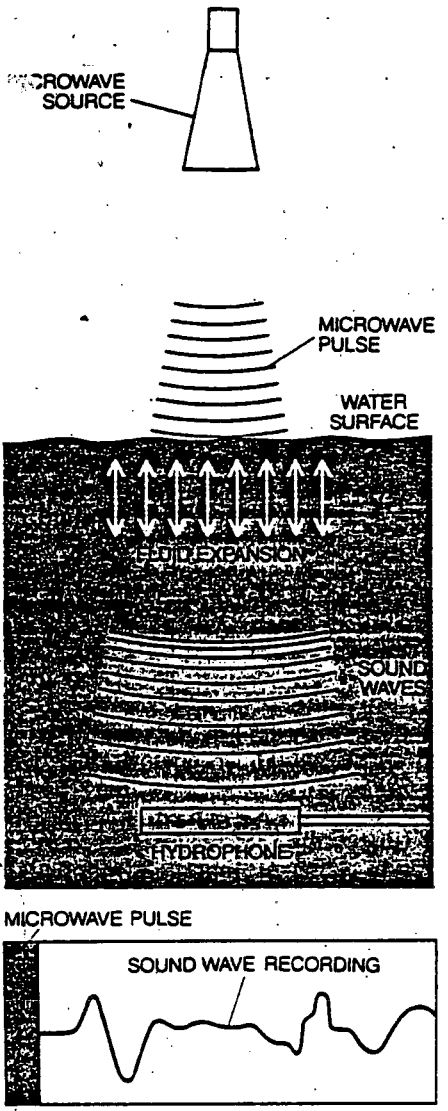
Even with improved coordination of research, it will not be possible to prove the absence of hazard, which means that regulators cannot guarantee total safety when they set standards. Standards are one way by which society balances the benefits of technology against potential risks. Traffic speed limits are set at a level that achieves some balance between the danger of excessive speed and the desire of most people to travel as quickly as possible. The limits offer no promise of zero risk at lower speeds but merely legitimize the speeds at which known hazards are unlikely to be a problem. Similarly, exposure limits for microwaves (and other environmental agents) can only be based on known hazards, with a safety factor built in. The revision of the 1982 ANSI standard is an example of this approach. When improved dosimetry revealed that the human body can absorb more energy at specific frequencies, ANSI in effect built in a wider safety margin for exposure at those frequencies.

The U.S. has no consistent approach to the setting of standards. Individual Federal agencies have been setting their own limits, as have some individual states, counties and nongovernmental organizations. Different regulations apply in different situations and have varying degrees of legal force. Standards can be voluntary or mandatory and can apply to occupational groups or to the general public. A town can impose regulations that differ from those of its county, which itself may impose regulations that differ from those of the relevant Federal agency. Such divergence can force users of the electromagnetic spectrum to abide by differing standards; it also creates burdens on the various governmental agencies.

The U.S. Environmental Protection Agency has begun a process that may eventually result in a more uniform standard for the general public. Earlier this year it published for public comment three possible guidelines: one similar to (but not identical with) the ANSI guideline, another five times lower than the ANSI guideline and one 10 times lower than the ANSI guideline. The EPA is probably more than a year away from promulgating its final standard, but if a regulation is adopted, it is likely to preempt other efforts to set standards for the general population.

It is a curious fact that the Soviet Union and the Warsaw Pact countries have recently relaxed their standards. At least one Soviet commentator has stated that an acceptable level of exposure is .4 watt per kilogram for one hour, comparable to the present ANSI limit of .4 watt per kilogram averaged over six minutes. The current Soviet standards for occupational exposure to microwave energy are two watts per square meter for stationary sources and 20 watts per square meter for mobile sources, both averaged over one hour. The levels for the general public are lower: .1 watt per square meter. Thus the Soviet and the American standards are approaching agreement.

The setting of standards is a surprisingly difficult process. Some hazards of microwave energy, such as burns or heat stress, are well established; effective standards can be set for these. Whether other hazards can arise from exposure to low levels of microwave energy is a matter of conjecture, which depends on the interpretation of a large body of often unreliable reports. How to deal with the uncertainty, balancing the benefits of technology against the costs of possible hazards, is an urgent problem, not only with respect to microwave energy but also for many other environmental agents whose potential hazards have only begun to be explored.



MICROWAVE AUDITORY EFFECT, a clicking noise heard when microwave pulses are beamed at the head, was explained in 1974 by one of the authors (Foster). He proposed that the head responds to microwaves much as water does. When water absorbs pulses of microwaves, it undergoes a rapid but tiny rise in temperature. The resulting expansion of the fluid generates a sound wave that propagates from the surface of the water and can be heard as a click or recorded by a hydrophone. The same process, Foster suggested, occurs in tissues of the head on exposure to pulsed microwaves. The hearing of clicks is one of the few unequivocal effects of microwave energy at average power densities that could be below the ANSI standard. The auditory effect was first reported in 1947 but its mechanism was only explained years later. The effect is not now considered to represent a hazard.

THE AUTHORS

KENNETH R. FOSTER and ARTHUR W. GUY ("The Microwave Problem") are interested in the interaction of electromagnetic fields and living tissue. Foster received a Ph.D. in physics from Indiana University in 1971. He began his research on the biological effects of microwaves while serving in the U.S. Navy and has pursued it since 1976 as associate professor of bioengineering at the University of Pennsylvania. Guy has been at the University of Washington since his undergraduate days, with the exception of seven years spent doing research on antennas for the Boeing Aerospace Company. He is now professor of bioengineering and director of the bioelectromagnetics laboratory. From 1970 to 1982 he was chairman of the American National Standards Institute committee that wrote guidelines for human exposure to microwaves. One of his hobbies is operating a ham radio, which he uses for a daily "rag chew" with his father in Montana.

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Is Amateur Radio Hazardous to our Health?

What really was said about cancer rates and Amateur Radio, and what we can do about it.

By Ivan A. Shulman, MD, WC2S
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When it was reported in an Associated Press release that there was an increased rate of death due to certain types of cancer in Amateur Radio operators, this information was rapidly picked up by the radio community. As a physician who specializes in cancer surgery, I received many calls from amateur and nonamateur friends to find out more about what was going on and what I thought about it. As in many reports on medical topics in the lay literature and on television, there frequently is a difference between what is reported and what actually was said in medical articles, and this and other recent reports are no different.

After much time and consideration, several important concepts became apparent to me, and I hope that by making this report in *QST*, it will help us all to better understand what really was said, and what is known about the reported association of leukemias and other blood cancers with Amateur Radio. This article does not purport to completely cover all the important articles and research studies which have ever been written on the effects of electromagnetic radiation on human biology, but is instead, an effort to review that literature which might be useful to Amateur Radio operators interested in responding to the questions that have been asked.

Biologic Background

Radio-frequency waves are a form of electromagnetic waves, and in the frequencies of concern to Amateur Radio operators, these represent a form of nonionizing radiation. The terms ionizing and nonionizing radiation are frequently confused, and it is helpful to clarify what I mean by these terms early in our discussion.

Ionization occurs when there is enough energy in the radiation to displace an electron from an atom. Radiation that produces this effect has a very short wavelength, a high frequency and high energy level, and is typically that described as X-rays and gamma rays. Nonionizing radiation is otherwise known as infrared and radio-frequency waves, which are at a lower energy level, and have lower frequencies and longer wavelengths than ionizing radiation. Ionizing radiation is dangerous to living organisms in that it affects cellular elements such as DNA in the cell nucleus, leading to genetic damage in the individual cell, and mutations in future generations of cells. Although the energy level of nonionizing radiation is lower and thus may not affect large molecules or generate measurable amounts of heat in the same manner as ionizing radiation, there is substantial evidence that nonionizing radiation has subtle effects at a more basic cellular level, including effects on hormones, enzymes and the cooperative mechanisms involved in maintaining the integrity of intracellular systems.[1]

Experiments regarding the effects on human tissue of nonionizing electromagnetic fields have been conducted for many years.[2] The findings of these studies indicate that a modulated electromagnetic field, that is, one in which the energy is cycled on and off or is varied by intensity or frequency, has a greater inhibitory effect on the ability of cells in the body to communicate with each other than does a field in which the current remains at a steady and unmodulated strength.

Studies indicate that even in a weak electromagnetic field there is a modification of calcium binding at the cell membrane, as well as an alteration of a variety of calcium dependent enzyme systems which work between cells.[3] Experiments have noted that the effect on calcium flow in and out of cells is frequency dependent, and that curves can be drawn demonstrating these "frequency windows." Specifically, the combination of a very high or ultra high frequency carrier (147 or 450 MHz) modulated at specific extremely low frequencies (16, 40 or 60 Hz) has been studied and appears to be of biologic significance.[4]

Other studies have looked at the effects of electromagnetic energy on cells that have specific immune functions. An important type of white blood cell called a T-lymphocyte is involved in the recognition and destruction of foreign and malignant cells. There is evidence that the normal functioning of these cells is significantly reduced by electric fields that simulate 60-Hz high voltage power line fields and by weak microwave fields that are amplitude modulated at 60 Hz.[5,6] The mechanism of this process is not clear, but may also be related to interactions at the level of the cell membrane.

More rapidly dividing cells, such as those in the bone marrow or small intestine, are usually more sensitive to the effects of both ionizing and nonionizing radiation than are those which divide more slowly. Thus, it is rapidly dividing cells that are more likely to demonstrate changes in response to exposure to these types of energy. However, cells which divide more slowly have less of an ability to repair any damage done to them by exposure over a long period of time. It is important to recognize that these effects are not necessarily dependent on damage to DNA or other cellular markers.

Evidence at this time seems to suggest that an appropriate interpretation of this data is not that nonionizing energy necessarily causes cancer, but that it may act instead to promote the efficacy of other agents in doing so.

Previous Studies

In 1979, initial questions were raised regarding a positive relationship between high current electrical configurations in homes and the incidence of cancer deaths in children living in the Denver area.[7] Later, similar findings were noted for adults living near high current 60-Hz wiring as well.[8] Because of criticisms relating to the methodologies and assumptions used in these studies, other investigators looked at these same issues again, and came to similar conclusions.[9,10]

It had been reported as early as 1982 that there appeared to be an increased death rate due to leukemia in people who were exposed to magnetic and electric fields in the course of their work.[11,12] Additional articles appeared in 1983[13,14] and 1985[15-18] which also suggested that electrical workers in general were at an increased risk of leukemia and that electromagnetic fields might be a cause of this form of cancer. A time/effect relationship has also been suggested for certain forms of brain tumors and occupational exposure to microwave and radio-frequency electromagnetic radiation,[19,20] where the risk was 10 times as great in those workers who had industrial exposure to soldering fumes, solvents and a variety of other chemicals. Other reports have reviewed the possible relationship between spontaneous abortion rates and the use of electric blankets,[21] video display terminals,[22] and ceiling cable electric heat.[23] Cataract formation and damage to the retina has also been reported in humans exposed to high intensity electromagnetic fields and microwaves.[24]

Dr Milham's Study

The recent report which stirred up the most

[1]Notes appear on page 33.

concern because it made particular reference to Amateur Radio operators, appeared in the January 1988 issue of the *American Journal of Epidemiology*, a respected and prestigious medical publication.[25]

In 1982, Samuel Milham, Jr, MD, MPH, who works in the Epidemiology section of the Washington State Department of Social and Health Services, reported that a study of workers whose stated occupation on death certificate records suggested an exposure to electrical or magnetic fields had a higher rate due to leukemia.[26] In 1985, at the suggestion of an Amateur Radio operator (W2EVE), he looked at all the "Silent Keys" listings that appeared in *QST* and studied the cause of death of amateurs who died between the years 1971 and 1983 and who lived in Washington State and California at the time of their death.[27]

To simplify things slightly, only males were studied as there were very few women among these deaths. A total of 1691 death certificates were identified with these Silent Keys.

Using a standard statistical analytic technique called proportionate mortality radio (PMR), and an analysis of all US deaths as a comparison group, 12.6 of the 1691 amateurs should have died from leukemia. Instead, 24 deaths were observed with a statistical significance of $p < 0.01$, meaning that there was less than a 1 in 100 chance that this was a random occurrence.

In the largest study reported,[28] Milham has expanded on his original work. He first identified all licensed amateurs with addresses in California and Washington State. This was followed by a computerized and manual review of all deaths of persons whose complete names and date of births corresponded to the list of known amateurs for the period January 1, 1979 to June 16, 1984.

A total of 67,829 amateurs were identified and 2485 deaths were studied. Eighty-four percent (2083 of 2485) deaths occurred in California, so this study was weighted heavily towards the California experience. After making certain statistical adjustments, the overall death rate for amateurs was no different than it was for the population of both states at large. Likewise, the overall death rate for all forms of cancer among amateurs was not significantly different from the larger population.

However, within this cancer death rate, there was a definite disproportion of deaths due to cancers of "other" lymphatic tissues, such as multiple myeloma and non-Hodgkin's lymphomas. The death rate for all leukemias was only slightly, but not statistically significantly, increased. Among those leukemias, however, one form particularly (acute myelogenous leukemia) was significantly increased. It was concluded that the increased number of only these highly specific forms of blood disorders, and not others, suggests that a biologic cause and effect is present.

It was not possible to make a direct analysis of any occupational link with these excess deaths due to the fact that this information was readily available only for Washington State deaths. It should be noted that of these 402 deaths, 31 percent of the amateurs apparently worked in or about electromagnetic fields as technicians, radio operators or television repairmen. Of all deaths in Washington State during this time, only 3 percent of the population worked at these occupations.

In addition, among Washington State amateurs, 5 of the 11 deaths due to leukemias, lymphomas or multiple myeloma, were in people who had such occupational electromagnetic exposures. It was pointed out that workers in these occupations also were exposed to other possible hazards, such as fumes from solder and toxic chemicals such as the polychlorinated biphenyls (PCBs), and asbestos, any of which in themselves might conceivably cause cancer as well.

No other cause of death was noted to be higher than normal in the amateur population, and in fact, several important and common causes of death were less than what would be expected from the population as a whole. Deaths due to cancer of the pancreas and the lung, as well as all deaths due to respiratory diseases (pneumonia, asthma, emphysema), circulatory diseases (those of the heart and blood vessels) and accidents were less in amateurs as a group than in the overall population. It was even suggested that there are fewer cigarette smokers among members of the American Radio Relay League than in the general US population as a whole.

Milham concluded that Amateur Radio licensees in California and Washington State do have a higher death rate due to acute myelogenous leukemia, multiple myeloma and possibly other specific types of lymphoma. He felt that exposure to magnetic or electrical fields either as a consequence of work or hobby should be considered among the cause of these rates.

Comments on these Studies

It is important to recognize that studies based upon death certificate data alone are always subject to certain limitations. Data inaccuracies, from input as well as in coding, are not uncommon, and when one is measuring the incidence of small or rare occurrences, this may cause an inadvertent diminution or magnification of the determination of these occurrences. None of the studies discussed here look at an actual measurement of the electromagnetic or toxic chemical exposure that any of the deceased individuals may have had. The issue of what is called "confounding factors" such as the interaction of the effect of toxic chemicals and electromagnetic fields of different levels of energy is certainly unknown. As a result, statisticians may frequently differ on the interpretation of identical data.

On the basis of these research papers, however, it is now apparent that the data derived so far must be considered significant enough to support further research into both the epidemiology of and the biologic mechanisms involved in these effects. Some of that research is presently being done both in the United States and abroad and new articles are being published in the scientific literature frequently.

Exposure Standards

The question of exposure standards also deserves comment. In 1982, the American National Standards Institute (ANSI), a private, commercially sponsored organization, published a list of standards based upon the thermal effects of electromagnetic fields upon tissue.[29] There is much controversy regarding the validity of measuring this type of effect on biologic tissues as there is clear evidence that adverse tissue effects can occur without a detectable rise in temperature.[30] It should be noted that Australia, Sweden and the Eastern bloc countries as well as localities in the states of Oregon and Massachusetts have issued standards which recommend significantly lower exposure levels. Another voluntary standard has been proposed by the National Council for Radiation Protection and Measurement (NCRP), which is notably more stringent than the current ANSI standards.[31] ANSI is presently in the process of revising their standards.

It is interesting to note that the US Environmental Protection Agency has recently decided to defer the issuance of standards for exposure to electromagnetic fields under its RF Radiation Guidance Program for budgetary restrictions and other priorities. Despite the requests of the Federal Communication Commission, the National Association of Broadcasters, the Electromagnetic Energy Policy Alliance (of which the ARRL is a senior associate member) and other national organizations to complete this important work, the EPA has decided to put aside many years of effort on these guidelines and to focus its attention on other matters which it considers to be of greater public concern.

Hand-Held Radios

An article published recently studied the specific absorption rates in models of the human head exposed to hand-held radios operating in the 800-MHz band, which is where most cellular telephones are used.[32] The authors studied the RF energy absorbed by simulated tissues in the head (eye, brain, muscle, fat and bone) while holding the transmitter in vertical and tilted positions about the head. Also, a ½-wavelength antenna operated at 1.0 W power output was compared to a 5/8-wavelength antenna operated at 1.0 and 1.8 W. This study indicated the presence of a "hot spot" in the eye while using a ½-wavelength antenna, and one in the frontal portion of the brain while using a 5/8-wavelength antenna.

The authors concluded that if the transmitter is operated in a vertical position and is held at a distance of about 2 inches (5 cm) from the face during normal use, the specific absorption rates would not be significant enough to warrant concern, at least with reference to the present ANSI standards. These current ANSI standards essentially consider any device generating less than 7 watts output to be safe, an assumption with which almost all experts currently would not agree. Other studies using hand-helds operating at lower frequencies and different power outputs are being conducted and evaluated with refer-

ence to more stringent standards.

What Does This Mean?

What does all this really mean for us as amateurs? We all know that there are intrinsic risks in all activities that we do every day. How many of us still smoke, or are overweight or do not bother to fasten our seat belts in our cars? Knowing about risks only sometimes causes us to change our ways. As Amateur Radio operators we certainly do not have any hesitations about discussing and protecting ourselves from the dangers of high voltage circuitry. Nor do we shy away from trying to prevent the risk of accidental falls from roofs or antenna towers.

Likewise, we should recognize a relatively newly identified environmental hazard which may be significant to those of us even without occupational exposure to electromagnetic fields or toxic substances. No one is absolutely certain about what may be causing this increased proportion of special cancers. Therefore, prudence dictates that Amateur Radio operators should take those simple measures which decrease the possibility of our personal exposure to electromagnetic fields or toxics that we may contact as a consequence of our interest in Amateur Radio. Articles have been published in *QST* and other Amateur Radio publications regarding some precautions in the past.[33-37] This current list includes some recommendations which are new, particularly in view of recent information.

Preventive Measures

1) Do not stand or sit close to your power supplies or linear amplifiers while operating, even when they are in stand-by mode.

2) Stay at least 24 inches away from any power transformer, electrical fans or other source of high level 60-Hz magnetic fields while in operation.

3) Do not tune up or operate a high powered linear amplifier while the shields or covers are off.

4) Run your transmission lines away from where you or other people sit in or near your shack.

5) Properly terminated coaxial transmission feed lines should be used in preference to open-wire or end-fed antenna installations which come directly into the transmitter, as the RF radiated from a coaxial feed line is much lower.

6) Use common sense about placing all antennas well away from yourself and others, especially for VHF, UHF and particularly microwave applications. No one should be in the near field of an antenna.[38]

7) No person should be near any transmitting antenna while it is operating. This is especially true for all mobile or ground mounted vertical antennas. The use of indoor transmitting antennas which are close to people in a house or apartment should be considered.

8) Use the minimal power needed to make a QSO, especially if the antenna is less than 35 feet above the ground.

9) Hand-held radios should be used on the lowest power setting needed to carry out communications.

10) Hand-helds should be kept as far from the head as possible when operating. The use of a separate microphone or similar device is recommended.

11) Transmissions using a hand-held radio should be kept as short as possible.

12) Power density measurements should be made before running more than 25 watts in a VHF mobile installation, particularly if the antenna is rear-deck mounted and passengers may ride in the back seat. The safest mobile antenna location is in the center of the metal roof.

13) The development of an accurate inexpensive power-density meter would be of major benefit to the Amateur Radio community so that RF power-density measurements could be taken in all radio installations. Because of the current high cost of such devices, groups of amateurs or clubs may wish to purchase one and share in its use.[39]

14) Soldering should only be done in a well ventilated area. A small fan should be used to blow away toxic fumes.

15) When using toxic chemicals, such as when etching PC boards or repairing fiberglass, wear gloves and goggles, use proper tools, and avoid contact with any of the chemicals. If accidentally contaminated, wash off the compounds immediately with copious quantities of water.[40] Again, the importance of always working in a well ventilated area with personal protective covering cannot be overemphasized.

16) Hazardous chemicals, such as those in the PCB class, are used in some capacitors and dummy loads. Use extreme care in handling these materials, and consult with the appropriate local authorities to determine the proper means of disposing of these chemicals in an environmentally responsible way.

Some Observations

To my knowledge, no other established guidelines are available to prevent potentially harmful exposure. Therefore until such time as a clearer picture emerges, we should follow these simple common sense precautions.

There is no question that additional information is needed and will ultimately be forthcoming on this important issue. This data will certainly be difficult to interpret, and confusing to many of us, both in the amateur and nonamateur community. We must therefore be prepared to work together to arrive at reasonable conclusions and appropriate actions.[41]

In preparing this paper, I personally communicated with several of these experts in the field whose works are referenced below. All of these experts, including Dr Milham, agreed that none of them would have any hesitation regarding their own personal use of currently available Amateur Radio equipment, provided that it was properly installed and operated, and that the recommended precautions were followed.[42] Unanimously, they all feel that no one should stop operating because of concern for the possible risk of illness, as these risks appear to be so relatively low.

Am I worried? Absolutely not. With common sense and safe operating practices, I look forward to many more years of enjoyment and satisfaction as an Amateur Radio operator.

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An Amateur Radio operator continuously since 1963, Ivan Shulman says he was raised on Amateur Radio, and credits his late father W2SBX with getting him started. As a Fellow of the American College of Surgeons, Dr Shulman works as a general surgeon with a special interest in cancer of the thyroid, breast and gastrointestinal tract. In addition to his medical and radio activities, he has served as the physician for the Los Angeles Philharmonic on tours to Mexico, Japan, Korea and Europe. He also occasionally plays extra oboe with the orchestra as the need arises. When he manages to get on the air, he enjoys DX chasing and a good rag chew either DX on 20 meters or on UHF. In whatever spare time is left, he is busy introducing his wife and two young children to the ways of Amateur Radio.

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(continued from page 33)

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Ranking Possible Carcinogenic Hazards

BRUCE N. AMES,* RENAE MAGAW, LOIS SWIRSKY GOLD

This review discusses reasons why animal cancer tests cannot be used to predict absolute human risks. Such tests, however, may be used to indicate that some chemicals might be of greater concern than others. Possible hazards to humans from a variety of rodent carcinogens are ranked by an index that relates the potency of each carcinogen in rodents to the exposure in humans. This ranking suggests that carcinogenic hazards from current levels of pesticide residues or water pollution are likely to be of minimal concern relative to the background levels of natural substances, though one cannot say whether these natural exposures are likely to be of major or minor importance.

EPIDEMIOLOGISTS ESTIMATE THAT AT LEAST 70% OF HUMAN cancer would, in principle, be preventable if the main risk and antirisk factors could be identified (*1*). This is because the incidence of specific types of cancer differs markedly in different parts of the world where people have different life-styles. For example, colon and breast cancer, which are among the major types of cancer in the United States, are quite rare among Japanese in Japan, but not among Japanese-Americans. Epidemiologists are providing important clues about the specific causes of human cancer, despite inherent methodological difficulties. They have identified tobacco as an avoidable cause of about 30% of all U.S. cancer deaths and of an even larger number of deaths from other causes (*1, 2*). Less specifically, dietary factors, or their absence, have been suggested in many studies to contribute to a substantial proportion of cancer deaths, though the intertwined risk and antirisk factors are being identified only slowly (*1, 3, 4*). High fat intake may be a major contributor to colon cancer, though the evidence is not as definitive as that for the role of saturated fat in heart disease or of tobacco in lung cancer. Alcoholic beverage consumption, particularly by smokers, has been estimated to contribute to about 3% of U.S. cancer deaths (*1*) and to an even larger number of deaths from other causes. Progress in prevention has been made for some occupational factors, such as asbestos, to which workers used to be heavily exposed, with delayed effects that still contribute to about 2% of U.S. cancer deaths (*1, 5*). Prevention may also become possible for hormone-related cancers such as breast cancer (*1, 6*), or virus-related cancers such as liver cancer (hepatitis B) and cancer of the cervix (papilloma virus HPV16) (*1, 7*).

Animal bioassays and in vitro studies are also providing clues as to which carcinogens and mutagens might be contributing to human cancer. However, the evaluation of carcinogenicity in rodents is expensive and the extrapolation to humans is difficult (*8–11*). We will use the term "possible hazard" for estimates based on rodent cancer tests and "risk" for those based on human cancer data (*10*).

Extrapolation from the results of rodent cancer tests done at high doses to effects on humans exposed to low doses is routinely attempted by regulatory agencies when formulating policies attempting to prevent future cancer. There is little sound scientific basis for this type of extrapolation, in part due to our lack of knowledge about mechanisms of cancer induction, and it is viewed with great unease by many epidemiologists and toxicologists (*5, 9–11*). Nevertheless, to be prudent in regulatory policy, and in the absence of good human data (almost always the case), some reliance on animal cancer tests is unavoidable. The best use of them should be made even though few, if any, of the main avoidable causes of human cancer have typically been the types of man-made chemicals that are being tested in animals (*10*). Human cancer may, in part, involve agents such as hepatitis B virus, which causes chronic inflammation; changes in hormonal status; deficiencies in normal protective factors (such as selenium or β-carotene) against endogenous carcinogens (*12*); lack of other anticarcinogens (such as dietary fiber or calcium) (*4*); or dietary imbalances such as excess consumption of fat (*3, 4, 12*) or salt (*13*).

There is a need for more balance in animal cancer testing to emphasize the foregoing factors and natural chemicals as well as synthetic chemicals (*12*). There is increasing evidence that our normal diet contains many rodent carcinogens, all perfectly natural or traditional (for example, from the cooking of food) (*12*), and that no human diet can be entirely free of mutagens or agents that can be carcinogenic in rodent systems. We need to identify the important causes of human cancer among the vast number of minimal risks. This requires knowledge of both the amounts of a substance to which humans are exposed and its carcinogenic potency.

Animal cancer tests can be analyzed quantitatively to give an estimate of the relative carcinogenic potencies of the chemicals tested. We have previously published our Carcinogenic Potency Database, which showed that rodent carcinogens vary in potency by more than 10 millionfold (*14*).

This article attempts to achieve some perspective on the plethora of possible hazards to humans from exposure to known rodent carcinogens by establishing a scale of the possible hazards for the amounts of various common carcinogens to which humans might be chronically exposed. We view the value of our calculations not as providing a basis for absolute human risk assessment, but as a guide to priority setting. One problem with this type of analysis is that few of the many natural chemicals we are exposed to in very large amounts (relative to synthetic chemicals) have been tested in animals for carcinogenicity. Thus, our knowledge of the background level of human exposure to animal carcinogens is fragmentary, biased in favor of synthetic chemicals, and limited by our lack of knowledge of human exposures.

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Ranking of Possible Carcinogenic Hazards

Since carcinogens differ enormously in potency, a comparison of possible hazards from various carcinogens ingested by humans must take this into account. The measure of potency that we have developed, the TD_{50}, is the daily dose rate (in milligrams per kilogram) to halve the percent of tumor-free animals by the end of a standard lifetime (*14*). Since the TD_{50} (analogous to the LD_{50}) is a dose rate, the lower the TD_{50} value the more potent the carcinogen. To calculate our index of possible hazard we express each human exposure (daily lifetime dose in milligrams per kilogram) as a percentage of the rodent TD_{50} dose (in milligrams per kilogram) for each carcinogen. We call this percentage HERP [*H*uman *E*xposure dose/*R*odent *P*otency dose]. The TD_{50} values are taken from our ongoing Carcinogenic Potency Database (currently 3500 experiments on 975 chemicals), which reports the TD_{50} values estimated from experiments in animals (*14*). Human exposures have been estimated from the literature as indicated. As rodent data are all calculated on the basis of lifetime exposure at the indicated daily dose rate (*14*), the human exposure data are similarly expressed as lifelong daily dose rates even though the human exposure is likely to be less than daily for a lifetime.

It would be a mistake to use our HERP index as a direct estimate of human hazard. First, at low dose rates human susceptibility may differ systematically from rodent susceptibility. Second, the general shape of the dose-response relationship is not known. A linear dose response has been the dominant assumption in regulating carcinogens for many years, but this may not be correct. If the dose responses are not linear but are actually quadratic or hockey-stick shaped or show a threshold, then the actual hazard at low dose rates might be much less than the HERP values would suggest. An additional difficulty is that it may be necessary to deal with carcinogens that differ in their mechanisms of action and thus in their dose-response relationship. We have therefore put an asterisk next to HERP values for carcinogens that do not appear to be active through a genotoxic (DNA damaging or mutagenic) mechanism (*15*) so that comparisons can be made within the genotoxic or nongenotoxic classes.

Table 1 presents our HERP calculations of possible cancer hazards in order to compare them within several categories so that, for example, pollutants of possible concern can be compared to natural carcinogens in the diet. A convenient reference point is the possible hazard from the carcinogen chloroform in a liter of average (U.S.) chlorinated tap water, which is close to a HERP of 0.001%. Chloroform is a by-product of water chlorination, which protects us from pathogenic viruses and bacteria.

Contaminated water. The possible hazards from carcinogens in contaminated well water [for example, Santa Clara ("Silicon") Valley, California, or Woburn, Massachusetts] should be compared to the possible hazard of ordinary tap water (Table 1). Of 35 wells shut down in Santa Clara Valley because of their supposed carcinogenic hazard, only two have HERP values greater than ordinary tap water. Well water is not usually chlorinated and typically lacks the chloroform present in chlorinated tap water. Water from the most polluted well (HERP = 0.004% per liter for trichloroethylene), as indicated in Table 1, has a HERP value orders of magnitude less than for the carcinogens in an equal volume of cola, beer, or wine. Its HERP value is also much lower than that of many of the common natural foods that are listed in Table 1, such as the average peanut butter sandwich. Caveats for any comparisons are given below. Since the consumption of tap water is only about 1 or 2 liters per day, the animal evidence provides no good reason to expect that chlorination of water or current levels of man-made pollution of water pose a significant carcinogenic hazard.

Pesticide residues. Intake of man-made pesticide residues from food in the United States, including residues of industrial chemicals such as polychlorinated biphenyls (PCBs), averages about 150 μg/day. Most (105 μg) of this intake is composed of three chemicals (ethylhexyl diphenyl phosphate, malathion, and chlorpropham) shown to be noncarcinogenic in tests in rodents (*16*). A carcinogenic pesticide residue in food of possible concern is DDE, the principal metabolite (>90%) of DDT (*16*). The average U.S. daily intake of DDE from DDT (HERP = 0.0003%) is equivalent to the HERP of the chloroform in one glass of tap water and thus appears to be insignificant compared to the background of natural carcinogens in our diet (Table 1). Even daily consumption of 100 times the average intake of DDE/DDT or PCBs would produce a possible hazard that is small compared to other common exposures shown in Table 1.

Nature's pesticides. We are ingesting in our diet at least 10,000 times more by weight of natural pesticides than of man-made pesticide residues (*12*). These are natural "toxic chemicals" that have an enormous variety of chemical structures, appear to be present in all plants, and serve to protect plants against fungi, insects, and animal predators (*12*). Though only a few are present in each plant species, they commonly make up 5 to 10% of the plant's dry weight (*12*). There has been relatively little interest in the toxicology or carcinogenicity of these compounds until quite recently, although they are by far the main source of "toxic chemicals" ingested by humans. Only a few dozen of the thousands present in the human diet have been tested in animal bioassays, and only some of these tests are adequate for estimating potency in rodents (*14*). A sizable proportion of those that have been tested are carcinogens, and many others have been shown to be mutagens (*12*), so it is probable that many more will be found to be carcinogens if tested. Those shown in Table 1 are: estragole (HERP = 0.1% for a daily 1 g of dried basil), safrole (HERP = 0.2% for a daily natural root beer), symphytine (a pyrrolizidine alkaloid, 0.03% for a daily cup of comfrey tea), comfrey tablets sold in health food stores (6.2% for a daily dose), hydrazines in mushrooms (0.1% for one daily raw mushroom), and allyl isothiocyanate (0.07% for a daily 5 g of brown mustard).

Plants commonly produce very much larger amounts of their natural toxins when damaged by insects or fungi (*12*). For example, psoralens, light-activated carcinogens in celery, increase 100-fold when the plants are damaged by mold and, in fact, can cause an occupational disease in celery-pickers and in produce-checkers at supermarkets (*12, 17*).

Molds synthesize a wide variety of toxins, apparently as antibiotics in the microbiological struggle for survival: over 300 mycotoxins have been described (*18*). They are common pollutants of human food, particularly in the tropics. A considerable percentage of those tested have been shown to be mutagens and carcinogens: some, such as aflatoxin and sterigmatocystin, are among the most potent known rodent carcinogens. The potency of aflatoxin in different species varies widely; thus, a bias may exist as the HERP uses the most sensitive species. The aflatoxin content of U.S. peanut butter averages 2 ppb, which corresponds to a HERP of 0.03% for the peanut butter in an average sandwich (Table 1). The Food and Drug Administration (FDA) allows ten times this level (HERP = 0.3%), and certain foods can often exceed the allowable limit (*18*). Aflatoxin contaminates wheat, corn (perhaps the main source of dietary aflatoxin in the United States), and nuts, as well as a wide variety of stored carbohydrate foodstuffs. A carcinogenic, though less potent, metabolite of aflatoxin is found in milk from cows that eat moldy grain.

There is epidemiologic evidence that aflatoxin is a human carcinogen. High intake in the tropics is associated with a high rate of liver cancer, at least among those chronically infected with the hepatitis B

us (*19, 20*). Considering the potency of those mold toxins that ve been tested and the widespread contamination of food with molds, they may represent the most significant carcinogenic pollution of the food supply in developing countries. Such pollution is ch less severe in industrialized countries, due to refrigeration and modern techniques of agriculture and storage, including use of synthetic pesticides and fumigants.

Preparation of foods and beverages can also produce carcinogens. Alcohol has been shown to be a human carcinogen in numerous epidemiologic studies (*1, 21*). Both alcohol and acetaldehyde, its

ble 1. Ranking possible carcinogenic hazards. *Potency of carcinogens*: A number in parentheses indicates a TD_{50} value not used in HERP calculation because s the less sensitive species; (−) = negative in cancer test. (+) = positive for carcinogenicity in test(s) not suitable for calculating a TD_{50}; (?) = is not quately tested for carcinogenicity. TD_{50} values shown are averages calculated by taking the harmonic mean of the TD_{50}'s of the positive tests in that species from the Carcinogenic Potency Database. Results are similar if the lowest TD_{50} value (most potent) is used instead. For each test the target site with the lowst TD_{50} value has been used. The average TD_{50} has been calculated separately for rats and mice, and the more sensitive species is used for calculating the pose hazard. The database, with references to the source of the cancer tests, is complete for tests published through 1984 and for the National Toxicology gram bioassays through June 1986 (*14*). We have not indicated the route of exposure or target sites or other particulars of each test, although these are reported in the database. *Daily human exposure*: We have tried to use average or reasonable daily intakes to facilitate comparisons. In several cases, such as contaminated well water or factory exposure to EDB, this is difficult to determine, and we give the value for the worst found and indicate pertinent rmation in the References and Notes. The calculations assume a daily dose for a lifetime; where drugs are normally taken for only a short period we have cketed the HERP value. For inhalation exposures we assume an inhalation of 9,600 liters per 8 hours for the workplace and 10,800 liters per 14 hours for oor air at home. *Possible hazard*: The amount of rodent carcinogen indicated under carcinogen dose is divided by 70 kg to give a milligram per kilogram of human exposure, and this human dose is given as the percentage of the TD_{50} dose in the rodent (in milligrams per kilogram) to calculate the *H*uman *E*xposure/Rodent *P*otency index (HERP).

Possible hazard: HERP (%)	Daily human exposure	Carcinogen dose per 70-kg person	Potency of carcinogen: TD_{50} (mg/kg)		References
			Rats	Mice	
		Environmental pollution			
0.001*	Tap water, 1 liter	Chloroform, 83 μg (U.S. average)	(119)	90	*96*
0.004*	Well water, 1 liter contaminated (worst well in Silicon Valley)	Trichloroethylene, 2800 μg	(−)	941	*97*
0.0004*	Well water, 1 liter contaminated, Woburn	Trichloroethylene, 267 μg	(−)	941	*98*
0.0002*		Chloroform, 12 μg	(119)	90	
0.0003*		Tetrachloroethylene, 21 μg	101	(126)	
0.008*	Swimming pool, 1 hour (for child)	Chloroform, 250 μg (average pool)	(119)	90	*99*
0.6	Conventional home air (14 hour/day)	Formaldehyde, 598 μg	1.5	(44)	*100*
0.004		Benzene, 155 μg	(157)	53	
2.1	Mobile home air (14 hour/day)	Formaldehyde, 2.2 mg	1.5	(44)	*28*
		Pesticide and other residues			
0.0002*	PCBs: daily dietary intake	PCBs, 0.2 μg (U.S. average)	1.7	(9.6)	*101*
0.0003*	DDE/DDT: daily dietary intake	DDE, 2.2 μg (U.S. average)	(−)	13	*16*
0.0004	EDB: daily dietary intake (from grains and grain products)	Ethylene dibromide, 0.42 μg (U.S. average)	1.5	(5.1)	*102*
		Natural pesticides and dietary toxins			
0.003	Bacon, cooked (100 g)	Dimethylnitrosamine, 0.3 μg	(0.2)	0.2	*40*
0.006		Diethylnitrosamine, 0.1 μg	0.02	(+)	
0.003	Sake (250 ml)	Urethane, 43 μg	(41)	22	*24*
0.03	Comfrey herb tea, 1 cup	Symphytine, 38 μg (750 μg of pyrrolizidine alkaloids)	1.9	(?)	*103*
0.03	Peanut butter (32 g; one sandwich)	Aflatoxin, 64 ng (U.S. average, 2 ppb)	0.003	(+)	*18*
0.06	Dried squid, broiled in gas oven (54 g)	Dimethylnitrosamine, 7.9 μg	(0.2)	0.2	*37*
0.07	Brown mustard (5 g)	Allyl isothiocyanate, 4.6 mg	96	(−)	*47*
0.1	Basil (1 g of dried leaf)	Estragole, 3.8 mg	(?)	52	*48*
0.1	Mushroom, one raw (15 g) (*Agaricus bisporus*)	Mixture of hydrazines, and so forth	(?)	20,300	*104*
0.2	Natural root beer (12 ounces; 354 ml) (now banned)	Safrole, 6.6 mg	(436)	56	*105*
0.008	Beer, before 1979 (12 ounces; 354 ml)	Dimethylnitrosamine, 1 μg	(0.2)	0.2	*38*
2.8*	Beer (12 ounces; 354 ml)	Ethyl alcohol, 18 ml	9110	(?)	*23*
4.7*	Wine (250 ml)	Ethyl alcohol, 30 ml	9110	(?)	*23*
6.2	Comfrey-pepsin tablets (nine daily)	Comfrey root, 2700 mg	626	(?)	*103*
1.3	Comfrey-pepsin tablets (nine daily)	Symphytine, 1.8 mg	1.9	(?)	
		Food additives			
0.0002	AF-2: daily dietary intake before banning	AF-2 (furylfuramide), 4.8 μg	29	(131)	*44*
0.06*	Diet Cola (12 ounces; 354 ml)	Saccharin, 95 mg	2143	(−)	*106*
		Drugs			
[0.3]	Phenacetin pill (average dose)	Phenacetin, 300 mg	1246	(2137)	*51*
[5.6]	Metronidazole (therapeutic dose)	Metronidazole, 2000 mg	(542)	506	*107*
[4]	Isoniazid pill (prophylactic dose)	Isoniazid, 300 mg	(150)	30	*108*
*	Phenobarbital, one sleeping pill	Phenobarbital, 60 mg	(+)	5.5	*50*
*	Clofibrate (average daily dose)	Clofibrate, 2000 mg	169	(?)	*52*
		Occupational exposure			
5.8	Formaldehyde: Workers' average daily intake	Formaldehyde, 6.1 mg	1.5	(44)	*109*
	EDB: Workers' daily intake (high exposure)	Ethylene dibromide, 150 mg	1.5	(5.1)	*55*

*Asterisks indicate HERP from carcinogens thought to be nongenotoxic.

major metabolite, are carcinogens in rats (*22, 23*). The carcinogenic potency of ethyl alcohol in rats is remarkably low (*23*), and it is among the weakest carcinogens in our database. However, human intake of alcohol is very high (about 18 g per beer), so that the possible hazards shown in Table 1 for beer and wine are large (HERP = 2.8% for a daily beer). The possible hazard of alcohol is enormous relative to that from the intake of synthetic chemical residues. If alcohol (*20*), trichloroethylene, DDT, and other presumptive nongenotoxic carcinogens are active at high doses because they are tumor promoters, the risk from low doses may be minimal.

Other carcinogens are present in beverages and prepared foods. Urethane (ethyl carbamate), a particularly well-studied rodent carcinogen, is formed from ethyl alcohol and carbamyl phosphate during a variety of fermentations and is present in Japanese sake (HERP = 0.003%), many types of wine and beer, and in smaller amounts in yogurt and bread (*24*). Another fermentation product, the dicarbonyl aldehyde methylglyoxal, is a potent mutagen and was isolated as the main mutagen in coffee (about 250 μg in one cup). It was recently shown to be a carcinogen, though not in a test suitable for calculating a TD_{50} (*25*). Methylglyoxal is also present in a variety of other foods, such as tomato puree (*25, 26*). Diacetyl (2,3-butanedione), a closely related dicarbonyl compound, is a fermentation product in wine and a number of other foods and is responsible for the aroma of butter. Diacetyl is a mutagen (*27*) but has not been tested for carcinogenicity.

Formaldehyde, another natural carcinogenic and mutagenic aldehyde, is also present in many common foods (*22, 26–28*). Formaldehyde gas caused cancer only in the nasal turbinates of the nose-breathing rodents and even though formaldehyde is genotoxic, the dose response was nonlinear (*28, 29*). Hexamethylenetetramine, which decomposes to formaldehyde in the stomach, was negative in feeding studies (*30*). The effects of oral versus inhalation exposure for formaldehyde remain to be evaluated more thoroughly.

As formaldehyde is almost ubiquitous in foods, one can visualize various formaldehyde-rich scenarios. Daily consumption of shrimp (HERP = 0.09% per 100 g) (*31*), a sandwich (HERP of two slices of bread = 0.4%) (*22*), a cola (HERP = 2.7%) (*32*), and a beer (HERP = 0.2%) (*32*) in various combinations could provide as much formaldehyde as living in some mobile homes (HERP = 2.1%; Table 1). Formaldehyde is also generated in animals metabolically, for example, from methoxy compounds that humans ingest in considerable amounts from plants. The level of formaldehyde reported in normal human blood is strikingly high (about 100 μ*M* or 3000 ppb) (*33*) suggesting that detoxification mechanisms are important.

The cooking of food generates a variety of mutagens and carcinogens. Nine heterocyclic amines, isolated on the basis of their mutagenicity from proteins or amino acids that were heated in ways that occur in cooking, have now been tested; all have been shown to be potent carcinogens in rodents (*34*). Many others are still being isolated and characterized (*34*). An approximate HERP of 0.02% has been calculated by Sugimura *et al.* for the daily intake of these nine carcinogens (*34*). Three mutagenic nitropyrenes present in diesel exhaust have now been shown to be carcinogens (*35*), but the intake of these carcinogenic nitropyrenes has been estimated to be much higher from grilled chicken than from air pollution (*34, 36*). The total amount of browned and burnt material eaten in a typical day is at least several hundred times more than that inhaled from severe air pollution (*12*).

Gas flames generate NO_2, which can form both the carcinogenic nitropyrenes (*35, 36*) and the potently carcinogenic nitrosamines in food cooked in gas ovens, such as fish or squid (HERP = 0.06%; Table 1) (*37*). We suspect that food cooked in gas ovens may be a major source of dietary nitrosamines and nitropyrenes, though it is not clear how significant a risk these pose. Nitrosamines were ubiquitous in beer and ale (HERP = 0.008%) and were formed from NO_2 in the gas flame–heated air used to dry the malt. However, the industry has switched to indirect heating, which resulted in markedly lower levels (<1 ppb) of dimethylnitrosamine (*38*). The dimethylnitrosamine found in human urine is thought to be formed in part from NO_2 inhaled from kitchen air (*39*). Cooked bacon contains several nitrosamines (HERP = 0.009%) (*40*).

Oxidation of fats and vegetable oils occurs during cooking and also spontaneously if antioxidant levels are low. The result is the formation of peroxides, epoxides, and aldehydes, all of which appear to be rodent carcinogens (*8, 12, 27*). Fatty acid hydroperoxides (present in oxidized oils) and cholesterol epoxide have been shown to be rodent carcinogens (though not in tests suitable for calculating a TD_{50}). Dried eggs contain about 25 ppm of cholesterol epoxide (a sizable amount), a result of the oxidation of cholesterol by the NO_2 in the drying air that is warmed by gas flames (*12*).

Normal oxidation reactions in fruit (such as browning in a cut apple) also involve production of peroxides. Hydrogen peroxide is a mutagenic rodent carcinogen that is generated by oxidation of natural phenolic compounds that are quite widespread in edible plants. A cup of coffee contains about 750 μg of hydrogen peroxide (*25*); however, since hydrogen peroxide is a very weak carcinogen (similar in potency to alcohol), the HERP for drinking a daily cup of coffee would be very low [comparable to DDE/DDT, PCBs, or ethylene dibromide (EDB) dietary intakes]. Hydrogen peroxide is also generated in our normal metabolism; human blood contains about 5 μ*M* hydrogen peroxide and 0.3 μ*M* of the cholesterol ester of fatty acid hydroperoxide (*41*). Endogenous oxidants such as hydrogen peroxide may make a major contribution to cancer and aging (*42*).

Caloric intake, which could be considered the most striking rodent carcinogen ever discovered, is discussed remarkably little in relation to human cancer. It has been known for about 40 years that increasing the food intake in rats and mice by about 20% above optimal causes a remarkable decrease in longevity and a striking increase in endocrine and mammary tumors (*43*). In humans, obesity (associated with high caloric intake) leads to increased levels of circulating estrogens, a significant cause of endometrial and gall bladder cancer. The effects of moderate obesity on other types of human cancer are less clear (*1*).

Food additives are currently screened for carcinogenicity before use if they are synthetic compounds. AF-2 (HERP = 0.0002%), a food preservative, was banned in Japan (*44*). Saccharin (HERP = 0.06%) is currently used in the United States (the dose-response in rats, however, is clearly sublinear) (*45*). The possible hazard of diethylstilbestrol residues in meat from treated farm animals seems miniscule relative to endogenous estrogenic hormones and plant estrogens (*46*). Some natural carcinogens are also widely used as additives, such as allyl isothiocyanate (*47*), estragole (*48*), and alcohol (*23*).

Air pollution. A person inhales about 20,000 liters of air in a day; thus, even modest contamination of the atmosphere can result in inhalation of appreciable doses of a pollutant. This can be seen in the possible hazard in mobile homes from formaldehyde (HERP = 2.1%) or in conventional homes from formaldehyde (HERP = 0.6%) or benzene (HERP = 0.004%; Table 1). Indoor air pollution is, in general, worse than outdoor air pollution, partly because of cigarette smoke. The most important indoor air pollutant may be radon gas. Radon is a natural radioactive gas that is present in the soil, gets trapped in houses, and gives rise to radioactive decay products that are known to be carcinogenic for humans (*49*). It has been estimated that in 1 million homes in the United States the level of exposure to products of radon decay may be higher than that

ceived by today's uranium miners. Two particularly contaminated uses were found that had a risk estimated to be equivalent to receiving about 1200 chest x-rays a day (*49*). Approximately 10% of e lung cancer in the United States has been tentatively attributed radon pollution in houses (*49*). Many of these cancers might be preventable since the most hazardous houses can be identified and modified to minimize radon contamination.

General outdoor air pollution appears to be a small risk relative to e pollution inhaled by a smoker: one must breathe Los Angeles smog for a year to inhale the same amount of burnt material that a moker (two packs) inhales in a day (*12*), though air pollution is haled starting from birth. It is difficult to determine cancer risk om outdoor air pollution since epidemiologists must accurately control for smoking and radon.

Some common drugs shown in Table 1 give fairly high HERP rcentages, primarily because the dose ingested is high. However, since most medicinal drugs are used for only short periods while the HERP index is a daily dose rate for a lifetime, the possible hazard ould usually be markedly less. We emphasize this in Table 1 by acketing the numbers for these shorter exposures. Phenobarbital (HERP = 16%) was investigated thoroughly in humans who had ken it for decades, and there was no convincing evidence that it used cancer (*50*). There is evidence of increased renal cancer in long-term human ingestion of phenacetin, an analgesic (*51*). Acetaminophen, a metabolite of phenacetin, is one of the most widely ed over-the-counter pain killers. Clofibrate (HERP = 17%) is ed as a hypolipidemic agent and is thought to be carcinogenic in rodents because it induces hydrogen peroxide production through roxisome proliferation (*52*).

Occupational exposures can be remarkably high, particularly for olatile carcinogens, because about 10,000 liters of air are inhaled in a working day. For formaldehyde, the exposure to an average orker (HERP = 5.8%) is higher than most dietary intakes. For a mber of volatile industrial carcinogens, the ratio of the permitted exposure limit [U.S. Occupational Safety and Health Administration (OSHA)] in milligrams per kilogram to the TD_{50} has been lculated; several are close to the TD_{50} in rodents and about two-irds have permitted HERP values >1% (*53*). The possible hazard estimated for the actual exposure levels of the most heavily exposed DB workers is remarkably high, HERP = 140% (Table 1). hough the dose may have been somewhat overestimated (*54*), it was still comparable to the dose causing cancer in half the rodents. An epidemiologic study of these heavily exposed EDB workers who haled EDB for over a decade did not show any increase in cancer, hough because of the limited duration of exposure and the relatively small numbers of people monitored the study would not ave detected a small effect (*54, 55*). OSHA still permits exposures ove the TD_{50} level. California, however, lowered the permitted evel over 100-fold in 1981. In contrast with these heavy workplace exposures, the Environmental Protection Agency (EPA) has banned e use of EDB for fumigation because of the residue levels found in rain (HERP = 0.0004%).

Uncertainties in Relying on Animal Cancer Tests for Human Prediction

Species variation. Though we list a possible hazard if a chemical is a rcinogen in a rat but not in a mouse (or vice versa), this lack of greement raises the possibility that the risk to humans is nonexistent. Of 392 chemicals in our database tested in both rats and mice, 26 were carcinogens in at least one test, but 96 of these were ositive in the mouse and negative in the rat or vice versa (*56*). This discordance occurs despite the fact that rats and mice are very closely related and have short life-spans. Qualitative extrapolation of cancer risks from rats or mice to humans, a very dissimilar long-lived species, is unlikely to be as reliable. Conversely, important human carcinogens may not be detected in standard tests in rodents; this was true for a long time for both tobacco smoke and alcohol, the two largest identified causes of neoplastic death in the United States.

For many of the chemicals considered rodent carcinogens, there may be negative as well as positive tests. It is difficult to deal with negative results satisfactorily for several reasons, including the fact that some chemicals are tested only once or twice, while others are tested many times. The HERP index ignores negative tests. Where there is species variation in potency, use of the more sensitive species, as is generally done and as is done here, could introduce a tendency to overestimate possible hazards; however, for most chemicals that are positive in both species, the potency is similar in rats and mice (*57*). The HERP may provide a rough correlate of human hazard from chemical exposure; however, for a given chemical, to the extent that the potency in humans differs from the potency in rodents, the relative hazard would be different.

Quantitative uncertainties. Quantitative extrapolation from rodents to humans, particularly at low doses, is guesswork that we have no way of validating (*1, 5, 10, 11, 58*). It is guesswork because of lack of knowledge in at least six major areas: (i) the basic mechanisms of carcinogenicity; (ii) the relation of cancer, aging, and life-span (*1, 10, 42, 59*); (iii) the timing and order of the steps in the carcinogenic process that are being accelerated; (iv) species differences in metabolism and pharmacokinetics; (v) species differences in anticarcinogens and other defenses (*1, 60*); and (vi) human heterogeneity—for example, pigmentation affects susceptibility to skin cancer from ultraviolet light. These sources of uncertainty are so numerous, and so substantial, that only empirical data will resolve them, and little of this is available.

Uncertainties due to mechanism in multistage carcinogenesis. Several steps (stages) are involved in chemical carcinogenesis, and the dose-response curve for a carcinogen might depend on the particular stage(s) it accelerates (*58*), with multiplicative effects if several stages are affected. This multiplicative effect is consistent with the observation in human cancer that synergistic effects are common. The three steps of carcinogenesis that have been analyzed in most detail are initiation (mutation), promotion, and progression, and we discuss these as an aid to understanding aspects of the dose-response relation.

Mutation (or DNA damage) as one stage of the carcinogenic process is supported by various lines of evidence: association of active forms of carcinogens with mutagens (*61*), the changes in DNA sequence of oncogenes (*62*), genetic predisposition to cancer in human diseases such as retinoblastoma (*63*) or DNA-repair deficiency diseases such as xeroderma pigmentosum (*64*). The idea that genotoxic carcinogens might show a linear dose-response might be plausible if only the mutation step of carcinogenesis was accelerated and if the induction of repair and defense enzymes were not significant factors (*65*).

Promotion, another step in carcinogenesis, appears to involve cell proliferation, or perhaps particular types of cell proliferation (*66*), and dose-response relations with apparent thresholds, as indicated by various lines of evidence: (i) The work of Trosko *et al.* (*67*) on promotion of carcinogenesis due to interference with cell-cell communication, causing cell proliferation. (ii) Rajewsky's and other work indicating initiation by some carcinogenic agents appears to require proliferating target cells (*68*). (iii) The work of Farber *et al.* (*69*) on liver carcinogenesis supports the idea that cell proliferation (caused by partial hepatectomy or cell killing) can be an important aspect of hepatocarcinogenesis. They have also shown for several chemicals that hepatic cell killing shows a toxic threshold with dose. (iv) Work on carcinogenesis in the pancreas, bladder and stomach

(*70*), and other tissues (*58*) is also consistent with results on the liver (*71, 72*) though the effect of cell proliferation might be different in tissues that normally proliferate. (v) The work of Mirsalis *et al.* (*71*) suggests that a variety of nongenotoxic agents are hepatocarcinogens in the B6C3F1 mouse (commonly used in cancer tests) because of their toxicity. Other studies on chloroform and trichloroethylene also support this interpretation (*72, 73*). Cell proliferation resulting from the cell killing in the mouse liver shows a threshold with dose (*71*). Also relevant is the extraordinarily high spontaneous rates of liver tumors (21% carcinomas, 10% adenomas) in the male B6C3F1 mouse (*74*). These spontaneous tumors have a mutant *ras* oncogene, and thus the livers in these mice appear to be highly initiated (mutated) to start with (*75*). (vi) Oncogenes: As Weinberg (*62*) has pointed out, "Oncogene-bearing cells surrounded by normal neighbors do not grow into a large mass if they carry only a single oncogene. But if the normal neighbors are removed . . . by killing them with a cytotoxic drug . . . then a single oncogene often suffices." (vii) Cell killing, as well as mutation, appears to be an important aspect of radiation carcinogenesis (*76*).

Promotion has also been linked to the production of oxygen radicals, such as from phagocytic cells (*77*). Since chronic cell killing would usually involve inflammatory reactions caused by neutrophils, one would commonly expect chemicals tested at the maximally tolerated dose (MTD) to be promoters because of the chronic inflammation.

Progression, another step in carcinogenesis, leading to selection for invasiveness and metastases, is not well understood but can be accelerated by oxygen radicals (*78*).

Chronic cell toxicity caused by dosing at the MTD in rodent cancer bioassays thus not only could cause inflammation and cell proliferation, but also should be somewhat mutagenic and clastogenic to neighboring cells because of the release of oxygen radicals from phagocytosis (*12, 79, 80*). The respiratory burst from phagocytic neutrophils releases the same oxidative mutagens produced by radiation (*77, 79*). Thus, animal cancer tests done at the MTD of a chemical might commonly stimulate all three steps in carcinogenesis and be positive because the chemical caused chronic cell killing and inflammation with some mutagenesis. Some of the considerable human evidence for chronic inflammation contributing to carcinogenesis and also some evidence for and against a general effect of inflammation and cytotoxicity in rodent carcinogenesis have been discussed (*81*).

Another set of observations may also bear on the question of toxicity and extrapolation. Wilson, Crouch, and Zeise (*82*) have pointed out that among carcinogens one can predict the potency in high-dose animal cancer experiments from the toxicity (the LD_{50}) of the chemical, though one cannot predict whether the substance is a carcinogen. We have shown that carcinogenic potency values are bounded by the MTD (*57*). The evidence from our database suggests that the relationship between TD_{50} and MTD has a biological as well as a statistical basis (*57*). We postulate that a just sublethal level of a carcinogen causes cell death, which allows neighboring cells to proliferate, and also causes oxygen radical production from phagocytosis and thus chronic inflammation, both important aspects of the carcinogenic process (*57*). The generality of this relationship and its basis needs further study.

If most animal cancer tests done at the MTD are partially measuring cell killing and consequent cell proliferation and phagocytic oxygen radical damage as steps in the carcinogenic process, one might predict that the dose-response curves would generally be nonlinear. For those experiments in our database for which life table data (*14*) were available, a detailed analysis (*83*) shows that the dose-response relationships are more often consistent with a quadratic (or cubic) model than with a linear model.

Experimentally, it is very difficult to discriminate between the various extrapolation models at low doses (*11, 58*). However, evidence to support the idea that a nonlinear dose-response relationship is the norm is accumulating for many nongenotoxic and some genotoxic carcinogens. Dose-response curves for saccharin (*45*), butylated hydroxyanisole [BHA (*84*)], and a variety of other nongenotoxic carcinogens appear to be nonlinear (*85*). Formaldehyde, a genotoxic carcinogen, also has a nonlinear dose response (*28, 29*). The data for both bladder and liver tumors in the large-scale study on acetylaminofluorene, a genotoxic chemical, could fit a hockey stick–shaped curve, though a linear model, with a decreased effect at lower dose rates when the total dose is kept constant (*86*), has not been ruled out.

Carcinogens effective at both mutating and killing cells (which includes most mutagens) could be "complete" carcinogens and therefore possibly more worrisome at doses far below the MTD than carcinogens acting mainly by causing cell killing or proliferation (*15*). Thus, all carcinogens are not likely to be directly comparable, and a dose of 1/100 the TD_{50} (HERP = 1%) might be much more of a carcinogenic hazard for the genotoxic carcinogens dimethylnitrosamine or aflatoxin than for the apparently nongenotoxic carcinogens trichloroethylene, PCBs, or alcohol (HERP values marked with asterisks in Table 1). Short-term tests for mutagenicity (*61, 87*) can have a role to play, not only in understanding mechanisms, but also in getting a more realistic view of the background levels of potential genotoxic carcinogens in the world. Knowledge of mechanism of action and comparative metabolism in rodents and humans might help when estimating the relative importance of various low-dose exposures.

Human cancer, except in some occupational or medicinal drug exposures, is not from high (just subtoxic) exposures to a single chemical but is rather from several risk factors often combined with a lack of antirisk factors (*60*); for example, aflatoxin (a potent mutagen) combined with an agent causing cell proliferation, such as hepatitis B virus (*19*). High salt [a possible risk factor in stomach cancer (*13*)] and high fat [a possible risk factor in colon cancer (*4*)] both appear to be effective in causing cell killing and cell proliferation.

Risk from carcinogenesis is not linear with time. For example, among regular cigarette smokers the excess annual lung cancer incidence is approximately proportional to the fourth power of the duration of smoking (*88*). Thus, if human exposures in Table 1 are much shorter than the lifetime exposure, the possible hazard may be markedly less than linearly proportional.

A key question about animal cancer tests and regulatory policy is the percentage of tested chemicals that will prove to be carcinogens (*89*). Among the 392 chemicals in our database that were tested in both rats and mice, 58% are positive in at least one species (*14*). For the 64 "natural" substances in the group, the proportion of positive results is similar (45%) to the proportion of positive results in the synthetic group (60%). One explanation offered for the high proportion of positive results is that more suspicious chemicals are being tested (for example, relatives of known carcinogens), but we do not know if the percentage of positives would be low among less suspicious chemicals. If toxicity is important in carcinogenicity, as we have argued, then at the MTD a high percentage of all chemicals might be classified as "carcinogens."

The Background of Natural Carcinogens

The object of this article is not to do risk assessment on naturally occurring carcinogens or to worry people unduly about an occasional raw mushroom or beer, but to put the possible hazard of man-made carcinogens in proper perspective and to point out that we

the knowledge to do low-dose "risk assessment." We also are most completely ignorant of the carcinogenic potential of the enormous background of natural chemicals in the world. For example, cholinesterase inhibitors are a common class of pesticides, both man-made and natural. Solanine and chaconine (the main alkaloids in potatoes) are cholinesterase inhibitors and were introduced generally into the human diet about 400 years ago with the dissemination of the potato from the Andes. They can be detected in the blood of almost all people (*12, 90*). Total alkaloids are present at a level of 15,000 μg per 200-g potato with not a large safety factor (about sixfold) from the toxic level for humans (*91*). Neither alkaloid has been tested for carcinogenicity. By contrast, malathion, the main synthetic organophosphate cholinesterase inhibitor in our diet (17 μg/day) (*16*), is not a carcinogen in rodents.

The idea that nature is benign and that evolution has allowed us to cope perfectly with the toxic chemicals in the natural world is not compelling for several reasons: (i) there is no reason to think that natural selection should eliminate the hazard of carcinogenicity of a plant toxin that causes cancer in old age past the reproductive age, though there could be selection for resistance to the acute effects of particular carcinogens. For example, aflatoxin, a mold toxin that presumably arose early in evolution, causes cancer in trout, rats, mice, and monkeys, and probably people, though the species are not equally sensitive. Many of the common metal salts are carcinogens (such as lead, cadmium, beryllium, nickel, chromium, selenium, and arsenic) despite their presence during all of evolution. (ii) Given the enormous variety of plant toxins, most of our defenses may be general defenses against acute effects, such as shedding the surface lining of cells of our digestive and respiratory systems every day; protecting these surfaces with a mucin layer; having detoxifying enzymes that are often inducible, such as cytochrome P-450, conjugating enzymes, and glutathione transferases; and having DNA repair enzymes, which would be useful against a wide variety of ingested toxic chemicals, both natural and synthetic. Some human cancer may be caused by interfering with these normal protective systems. (iii) The human diet has changed drastically in the last few thousand years, and most of us are eating plants (such as coffee, potatoes, tomatoes, and kiwi fruit) that our ancestors did not. (iv) Normal metabolism produces radiomimetic mutagens and carcinogens, such as hydrogen peroxide and other reactive forms of oxygen. Though we have defenses against these agents, they still may be major contributors to aging and cancer. A wide variety of external agents may disturb this balance between damage and defense (*12, 42*).

Implications for Decision-Making

For all of these considerations, our scale is not a scale of risks to humans but is only a way of setting priorities for concern, which should also take into account the numbers of people exposed. It should be emphasized that it is a linear scale and thus may overestimate low potential hazards if, as we argue above, linearity is not the normal case, or if nongenotoxic carcinogens are not of very much concern at doses much below the toxic dose.

Thus, it is not scientifically credible to use the results from rodent tests done at the MTD to directly estimate human risks at low doses. For example, an EPA "risk assessment" (*92*) based on a succession of worst case assumptions (several of which are unique to EDB) concluded that EDB residues in grain (HERP = 0.0004%) could cause 3 cases of cancer in 1000 people (about 1% of all U.S. cancer). A consequence was the banning of the main fumigant in the country. It would be more reasonable to compare the possible hazard of EDB residues to that of other common possible hazards. For example, the aflatoxin in the average peanut butter sandwich, or a raw mushroom, are 75 and 200 times, respectively, the possible hazard of EDB. Before banning EDB, a useful substance with rather low residue levels, it might be reasonable to consider whether the hazards of the alternatives, such as food irradiation, or the consequences of banning, such as increased mold contamination of grain, pose less risk to society. Also, there is a disparity between OSHA not regulating worker exposures at a HERP of 140%, while the EPA bans the substance at a HERP of 0.0004%. In addition, the FDA allows a possible hazard up to a HERP of 0.3% for peanut butter (20 ppb), and there is no warning about buying comfrey pills.

Because of the large background of low-level carcinogenic and other (*93*) hazards, and the high costs of regulation, priority setting is a critical first step. It is important not to divert society's attention away from the few really serious hazards, such as tobacco or saturated fat (for heart disease), by the pursuit of hundreds of minor or nonexistent hazards. Our knowledge is also more certain about the enormous toll of tobacco—about 350,000 deaths per year (*1, 2*).

There are many trade-offs to be made in all technologies. Trichloroethylene and tetrachloroethylene (perchloroethylene) replaced hazardous flammable solvents. Modern synthetic pesticides displaced lead arsenate, which was a major pesticide before the modern chemical era. Lead and arsenic are both natural carcinogens. There is also a choice to be made between using synthetic pesticides and raising the level of plants' natural toxins by breeding. It is not clear that the latter approach, even where feasible, is preferable. For example, plant breeders produced an insect-resistant potato, which has to be withdrawn from the market because of its acute toxicity to humans due to a high level of the natural plant toxins solanine and chaconine (*12*).

This analysis on the levels of synthetic pollutants in drinking water and of synthetic pesticide residues in foods suggests that this pollution is likely to be a minimal carcinogenic hazard relative to the background of natural carcinogens. This result is consistent with the epidemiologic evidence (*1*). Obviously prudence is desirable with regard to pollution, but we do need to work out some balance between chemophobia with its high costs to the national wealth, and sensible management of industrial chemicals (*94*).

Human life expectancy continues to lengthen in industrial countries, and the longest life expectancy in the world is in Japan, an extremely crowded and industrialized country. U.S. cancer death rates, except for lung cancer due to tobacco and melanoma due to ultraviolet light, are not on the whole increasing and have mostly been steady for 50 years. New progress in cancer research, molecular biology, epidemiology, and biochemical epidemiology (*95*) will probably continue to increase the understanding necessary for lengthening life-span and decreasing cancer death rates.

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96. A national survey of U.S. drinking water supplies identified the concentrations of about 20 organic compounds. The mean total trihalomethane concentration was 117 μg/liter, with the major component, chloroform, present at a mean concentration of 83 μg/liter (83 ppb). Raw water that is relatively free of organic matter results in drinking water relatively free of trihalomethanes after chlorination. These studies are reviewed in S. J. Williamson, *The Science of the Total Environment* 18, 187 (1981).
97. Public and private drinking water wells in Santa Clara Valley, California, have been found to be contaminated with a variety of halogenated hydrocarbons in small amounts. Among 19 public water system wells, the most commonly found contaminants were 1,1,1-trichloroethane (TCA), and 1,1,2-trichloro-1,2,2-trifluoroethane (Freon-113). TCA was found in 15 wells generally at concentrations of less than 30 ppb, though one well contained up to 8800 ppb, and Freon-113 was found in six wells at concentrations up to 12 ppb. Neither chemical has been adequately tested for carcinogenicity in long-term bioassays. In addition to these compounds, three wells also contained carcinogenic compounds at low concentrations. Water from public supply wells may be mixed with treated surface water before delivery, thus the concentrations of these compounds that people actually receive may be somewhat reduced. Thirty-five private drinking water supply wells were examined; the major contaminant was the carcinogen trichloroethylene (TCE), at levels up to 2800 ppb. TCA and Freon-113 were also found in some wells, at maximum levels of 24 ppb and 40 ppb, respectively. Though fewer people drink from private water wells, the contaminant concentrations may be higher because the water is not mixed with water from other sources [California Department of Health Services, California Regional Water Quality Control Board 2, Santa Clara County Public Health Department, Santa Clara Valley Water District, U.S. Environmental Protection Agency, *Ground Water and Drinking Water in the Santa Clara Valley: A White Paper* (1984), table 8]. Trichloroethylene may not be a carcinogen in humans at low doses [R. D. Kimbrough, F. L. Mitchell, V. N. Houk, *J. Toxicol. Environ. Health* 15, 369 (1985)].
98. Contaminated drinking water in the area of Woburn, Massachusetts, was found to contain 267 ppb trichloroethylene, 21 ppb tetrachloroethylene, 12 ppb chloroform, 22 ppb trichlorotrifluoroethane, and 28 ppb 1,2-*trans*-dichloroethylene [S. W. Lagakos, B. J. Wessen, M. Zelen, *J. Am. Stat. Assoc.* 81, 583 (1986)].
99. The amount of chloroform absorbed by a 6-year-old child in a chlorinated freshwater swimming pool has been estimated [J. A. Beech, *Med. Hypotheses* 6, 303 (1980)]. Table 1 refers to the chloroform in an average pool (134 μg/liter) and for a 37-kg child. Three other trihalomethanes were identified in these freshwater pools: bromoform, bromodichloromethane and chlorodibromomethane. U. Lahl, J. Vondusze, B. Gabel, B. Stachel, W. Thiemann [*Water Res.* 15, 803 (1981)] have estimated absorption in covered swimming pools.
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101. The average adult daily PCB intake from food estimated by the FDA in fiscal years 1981/1982 was 0.2 μg/day (*16*). Many slightly different PCB mixtures have been studied in long-term animal cancer bioassays; the calculation of TD_{50} was from a test of Aroclor 1260 which was more potent than other PCBs (*14*).
102. The average consumption of EDB residues in grains has been estimated by the EPA for adults as 0.006 μg kg^{-1} day^{-1} and for children as 0.013 μg kg^{-1} day^{-1} [U.S. EPA Office of Pesticide Programs, *Ethylene Dibromide (EDB) Scientific Support and Decision Document for Grain and Grain Milling Fumigation Uses* (8 February 1984)].
103. The leaves and roots of Russian comfrey are widely sold in health food stores and are consumed as a medicinal herb or salad plant or are brewed as a tea. Comfrey leaf has been shown to contain 0.01 to 0.15%, by weight, total pyrrolizidine alkaloids, with an average level of 0.05% for intermediate size leaves [C. C. J. Culvenor, J. A. Edgar, J. L. Frahn, L. W. Smith, *Aust. J. Chem.* 33, 1105 (1980)]. The main pyrrolizidine alkaloids present in comfrey leaves are echimidine and 7-acetyllycopsamine, neither of which has been tested for carcinogenicity. Almost all tested 1,2-unsaturated pyrrolizidine alkaloids have been shown to be genotoxic and carcinogenic [H. Mori *et al.*, *Cancer Res.* 45, 3125 (1985)]. Symphytine accounts for 5% of the total alkaloid in the leaves and has been shown to be carcinogenic [C. C. J. Culvenor *et al.*, *Experientia* 36, 377 (1980)]. We assume that 1.5 g of intermediate size leaves are used per cup of comfrey tea (Table 1). The primary alkaloids in comfrey root are symphytine (0.67 g per kilogram of root) and echimidine (0.5 g per kilogram of root) [T. Furuya and M. Hikichi, *Phytochemistry* 10, 2217 (1971)]. Comfrey-pepsin tablets (300 mg of root per tablet) have a recommended dose of one to three tablets three times per day. Comfrey roots and leaves both induce liver tumors in rats [I. Hirono, H. Mori, M. Haga, *J. Natl. Cancer Inst.* 61, 865 (1978)], and the TD_{50} value is based on these results. Those pyrrolizidine alkaloids tested have been found to be at least

Expression of Risks

Just as a comparison of risks is an aid in understanding them, so is a careful selection of the methods of expression. It is hard to comprehend the statistical (stochastic) nature of risk. There are ways to mitigate this difficulty in comprehension. We are almost all used to one such statistical concept—the expectation of life. When we talk about the expectation of life being 79 years (for a nonsmoking male in the United States) we all know that some die young and that many live to be over 80. Thus the expression of a risk as the reduction of life expectancy caused by the risky action conveys some of the statistical concept essential to its understanding. One particular calculation of this type can be used as an anchor for many people, because it is easy to remember. The reduction of life expectancy by smoking cigarettes can be calculated from the risk, one in 2 million, of smoking one cigarette, multiplied by the difference of the average life-span of a nonsmoker and a lung cancer victim. This turns out to be 5 minutes, or the time it takes to smoke the one cigarette.

It is important to realize that risks appear to be very different when expressed in different ways (*19*). One example of this can be seen if we consider the cancer risk to those persons exposed to radionuclides after the Chernobyl disaster. According to the Soviets (*20*), the 24,000 persons between 3 and 15 kilometers from the plant, but excluding the town of Pripyat, received and are expected to receive 1.05 million man-rems total integrated dose, or about 44 rems average. Even if we assume a linear dose-response relation, with 8000 man-rems per cancer, the risk may be expressed in different ways. Dividing 1.05 million man-rems by 8000 gives 131 cancers expected in the lifetimes of that population. This is larger than, and for some people more alarming than, the 31 people within the power plant itself who died within 60 days of acute radiation sickness combined with burns. Dividing the 131 again by the approximately 5000 cancer deaths expected from other causes, the accident caused "only" a 2.6% increase in cancer. This seems small compared to the 30% of cancers attributable to cigarette smoking. The difference is even more striking if we consider the 75 million people in Byelorussia and the Ukraine who received, and will receive, 29 million man-rems over their lifetimes. On the linear dose-response relation this leads to 3500 "extra cancers," surely a large number for one accident. But dividing by the 15 million cancers expected in this population leads to an "insignificant" increase of 0.0047%. Of course, none of the methods of expressing the risk can be considered "right" in an absolute sense. Indeed, it is our belief that a full understanding of the risk involves expressing it in as many different ways as possible.

Cost of Reducing a Risk

Another interesting and instructive way of comparing risks is by comparing the amount people have paid in the past to reduce them. It might be thought that people would try to adjust their activities until the amount spent is roughly the same. Cohen (*21*) has shown that the amounts spent vary by a factor of more than a million. He shows that it would be possible even for an American to save lives in Indonesia by aiding in immunization at $100 per life saved. Society is willing to spend more on environmental protection to prevent cancer (over $1 million per life) than on cures (about $50,000 per life with the high value of $200,000 for kidney dialysis raising some objections). This ratio is in rough accord with the maxim "an ounce of protection is better than a pound of cure." People are willing to spend still more on radiation protection at nuclear power plants and on waste disposal. Economists and others often argue that efficiency depends on adjusting society until the amounts spent to save lives in different situations are equalized. It seems to us that society does not work that way. People are aware of the order of magnitude of these differences, and approve of them. Nonetheless, we believe that providing this information to a decision-maker is essential for an informed decision.

Table 3. Comparison of two very toxic chemicals, aflatoxin B1 (*22*) and dioxin (*23*); CDC, Centers for Disease Control.

Measure	Aflatoxin B1	Dioxin
Acute toxicity	High	Equal
Carcinogenic potency to people [(kg · day)/mg]	~500	Unknown
Carcinogenic potency to rats [(kg · day)/mg]	~5000	~5000
Mutagenic	Yes	No
Certainty of information on human carcinogenicity	High	Low
Activity (initiator or promoter)	Initiator	Promoter (?)
Possibility of threshold dose response	Low	High
Source	Natural	Artificial
Common knowledge	Little known	Agent Orange
FDA action level in peanuts (ppb)	20	
CDC level of concern in soil (ppb)		1

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Testing of Police Traffic Radar Device

Volume I
Test Program Summary
April 1984

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TESTING OF POLICE TRAFFIC RADAR DEVICES TO THE MODEL PERFORMANCE SPECIFICATIONS FOR POLICE TRAFFIC RADAR DEVICES

VOLUME I
TEST PROGRAM SUMMARY

APRIL 1984

PREPARED BY THE
PERFORMANCE TEST CENTER (PTC)
RESEARCH AND DEVELOPMENT DIVISION
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UNDER

COOPERATIVE AGREEMENT NUMBER NB82NAHA3038
LAW ENFORCEMENT STANDARDS LABORATORY (LESL)
NATIONAL BUREAU OF STANDARDS
U. S. DEPARTMENT OF COMMERCE
WASHINGTON, D.C. 20234

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Director
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PREFACE

This document is one of two prepared to make available the results of the police traffic radar testing program conducted by the International Association of Chiefs of Police. Twenty-four radar devices were tested for conformance to the minimum requirements of the Model Performance Specifications for Police Traffic Radar Devices published by the National Highway Traffic Safety Administration. Titles of the reports and identification of the contents of each are as follows:

Vol. I: Testing of Police Traffic Radar Devices to the Model Performance Specifications for Police Traffic Radar Devices Volume I: Test Program Summary

This volume provides an overview of the police traffic radar testing program. Requirements of the model specifications are discussed along with comments on the test results. A Consumer Products List (CPL) of radar devices found to be in full compliance with the model performance requirements is provided.

Vol. II: Testing of Police Traffic Radar Devices to the Model Performance Specifications for Police Traffic Radar Devices Volume II: Test Data

Volume II contains a report of the test data for each of the 24 radar devices tested. Each report of the test data follows the same format and numbering as the model performance specifications. This volume will be of more interest to the technically-oriented reader.

CONTENTS

EXECUTIVE SUMMARY

Twenty-four models of police traffic radar devices were tested for performance characteristics and compliance to the Model Performance Specifications for Police Traffic Radar Devices developed for the National Highway Traffic Safety Administration (NHTSA) by the Law Enforcement Standards Laboratory (LESL) of the National Bureau of Standards (NBS). The specifications were adopted by the International Association of Chiefs of Police (IACP), and the testing program was conducted by them in accordance with a cooperative agreement with NBS/LESL. Testing was accomplished by two independent testing laboratories during the period June 1983 to January 1984.

The radar devices were subjected to examination, including documents and accessories, laboratory evaluation and operational testing. During the period of testing, the equipment manufacturers were afforded the opportunity to make minor modifications to their products to achieve compliance with the model specification requirements. The development of the performance specifications and testing of radar devices to the requirements of the model specification have led to definitive improvements in radar devices that will be available in the future, and to the listing of 24 models on the IACP Consumer Products List (CPL). See Appendix A for a copy of the CPL.

It is recommended by the IACP that every agency procuring radar units require the successful bidder to certify that the radar devices are included on the IACP CPL and meet the NHTSA/IACP Model Performance Specifications for Police Traffic Radar Devices. This dual certification is important because some manufacturers have indicated that model numbers on both conforming and non-conforming units may be identical.

INTRODUCTION

For more than 30 years, police traffic radar has been used beneficially in the enforcement of speed laws and development of radar evidence to support speeding violations. Over this span of time, new technologies led to the development of radar devices more mobile and efficient in traffic law enforcement. Radar devices became smaller, more portable and capable of monitoring vehicle speeds in the moving mode. Many were equipped with special automated features intended to improve radar operator efficiency.

Along with the technological innovations came a growing concern of the public, and also the courts, about the reliability of vehicle speed data obtained from some of the more technically-sophisticated speed measuring radar devices. This concern was particularly strong regarding radar operators who may not have received adequate training in the use of some newer model radar devices and in the recognition and avoidance of electronic anomalies associated with them. This developing dilemma in radar usage was not helped by the fact that, at that time, a nationally-recognized equipment performance standard for radar devices did not exist, nor was there a generally accepted comprehensive program of operator training.

It was against this background that the International Association of Chiefs of Police (IACP) passed a resolution in 1976 calling for federal government concern and involvement in the development of health, safety and performance standards for speed measuring devices, the testing of the devices and publication of the results.

In August 1977, the National Highway Traffic Safety Administration (NHTSA) entered into an interagency agreement with the Law Enforcement Standards Laboratory (LESL) of the National Bureau of Standards (NBS) to perform a series of tasks relating to speed measuring devices, including the development of a performance standard for police speed measuring radar devices. In separate actions, NHTSA addressed the matters of police radar device operator training and the possible health hazards associated with radar use. (See the Bibliography for listings.) The draft performance standard for police speed measuring radar devices, developed by LESL, appeared in the Federal Register (23 CFR 1221) on January 8, 1981, as a proposed rule making. Following an extensive review and comment period, the NHTSA published the revised performance standard as Model Performance Specifications for Police Traffic Radar Devices (DOT-HS-806-191). Subsequently, based on the recommendation of the IACP Technology Assessment Program Advisory Council, the IACP adopted these specifications as an IACP specification for police radar devices.

Publication of the model performance specifications prompted an IACP proposal to test police speed measuring radar devices to determine compliance with the model specifications and to prepare a Consumer Products List (CPL) in the same manner that other types of police equipment were being tested by the IACP as part of the National Institute of Justice (NIJ) Technology Assessment Program. After review and agreement by NHTSA, the unsolicited IACP proposal was accepted and, in October 1982, the IACP entered into a cooperative agreement with NBS/LESL to accomplish the testing of radar devices and publish the test results.

During the time following issuance of the proposed rule making, up to and including the period of radar testing, manufacturers of radar devices made several modifications to their products directed toward achieving compliance with the model performance specifications.

All known manufacturers and distributors of speed measuring radar devices were given an opportunity to submit devices for inclusion in this test program. However, only five of the seven who were contacted sent in radar devices to be evaluated. See Appendix B for a list of manufacturers.

This report summarizes the results of the testing program in which 24 radar devices met the requirements in full for listing on the IACP Consumer Products List.

THE MODEL PERFORMANCE SPECIFICATIONS

The Model Performance Specifications for Police Traffic Radar Devices (model performance specifications) used for this radar equipment testing program were developed by the Law Enforcement Standards Laboratory (LESL) of the National Bureau of Standards (NBS) under an interagency agreement with the National Highway Traffic Safety Administration (NHTSA). A brief review of the development of, and contents of, the performance specifications will improve the reader's understanding of the scope of this program and help each to appreciate the results achieved in radar development and refinement over the past several years.

Development of the model performance specifications began with research by LESL in a number of scientific areas including the compilation of the performance parameters of the then currently-used police speed measuring devices; a cataloging of technical data and useful, desirable and undesirable features of each; an analysis of the field operational characteristics associated with the various types of speed measuring devices; an analysis of the anomalies due to the electromagnetic environment that had the potential to cause operator error in making speed readings; the development of performance characteristics for the radar devices consistent with the technology available; and finally the development of procedures by which radar device performance could be tested for conformance to the performance specifications.

Prior to being forwarded to NHTSA, the initial draft performance standard for radar devices was circulated for comment by NBS. Thirty-five comments were received from 16 law enforcement agencies, five manufacturers, four universities, four individuals, four district attorneys or judges, one research institute and the Federal Communications Commission.

After appropriate changes were made to the standard, it was forwarded to NHTSA and published in the Federal Register (23 CFR 1221) for review and comment as a proposed rule making. At this time, comments were received from six individuals, nine law enforcement departments, three radar manufacturers and one university. Following this review and comment period, the revised standard was published, with a comprehensive discussion of the rationale for the revision, by NHTSA as model performance specifications for police traffic radar.

The performance requirements and test procedures of the model specifications are too lengthy for full comment here. Those readers desiring additional details should obtain a copy of the model specifications from the issuing agency (see bibliography). However, the comprehensive nature of the performance characteristics and the testing prescribed will be evident from the following outline of the general topics addressed by the model specifications.

1. Physical Examination and Inspection

   Manufacturer-Provided Information
   Tuning Fork Certificate
   Tuning Fork Markings
   Radar Device Labeling

2. Laboratory Tests

Tuning Fork Calibration
Radar Device Tuning Fork Tests
Microwave Transmission

- Frequency Stability
- Input Current Stability
- Output Power Stability
- Beam Width
- Power Density

Environmental Tests

- Low Temperature (-22^{0}F)
- High Temperature (+140^{0}F)
- High Humidity (90% at 99^{0}F)
- Vibration

Function Tests

- Low Voltage Alert
- Doppler Audio
- Speed Monitor Alert
- Power Surge

Speed Display and Signal Processing Channel

- Character Height and Contrast
- Speed Lock and Display Clear Functions
- Internal Test
- Signal Processing Channel Sensitivity
- Low and High Speed Displays

Electromagnetic Interference Susceptibility

- Vehicle Alternator
- Heater/Air Conditioner Motor
- Windshield Wiper Motor
- Police FM Transceiver
- Citizens Band Transceiver

3. Operational Tests

Electromagnetic Interference Susceptibility

- Police FM Transceiver
- Citizens Band Transceiver

Speed Accuracy Tests

- Conducted over a measured course at speeds of 20, 50 and 70 mph in stationary and moving modes, as applicable.

More details of the performance requirements and the test procedures appear in the summary of the test results.

THE TESTING LABORATORIES

Consistent with the procedures presently used by the International Association of Chiefs of Police (IACP) for the National Institute of Justice (NIJ) Equipment Testing Program, the IACP issued a request for proposal for testing radar devices in accordance with procedures incorporated in the Model Performance Specifications for Police Traffic Radar Devices. Requests for testing program documents were received from over 50 independent testing organizations. Of the laboratories submitting proposals, the two selected to perform the testing were: Dayton T. Brown, Inc., Bohemia, Long Island, New York; and Department of Electrical Engineering and Systems Science, Michigan State University, East Lansing, Michigan. Selections were made on the basis of cost and assessment of the laboratory's facilities, equipment, personnel and overall competency to conduct the tests in accordance with the specified procedures.

Each selected laboratory was initially supplied with one radar device for preliminary testing. During this phase of the testing, technically qualified personnel from the National Bureau of Standards, National Highway Traffic Safety Administration and the IACP visited each laboratory to ensure that the testing equipment was proper and that qualified personnel were conducting selected tests in the proper manner.

The testing laboratories were given the main quantity of radar devices during June and July 1983, and initial testing of them was completed by October 1, 1983. Additional visits were made to the laboratories during this testing period to review test results, follow progress of the testing, and to resolve any questions that may have arisen during the testing activities.

Based on the initial test results, the radar manufacturers were afforded the opportunity to correct any deficiencies in their radar units before the final testing phase. Final laboratory test reports were submitted to the IACP in January 1984. During subsequent analysis of the test data and the preparation of this report, it was necessary to verify certain test results from raw data, and to conduct tests in those few instances in which the test results were not complete.

SUMMARY OF TEST RESULTS

As noted earlier, the testing was accomplished in two phases. Upon completion of the initial testing, representatives of the IACP, NBS, and NHTSA reviewed the laboratory test results in detail. Many of the individual radar devices failed to comply with the model specification because the required manufacturer information was not provided with the unit. For example, some devices lacked operating or installation instructions while, for others, the required tuning fork certificate was either incomplete or not provided. In addition, there are a number of instances in the model specifications where the speed measuring radar device is tested to the manufacturer's specification for a given parameter if the manufacturer claims a greater capability than that required by the model specification. For example, the model specification places a limit on signal processing sensitivity variation for targets traveling at speeds of 20 to 90 mph. In a number of cases, although a radar unit met this requirement of the model specifications, the manufacturer data sheet claimed an operating capability below 20 mph and the unit, when tested at the lower speed, did not meet the signal processing channel sensitivity requirement.

Similarly, there were a number of radar units that did not comply with the display labelling requirements of the model specification. Very few of the radar units provided by the manufacturers were in full compliance with the labeling and the manufacturer-provided information requirements of the model specification at the time of initial testing. However, it was clear from earlier discussions with the manufacturers, in advance of the testing, that the manufacturers were willing to correct differences in the information provided with the units, specifications, and labeling to fully comply with the requirements of the model specification.

There were some areas of noncompliance with the requirements of the model specification that required minor modifications to the individual radar unit to achieve full compliance. Other than labeling and instructions, the most common deficiency was the failure to meet the signal processing channel sensitivity requirement. This deficiency was corrected, in most cases, by a change in the values of the filter components used to control this sensitivity. The second most common deficiency was in the display readability capability of the units, i.e., character height and luminance contrast had to be improved. The design of the radar units is such that either individual components or the display module itself can be easily replaced in order to comply with the readability requirements of the model specifications.

Other areas of noncompliance included such items as frequency stability under conditions of high and low temperature or high humidity. In examining all of the initial tests results, it was apparent that the manufacturers could easily make minor modifications to their products at a minimal expense to achieve full compliance with the requirements of the model specification.

The IACP was of the opinion that the law enforcement community would benefit from the wide availability of speed measuring radar devices that were in full compliance with the requirements of the model specification, and recommended that all of the manufacturers be given the opportunity to make those modifications to their products that were necessary to achieve full compliance. The NHTSA accepted this recommendation, which was also acceptable to NBS.

The IACP provided each manufacturer with the results of the initial tests conducted on their products only, and notified the manufacturers that they would have the opportunity to correct their product literature and to make minor modifications to the individual speed measuring devices to achieve compliance with the model specifications. The conditions of this offer included: 1) any required modification was to be made to the same unit that had been tested during initial testing, 2) the modification was to be made at the test facility in the presence of testing laboratory personnel, 3) the NBS technical staff was to be provided copies of any circuit modifications so that NBS could advise IACP as to what additional testing would be required based on the modifications that were made, and 4) it would be the manufacturer's responsibility to pay for any required testing following modification.

All five manufacturers accepted the offer. The modifications were made, and the laboratories provided final phase test results to the IACP. During the course of the testing and subsequent data review by IACP and NBS, all of the deficiencies were corrected to bring the radar devices into full compliance with the model specifications. The radar devices involved in this test program are identified in Table 1 (see p. 20).

The deficiencies noted among the radar units as the testing progressed are summarized in the discussion that follows and in Table 2 at the end of this section. This discussion is done in a topical manner rather than by individual radar units, as is done in the table and in Volume II of this report, which presents complete test data.

PHYSICAL EXAMINATION AND INSPECTION

Manufacturer-Provided Equipment

Each radar device is required to be accompanied by one or more tuning forks, as applicable, for routine use in checking the operational condition of the radar on a day-to-day basis. The tuning fork supplied with one radar unit was of an incorrect frequency for the radar device.

Manufacturer-Provided Information

In this area, many radar devices failed to conform to the requirements of the model specifications. The information required is listed, followed by the number of radar units lacking the required data.

Installation Instructions	6
Precautions to Avoid Interference from:	
Vehicle Ignition	19
Heater A/C Defroster Blowers	14
Operating Instructions, including:	
Test Procedures	5
Internal Test Circuit Data	3
Required Maintenance	16

Characteristics Indicative of Radar Malfunctioning	15
Power Supply Data	12
Microwave Frequency Band of Operation	0
Nominal Microwave Output Power	0
Operational Voltage Limits	3
Maximum Microwave Power Density	1
Antenna Horizontal Beam Width	1
Antenna Polarization	0
Operating Speeds of the Radar	7
Designed Operational Temperature and Humidity Extremes	1

Tuning Fork Calibration Certificate

Each tuning fork shall have a calibration certificate that includes the tuning fork serial number, nominal speed, frequency, frequency band (X or K) and temperature correction factor.

Certificate was supplied but contained incomplete information	5
No certificate was provided	12

Labeling Requirements

Tuning Fork Permanent Markings of Serial Number, Frequency and X- or K- Band	1
Radar Device Control Functions	10

A radar device may have approximately 20 or more switches, controls, and functions depending upon the particular type unit. Labeling was generally satisfactory. The low voltage indicator and speed lock were most lacking in marking, with 4 deficiencies each.

Each test is described, and the number of radar devices in noncompliance is given.

Tuning Fork Calibration 12

This test requires measurement of the frequency of the tuning fork to ensure that it is within ± ½% of the frequency specified in the certificate of calibration. This could not be accomplished in the case of 12 radar units as no calibration certificate was supplied by the manufacturer.

Radar Device Tuning Fork Tests 4

In these tests the tuning fork(s) supplied with a radar unit are used to generate a pseudo-doppler signal to check the operational condition of the radar unit. If the radar is functioning properly, the speed for which the tuning fork is calibrated should appear in the speed display with a tolerance of ± 1 mph. With moving radar devices, both target and patrol vehicle displays must show the correct speed readings. The tuning fork tests are conducted under standard test conditions, at specified low and high temperatures, under high humidity conditions and during vibration. All radar devices were in compliance at ambient temperature. However, 4 devices failed to comply during environmental tests. Three radar units did not function properly at low temperature. One of the three also did not function properly under the high temperature test conditions, and another one of the three failed during the high humidity test. The speed display on one additional unit showed erroneous readings during the vibration testing.

Microwave Transmission

The microwave transmission tests check the stability of operation of the radar device under conditions that could be encountered in the operational environment. These tests were repeated a number of times to record performance data under standard test conditions (68-80^{0}F), under conditions of low temperature (-22^{0}F), high temperature (+140^{0}F) and high humidity (90%) with temperature at least 99^{0}F. All tests were repeated at three voltage levels: nominal voltage (13.6V), nominal voltage plus 20% (16.3V), and at minus 20% (10.8V) or a lower voltage that a manufacturer may have specified. The following characteristics were tested as specified above:

(a) Frequency Stability 5

Five of the radar devices did not maintain frequency stability within the allowable tolerance during these tests. One failed at all conditions, while three others failed the low temperature test. A fifth device failed during the high humidity test. X-Band radars must operate between 10,500 and 10,550 MHz, and K-Band radars must transmit between 24,050 and 24,250 MHz.

(b) Input Current Stability 8

Three of the same radar units that did not meet the frequency stability requirement also did not meet the input current stability requirement of less than a 10% variation and no change in numerical display under one or more of the test conditions, two at low temperature and one at high temperature. Three additional units were unstable during the low temperature tests, while a total of four failed at high temperatures. The same three plus one other device failed the high humidity test.

(c) Radiated Output Power Stability 4

Four radar units did not maintain the radiated power output within the plus or minus 1.5 dB from nominal as required. Two did not comply at low temperature conditions and one failed the high temperature test. That one, plus one other radar device, did not function properly under high humidity conditions.

(d) Antenna Horizontal Beam Width 4

The maximum horizontal beam width allowed by the model specifications is 18 degrees for X-Band radar and 15 degrees for K-Band. The permissible bandwidth was initially exceeded by three X-Band and one K-Band radar devices.

(e) Antenna Near-Field Power Density 4

The requirement for this test is that the measured power density may not exceed that specified by the manufacturer. Four radars did not meet this requirement.

Low Voltage Supply 12

Each radar device is required to operate to a specific low voltage point and to have a low voltage indicator capable of being heard or seen by the operator. The required low voltage operating point is 10.8V or the lowest voltage specified by the manufacturer. At the specified low voltage the speed display must show either no reading or no erroneous reading. Seven radar units did not meet the performance requirement. Five met the requirement of the model specifications, but not the lower voltage specified by the manufacturer.

Doppler Audio

(a) Audio Output and Volume Control 3

The radar device is required to emit a doppler audio tone that correlates with the received doppler signal and to have an audio volume control. The doppler audio tone is beneficial to the radar operator as an aid in correlating visual observation of a target vehicle with the radar speed display, and in alerting the operator to the presence of interference that may affect his or her ability to operate that radar device. Three radar units were not in compliance.

(b.) Audio Squelch and Squelch Override 2

The audio tone must be squelched in the absence of a target signal. The radar device must also permit the operator to inhibit the squelch action while keeping the receiver open. Two exceptions to this requirement were noted.

(c) Audio Track-Through-Lock 1

For radar devices with a track-through-lock feature, the doppler audio must continue after the speed-lock switch is activated. One radar unit did not meet this specification when initially tested.

Speed Monitor Alert 0

This capability, which alerts the operator when a target speed signal is received that is equal to or above some preselected threshold speed, is not permitted by the model specifications. All radar devices were in compliance at the time of initial testing.

Power Surge Test 0

This test is conducted to verify that switching the radar device from the standby to on mode will cause no erroneous speed reading with a target present. All radar devices were in compliance at the time of initial testing.

Speed Display

Under this caption is a lengthy series of tests designed to measure the performance characteristics of the radar device signal processing channel, the speed display(s) and associated functions.

(a) Display Readability 14

The illuminated segments used to indicate speed readings are required to have a minimum height of 0.4 inch and a daylight luminance contrast of not less than 2.5 for ease of operator reading. Three radar devices met neither requirement, while six other radars did not meet the display height requirement and five others did not meet the luminance contrast requirement.

(b) Display Speed Lock 0

A speed lock, if provided, must be manually operated, must preserve the displayed reading(s) and must not recall any previous reading when the speed-lock switch is activated. It should be noted that the elimination of the automatic self-locking capability represents one of the most significant features of the model specifications, since by using a radar device without this feature, a skilled and knowledgeable operator can develop a vehicle's tracking history and thereby avoid virtually all of the alleged anomalous readings ascribed to radar operation. No exceptions to this requirement were noted.

(c) Display Clear 0

It is required that the selection of a different mode of operation of the radar device shall automatically clear the radar device of all displayed readings whether or not the speed-lock switch is activated. There were no exceptions.

(d) Internal Circuit Test 4

The radar device must have a self-test function that, when activated, determines whether the internal signals will be processed and displayed within the limits of ± 1 mph. The displayed reading must clear upon switching to another mode of operation and it must be impossible for the reading to be locked in the speed display. Four radar devices did not meet this requirement during initial testing.

(e) Speed Display Transfer 0

This requirement, applicable to moving mode radar devices, specifies that it shall be impossible to transfer the patrol speed reading from the patrol display to the target display. All radar units were in compliance.

(f) Signal Processing Channel Sensitivity 15

These tests are made to evaluate the degree to which the radar device sensitivity varies in detecting and processing signals from slow-moving vehicles as compared to fast-moving target vehicles. Limits of sensitivity variation permitted by the model specification and results of the test are tabulated below.

Mode of Operation	Sensitivity Requirement	Noncompliance
Stationary	≤ 10 dB Speeds 20-90 mph	8
	≤ 10 dB Mfrs. Spec. to 90 mph	4
	≤ 3 dB Speeds 60-90 mph	10
Moving Mode-Patrol 25 mph	≤ 10 dB Speeds 40-90 mph	1
	≤ 3 dB Speeds 60-90 mph	2

The first eight radar units listed did not comply with requirements of the model specifications while the next four radar units are listed because they did not detect and process signals at target speeds below 20 mph, usually 10-15 mph specified by the manufacturer, without exceeding the requirement of the specification. In these four cases, the manufacturer's specifications were changed to bring the radar devices into compliance.

(g) Target Channel Speed Display 9

The target signal processor channel and target speed display shall function properly for targets traveling at a speed of 20 mph or at a lower speed specified by the manufacturer, in both the stationary and moving modes. The same is true for targets traveling at a speed of 100 mph in the stationary mode, and for closing speeds of 155 to 209 mph in the moving mode.

Speed/Mode	Speed Requirement	Noncompliance
Low Speed—Stationary	20 mph, or	0
	Mfrs. Spec.	4
Moving	20 mph, or	2
	Mfrs. Spec.	2
High Speed—Stationary	100 mph, or	1
	Mfrs. Spec.	5
Moving	100 mph (Patrol 20 mph)	0
	Mfrs. Spec.	2
Closing Speed—Patrol 55	100-154 mph	0

(h) Patrol Channel Speed Display 2

The patrol signal processor channel and patrol speed display shall function correctly at 20 mph or at a lower speed specified by the manufacturer when operating in the moving mode. The same is true for a speed of 55 mph or a higher speed specified by the manufacturer.

Low Speed	20 mph	0
High Speed	55 mph	0
	Mfrs. Spec.	1
Patrol Speed Change Tracking	± 1 mph	1

Electromagnetic Interference 4

Electromagnetic interference tests were conducted in the laboratory by impressing, on the radar devices, electronically-generated signals similar in frequency, wave form and amplitude to electromagnetic energy found in and around an operating motor vehicle. The signals were generated to simulate those given off by a vehicle alternator, vehicle ignition, air conditioner/heater motor, and windshield wiper motor. Test signals were also generated to simulate those characteristics of police FM transceiver operation and of the operation of citizens band (CB) transceivers in and around a patrol vehicle. While conducting the tests in both stationary and moving modes, no incorrect speed reading should be displayed. Depending on the particular radar device, the speed display may not change, or may go blank, in the presence of spurious electromagnetic interference. The requirement is met under either condition. In addition, the radar device may have an interference alert circuit that produces a warning signal to the operator whenever interference is detected.

Three of the radar devices tested displayed erroneous readings when subjected to interference during the simulated police FM transceiver interference tests. One of these three as well as one other radar device, displayed erroneous readings during the simulated CB interference testing.

OPERATIONAL TESTS

Electromagnetic Interference 3

The operational tests were conducted with the radar device properly installed in a patrol vehicle of the type normally used for law enforcement purposes. The test vehicle had an FM transceiver and antenna and a citizens band transceiver and antenna, each installed in accordance with the manufacturer's instructions. A handheld FM transceiver was also positioned in the vehicle for use by the driver. While the radar was tracking an acquired target vehicle traveling at 50 mph, audio tones from 500 to 3000 Hz generated by a slide whistle were transmitted via the microphone of the FM and CB transceivers. The radar display was observed for any erroneous readings caused by the slide whistle transmissions from the transceivers. At least two tests were conducted with each transceiver. The tests were repeated similarly with the patrol vehicle, in stationary mode, tracking a target vehicle while a third vehicle equipped with the FM and CB transceivers passed within 10 feet, first on one side of the patrol vehicle and then on the other.

One radar device was subject to interference from the FM transceiver when initially tested, while two others were interfered with by the CB transceiver.

Speed Accuracy Tests

The speed accuracy tests were conducted on a half-mile measured course, over which the target vehicle was driven at constant speeds of 20, 50 and 70 mph. The true speed of the target vehicle for each test was calculated from the elapsed time to travel the known distance, or measured with a fifth wheel speed measuring device. The speed displayed on each radar device during the runs was compared to the true target vehicle speed to determine whether or not the radar-displayed speed was within the allowable variation of +1, -2 mph in the stationary mode and $\pm$2 mph in the moving mode of operation.

It should be noted that the model radar specifications, as published, require that the speed measuring radar devices provide a speed accuracy of $\pm$ 1 mph in the stationary mode. During subsequent review of the specifications, it was recognized that this requirement, while realistic for the inherent accuracy of radar devices, did not take into account the manner in which the measured speed is displayed. When a radar unit processes the signal from a target vehicle to determine the speed that the signal represents, it measures the speed in units of 0.1 or even 0.01 mph. Prior to displaying the speed, the measured value is typically truncated (i.e., the fractional or decimal units of speed are discarded) and the speed is displayed in units of one mph. For example, if a radar unit measures a vehicle speed of 55.9 mph, a speed of 55.0 mph is displayed. Because of this, a radar device could measure the speed of a vehicle traveling at a rate of 50.5 mph as 49.9 mph and would display a speed of 49.0 mph, or 1.5 mph less than the true speed.

The requirement for speed measuring accuracy of radar devices of $\pm$ 1 mph, as stated in the model radar specification, was changed to +1, -2 mph for this program, and the model specification will be changed as well at such time as it is reissued.

During the initial testing, five radar devices were reported as not complying with the speed accuracy requirements of the model specifications; three in the stationary mode/moving target test and one of these three, in addition to two others, in the moving mode/approaching target test. However, these particular test data are considered questionable, since all five units met the speed accuracy requirements of all other speed tests required. The three radar devices that were not in compliance in the stationary mode/moving target test situation at 70 mph were in compliance when tested at speeds of 20 and 50 mph in the initial testing phase. Similarly, the three radar devices that did not meet the compliance requirement in the moving mode/approaching target (patrol 20 mph - target 55 mph) met the requirement in the higher speed test (patrol 55 mph - target 70 mph), and also the stationary tests at speeds of 20, 50 and 70 mph. All five units were found to comply fully with the speed accuracy requirements when retested during the final phase of testing. It should be noted that the final testing was conducted without adjustment or modification of the speed measuring circuitry of any of the five radar devices.

Minimum Operating Range

It should also be noted that the model specifications do not require that the radar device have a minimum operating range. If range is an important consideration for the conditions under which the radar device is intended for use, minimum range should be specified by the purchaser.

TABLE 1

RADAR DEVICE IDENTIFICATION

Stationary Mode Radars

IACP Test Lot Number	Radar Manufacturer	Model	Type	Operating Freq. Band (X or K)
2556	Decatur	RA-GUN GN-1	I	X
2557	MPH	K-15 (K)	III	K
2561	MPH	K-35 (K)	III	K
2563	Kustom	Falcon	III	K
2565	Kustom	Roadrunner	III	K
2568	Kustom	HR-8	III	K
2570	MPH	K-35 (X)	I	X
2574	MPH	K-15 (X)	I	X
2578	Decatur	RA-GUN KN-1	III	K

Moving Mode Radars

2555	Kustom	KR-10-SP	IV	K
2558	MPH	S-80 (K)	IV	K
2559	MPH	S-80MC (K)	IV	K
2560	MPH	K-55E (X)	II	X
2562	Kustom	Trooper	IV	K
2564	Kustom	KR-11	IV	K
2566	BEE	BEE-36 (X)	II	X
2567	Kustom	HR-12	IV	K
2569	BEE	BEE-36 (K)	IV	K
2571	MPH	K-55	IV	K
2572	MPH	S-80MC (X)	II	X
2573	MPH	S-80 (X)	II	X
2576	Decatur	MV-724	IV	K
2577	Decatur	MV-715	II	X
2580	CMI (Speedgun Magnum)		II	X

TABLE 2

SUMMARY OF INITIAL TESTS

REQUIREMENTS/TEST	Radar Device IACP Number — Types I and III									Types II and IV												
	2556	2557	2561	2563	2565	2568	2570	2574	2578	2555	2558	2559	2560	2562	2564	2566	2567	2569	2571	2572	2573	2576	2577	2580
1221.11 MANUFACTURER-PROVIDED EQUIPMENT									1													1	1	
Tuning Fork(s)											S													
1221.12a MANUFACTURER-SUPPLIED INFORMATION																								
(1) Installation Instructions		S	S					S						S				S			S			
Precautions to Avoid Interference From-																								
Vehicle Ignition	S	S	S	S	S		S	S		S	S	S	S	S	S	S		S	S	S	S			S
Heater/Air Conditioner/Defroster Blowers	S	S	S				S	S			S	S	S		S	S		S	S	S	S			
(2) Operating Instructions, Including-																								
Test Procedures		S	S				S	S													S			
Internal Circuit Test Data							S	S													S			
Required Maintenance		S	S	S	S	S	S	S			S	S	S	S	S			S	S	S	S			
Characteristics Indicative of Radar Malfunctioning	S	S	S		S		S	S			S	S	S	S		S		S	S	S	S			
(3) Power Supply Data: Voltage and Current-																								
With Display(s) Illuminated	S					S	S	S						S			S	S	S	S				
Without Displays Illuminated	S					S	S	S		S				S			S	S	S	S				
With Target Present	S					S	S	S		S				S			S	S	S	S	S			
Without Target Present	S					S	S	S		S				S			S	S	S	S				
In Standby Mode (if applicable)	S						S	S		S				S			S	S	S	S				S
(4) Microwave Frequency Band of Operation																								
(5) Nominal Output Power																								
(6) Operational Voltage Limits-																								
High Limit	S																							
Low Limit	S																							
Low Voltage Alert Threshold	S					S		S																
(7) Maximum Microwave Power Density	S																							
(8) Antenna Horizontal Beam Width	S																							
(9) Antenna Polarization																								
(10) Operating Speeds of Radar																								
Minimum Target Speed	S										S	S	S			S			S		S			
Maximum Target Speed	S											S							S					
Minimum Patrol Speed (Types II & IV only)																								
Maximum Patrol Speed (Types II & IV only)																								
(11) Designed Operational Temperature Extremes-																								
High and Low	S																							
(12) Designed Operational Humidity Extremes	S																							
1221.12b TUNING FORK CALIBRATION CERTIFICATE																								
Serial Number		S	S				S	S			S	S	S			S		S	S	S	S			
Nominal Design Speed		S	S				S	S			S	S	S			S		S	S	S	S			
Frequency Calibration at 70°F		S	S				S	S			S	S	S			S		S	S	S	S			
Microwave Frequency Band for Which Used		S	S				S	S			S	S	S			S		S	S	S	S			
Calibrated Frequency and Associated mph		S	S				S	S			S	S	S			S		S	S	S	S			
Correction Factor for 70°F Calibration	S	S	S			S	S	S		S	S	S	S			S	S	S	S	S	S			S
1221.13 LABELLING REQUIREMENTS																								
Tuning Fork Permanent Markings																								S
Radar Device Control Functions	S	S		S	S	S		S							S	S	S							S
LABORATORY TESTS																								
1221.71 TUNING FORK CALIBRATION		N	N				N	N			N	N	N			N		N	N	N	N			
1221.72 RADAR DEVICE TUNING FORK TEST											4													
1221.73 MICROWAVE TRANSMISSION-																								
Frequency Stability	S																							
Input Current Stability																								
Radiated Power Output Stability																								
Antenna Horizontal Beam Width	S	2				S					3		S								S			
Antenna Near-Field Power Density						M				M					M		M							

NOTE: S = Noncompliance with the Model Specifications.
M = Compliance with the Model Specifications, but noncompliance with the manufacturer's specifications.
N = Noncompliance because no tuning fork calibration certificate was provided.

[1]Received at test site on November 30, 1983. Tests conducted during final phase.
[2]No test conducted. Antenna fell out after vibration test.
[3]Insufficient output power for this test.
[4]Used tuning fork from another radar, as incorrect ones were provided by manufacturer. No target display in moving mode.

TABLE 2—*Continued*

SUMMARY OF INITIAL TESTS

		Radar Device IACP Number																				
		Types I and III								Types II and IV												
		2556	2557	2561	2563	2565	2568	2570	2574	2578	2555	2558	2559	2560	2562	2564	2566	2567	2569	2571	2572	2573	2576	2577	2580
1221.74	ENVIRONMENTAL TESTS																								
	(a) Temperature Tests																								
	Low Temperature																								
	Tuning Fork Test(s)			S								4	S											S	
	Frequency Stability	S														S								S	S
	Input Current Stability	S				S										S			S					S	
	RAD Output Power Stability	3														S								S	
	High Temperature																								
	Tuning Fork Test(s)			S								4													
	Frequency Stability	S																							
	Input Current Stability	S							S									S	S						
	RAD Output Power Stability		3																S						
	(b) Humidity Test																								
	Tuning Fork Test(s)			6								4	S												
	Frequency Stability	S																S							
	Input Current Stability	S																S	S		S				
	RAD Output Power Stability																	S	S						
	(c) Vibration Test		S	*		7						5	7												
1221.75	LOW VOLTAGE SUPPLY	S	M	*		*	S	S	M	S		*	*	M		S	S		M	M					S
1221.76	DOPPLER AUDIO		S	*		*		S	S			*	*												
1221.77	SPEED MONITOR ALERT			*		*						*	*												
1221.78	POWER SURGE TEST			*		*						*	*												
1221.79	SPEED DISPLAY																								
	(a) Readability - Character Height			S			S					S	S	S					S	S	S	S			
	Luminance Contrast		S				S	S							S	S			S	S			S		9
	(b) Display Lock			*		*						*	*												
	(c) Display Clear			*		*						*	*												
	(d) Internal Circuit			*		*			S			*	*						S		S	S			
	(e) Speed Display Transfer			*		*						*	*												
	(f) Signal Processing Channel Sensitivity																								
	(1) Stationary Mode: 20-90 mph	S	S	*	M	S		S			M		*	S	M		S	S	M						S
	60-90 mph		S	*		S		S			S	S	*	S		S	S		S	S					
	(2) Moving Mode:																								
	Patrol 25 40-90 mph											S	*												
	Patrol 50 60-90 mph											S	*			S									
	(g) Target Channel Speed Display:																								
	(1) Low - Stationary			*	M	*					M	*	*						M		M				
	Moving										M	*	*	S	M		S								
	(2) High - Stationary			*	M	*					M	*	*			S	M		M		M				
	Moving										M	*	*										M		
	(3) Closing			*		*						*	*												
	(h) Patrol Channel Display:																								
	(1) Low Patrol Speed			*		*						*	*												
	High Patrol Speed			*		*					M	*	*												
	(2) Patrol Tracking											*	*			S									
1221.80	ELECTROMAGNETIC INTERFERENCE																								
	(a) Vehicle Alternator			*		*						*	*	10											
	(b) Ignition, A/C Heater Motor, Windshield Wiper Motor			*		*						*	*	*											
	(c) Police FM Transceiver			*		*			S			*	*	*							S		S		
	(d) Citizens Band (CB) Transceiver			*		*						*	*	*							S	S			
	OPERATIONAL TESTS																								
1221.81	ELECTROMAGNETIC INTERFERENCE																								
	(a) Vehicle Alternator			*		*							*												
	(b) Citizens Band Transceiver			*		*							*												
	(c) Adjacent Vehicle Interference-																								
	Police FM Transceiver			*		*	8					S	*												
	CB Transceiver			*		*		S	S				*												
1221.82	SPEED ACCURACY TESTS																								
	(a) Stationary Mode - Moving Patrol			*		*							*												
	(b) Moving Mode - Moving Patrol												*												
	(c) Stationary Mode - Moving Target			*		*		R					*					R		R					
	(d) Moving Patrol - Approaching Target																								
	Patrol 20 - Target 55											5	*			R				R		R			
	Patrol 55 - Target 70											5	*												

*Test could not be completed because of technical problems with the radar device.

[5]Testing terminated due to lack of target display in moving mode.
[6]Testing terminated due to lack of target display.
[7]Testing terminated due to failure of right-hand digit in target display.
[8]Displayed zero reading during time of interference.
[9]Red display only. Green display did not provide sufficient contrast.
[10]Testing terminated due to unusually low power density.

R = During the final testing phase, without adjustment or modification, at those conditions identified by R on the table, the speed accuracy of these devices was verified.

CONCLUSION

The model performance specifications development and the radar device testing program have led to a definitive improvement in the radar units that will be available in the future. Undesirable features, such as the automatic speed lock, have been removed and positive features have been added, including radio frequency-interference detection circuits that alert the operator to the interference and/or disable the radar speed display before spurious readings are encountered; low-voltage detection circuits that provide a warning indicator and/or disable the radar's speed display windows before spurious readings are encountered as a result of low voltage operating conditions. In addition, the performance specifications and the testing program have assisted the manufacturers in improving the readability of the speed-display windows, the signal-strength sensitivity versus speed characteristics in both stationary and moving modes, performance at both high and low temperatures and, finally, operator's manuals which provide radar users with key information regarding the operation maintenance and performance of the particular radar units.

The overall NHTSA/NBS/IACP program, including the testing that is described in this report, has produced improved radar devices that are better understood, are easier to operate, and have less probability for errors than their predecessors. Coupled with the Basic Training Program in Radar Speed Measurement developed by NHTSA, the performance of speed measuring radar devices has been improved signficantly.

The role of police traffic radar in traffic safety enforcement continues to be of critical importance. Police traffic radar devices provide a means of increasing enforcement effectiveness and thus enable police administrators to better cope with the scarcity of manpower resources and rapidly increasing fuel costs.

Highway safety and law enforcement officials should recognize the fallacy of purchasing radar devices solely on the basis of economy without due regard to their performance capabilities. Continued testing by IACP and individual law enforcement agencies, using IACP approved laboratories, will ensure that the radar units being manufactured and sold to law enforcement agencies meet or exceed the performance characteristics of those individual units that were tested and found to be qualified for the IACP CPL.

Recommendations

The IACP recommends that every agency procuring radar units (after January 31, 1984) require the successful bidder to certify that the radar units being supplied are included in the IACP Consumer Products List and meet or exceed the NHTSA/IACP model specifications. This dual certification is important because some manufacturers have indicated that model numbers on both conforming and nonconforming units may be identical.

The IACP recommends that, if an agency is purchasing a large number of units, it have one or more units tested to the model specifications at the manufacturer's expense. If the test unit does not comply with the model specification, then the procuring agency should have the option of allowing the manufacturer to correct the deficiencies or cancel the order and procure the radar from another qualified bidder.

The IACP also recommends that every officer operating a radar speed measuring device receive training that is equal to or greater than the Basic Training Program in Radar Speed Measurement developed by NHTSA.

BIBLIOGRAPHY

1. Basic Training Program in Radar Speed Measurement[1], DOT HS-900-070 through DOT HS-900-072, National Highway Traffic Safety Administration, U.S. Department of Transportation, 400 7th Street, S.W., Washington, D.C. 20590.

2. Field Strength Measurements of Speed Measuring Radar Units, NHTSA Technical Report No. DOT HS-805-928[2], June 1981, National Highway Traffic Safety Administration, U.S. Department of Transportation, 400 7th Street, S.W., Washington, D.C. 20590. (Also available as NBSIR 81-2225, same title, National Bureau of Standards, Washington, D.C. 20234.)

3. Police Traffic Radar, NHTSA Issue Paper, DOT HS-805-254, February 1980, National Highway Traffic Safety Administration, U.S. Department of Transportation, 400 7th Street, S.W., Washington, D.C. 20590.

4. Model Performance Specifications for Police Traffic Radar Devices, DOT HS-806-191, March 1982, National Highway Traffic Safety Administration, U.S. Department of Transportation, 400 7th Street, S.W., Washington, D.C. 20590.

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[1]Copies may be obtained from the Superintendent of Documents, Stock Number 050-003-00412-0, Government Printing Office, Washington, D.C. 20402.
[2]Copies may be obtained from the NTIS, Springfield, Virginia 22162, under #PB 81-240 079.

APPENDIX A

CONSUMER PRODUCTS LIST

Police Traffic Radar Speed Measuring Devices

CONSUMER PRODUCTS LIST (CPL)

January 31, 1984

Revised April 30, 1984

Manufacturer	Model	Radar Band	Type	Compliance
Broderick Enforcement Electronics (BEE)	BEE-36 (K)	K	IV-Moving	Full
	BEE-36 (X)	X	II-Moving	Full
CMI (Federal Signal Corp.)	Speedgun Magnum	X	II-Moving	Full
Decatur Electronics	MVR-715	X	II-Moving	Full
	MVR-724	K	IV-Moving	Full
	RA-GUN *KN-1*	K	III-Stationary	Full
	RA-GUN *GN-1*	X	I-Stationary	Full
Kustom Quality Electronics	Falcon	K	III-Stationary	Full
	HR-8	K	III-Stationary	Full
	HR-12	K	IV-Moving	Full
	KR-10SP	K	IV-Moving	Full
	KR-11	K	IV-Moving	Full*
	Road Runner	K	III-Stationary	Full
	Trooper	K	IV-Moving	Full
MPH Industries, Inc.	K-15 (K)	K	III-Stationary	Full
	K-15 (X)	X	I-Stationary	Full
	K-35 (K)	K	III-Stationary	Full
	K-35 (X)	X	I-Stationary	Full
	K-55 (K)	K	IV-Moving	Full
	K-55 (X)	X	II-Moving	Full
	S-80 (K)	K	IV-Moving	Full
	S-80 (X)	X	II-Moving	Full
	S-80MC (K)	K	IV-Moving	Full
	S-80MC (X)	X	II-Moving	Full

* The KR-11 was in full compliance with the standard except for the acceleration testing requirement. The unit did not give any false readings, but its microprocessor program prevents the KR-11 from reading speeds unless the patrol speed is stable. Therefore the KR-11 passes the intent of the test and the standard.

PERFORMANCE TEST CENTER (PTC)
INTERNATIONAL ASSOCIATION OF CHIEFS OF POLICE
THIRTEEN FIRSTFIELD ROAD
GAITHERSBURG, MARYLAND 20878
(301) 948-0922
(800) 638-4080

Police Traffic Radar Speed Measuring Devices

In 1976, the International Association of Chiefs of Police (IACP) passed a resolution calling for Federal government concern and involvement in the development of health, safety, and performance standards for speed measuring devices, testing of the devices, and publication of the test results. In August 1977, the National Highway Traffic Safety Administration (NHTSA) entered into an interagency agreement with the Law Enforcement Standards Laboratory (LESL) of the National Bureau of Standards (NBS) to develop performance standards for police speed measuring devices. On January 8, 1981, the draft performance standard for speed measuring radar devices appeared as a proposed rulemaking in the Federal Register (23CFR1221). Following an extensive review and comment period, the NHTSA published the revised standard as Model Performance Specifications for Police Traffic Radar Devices (DOT-HS-806-191) in March 1982. The IACP subsequently adopted these specifications, at the recommendation of the Technology Assessment Program Advisory Council, as an IACP specification for radar devices.

During the time following issuance of the proposed rulemaking, the manufacturers of radar devices have continued to make modifications to their products directed toward achieving compliance with the model specification. Publication of the model specification prompted an IACP proposal to test speed measuring radar devices to determine compliance with the model specification and to prepare a consumer products list in the same manner that other types of equipment were being tested by IACP as part of the National Institute of Justice (NIJ), Technology Assessment Program. The unsolicited IACP proposal was accepted and in October 1982, the IACP entered into a cooperative agreement with NBS/LESL to accomplish the testing of radar devices and publish the test results.

Consistent with the procedures used for the NIJ Equipment Testing Program, the IACP issued a request for proposal for the testing by independent testing laboratories and received over 50 requests for the documentation. Of those laboratories submitting proposals, two were selected: Dayton T. Brown, Bohemia, Long Island, New York; and Michigan State University, East Lansing, Michigan. Each laboratory was given one radar device for preliminary testing. A team of experts from NBS, NHTSA, and IACP visited each laboratory to ensure that the laboratories had qualified personnel, the proper test equipment and were conducting the test in the proper manner.

The radar manufacturers submitted their radar devices to the testing laboratories in June-July 1983 and initial testing was completed by October 1, 1983. Based on the initial test results, the manufacturers were afforded an opportunity to make minor changes in their radar units before the final testing phase. The final testing was completed and reports were received at IACP on January 16, 1984. The Consumer Products List on the following page is the result of the aforementioned testing. A full test report will be available on or about March 31, 1983.

The IACP recommends that every agency procuring radar units after January 31, 1984 require the successful bidder to certify that the radar units being supplied are included in the IACP Consumer Products List and meet or exceed the NHTSA/IACP model specifications. The certification is important because some manufacturers have indicated that model numbers on both conforming and noncomforming devices may be identical. The IACP intends to monitor the performance of speed measuring radar device production units through random testing of units purchased by individual departments.

APPENDIX B

LIST OF MANUFACTURERS

Appendix B: List of Manufacturers

Broderick Enforcement Electronics
7155 Antigua Place
Sarasota, Florida 33581

CMI, Incorporated
P.O. Box 38586
Denver, Colorado 80238

Decatur Electronics, Incorporated
715 Bright Street
Decatur, Illinois 62522

Federal American Research (None Tested)
41011 Highway 6
Minturn, Colorado 81645

Kustom Quality Electronics, Incorporated
8320 Nieman Road
Lenexa, Kansas 66214

M.P.H. Industries, Incorporated
15 South Highland
Chanute, Kansas 66720

Tribar Industries, Ltd. (None Tested)
Muni Quip Radar Division
3650 Weston Road
Weston, Ontario, Canada M9L 1W2

Battelle
. . . Putting Technology To Work
505 King Avenue
Columbus, Ohio 43201-2693
Telephone (614) 424-6424
Facsimile (614) 424-5263

January 18, 1991

Dr. James Wasil
Technology and Science Advisor
Division of Safety and Hygiene
Bureau of Worker's Compensation
246 North High Street
Columbus, OH 43215

Dear Dr. Wasil:

INTRODUCTION

This letter report presents the results of electromagnetic environment measurements performed by Battelle on December 13, 1990. The purpose of the measurements was to provide the Ohio State Patrol (OSP) with quantitative data with which to assess radiation hazard risks associated with the use of communications and radar equipment. In addition, an abbreviated radiation level measurement was performed with a handheld transceiver, which would be in close proximity to the user's body in normal use. Measurement results are compared with two relevant radiation exposure limit standards. Generally, the measured levels were well below the standard limits. In a small number of specific cases, measured levels exceeded the limits and some changes in equipment location and/or operation may be warranted.

MEASUREMENT TECHNIQUES

The device used to determine the electromagnetic energy levels is a Narda model 8611 radiation probe. The level is read directly from a front panel meter and is expressed in units of milliwatts per square centimeter (mW/cm^2). This is an expression of the amount of power over an area, or power density.

Six patrol vehicles were tested to provide a variety of equipment configurations. These variations included antenna mounting locations, types of vehicles, and the presence of a cage. Measurements were taken at four passenger locations and four locations outside each vehicle. With the probe at each of these locations, the various transmitters were triggered to radiate individually, and the resulting power density recorded. The probe was moved for a peak reading within proximity of the selected location. The handheld measurements consisted of two tests: (1) measuring power density levels by scanning the body while the radio was (a) placed on belt, and (b) held at face level, and (2) searching for a maximum free-space level in the immediate vicinity of the radio, while the radio was held away from the body.

PRESENTATION OF DATA

Figures 1 through 12 present the results of measurements in graphic form. Referring to Figure 1, the top portion is a view looking down at the vehicle, with the vehicle make and serial number annotated in the upper left corner. To the right of the vehicle depiction is an antenna list showing antenna location and characteristics of equipment connected to them. (The letters on and around the vehicle represent measurement locations.) On the lower portion of each figure is a chart of the levels measured at each location for each source. If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

The diagrams are self explanatory, but a few special cases will be noted as follows. In Figure 5, the only example of an 800 MHz test was performed with a 1 watt cellular telephone connected to an on-glass antenna. This may be useful for future applications involving 800 MHz equipment. Also in this figure are the results of a higher power speed radar. Figure 7 gives maximum power densities for 45 and 155 MHz radios employing disguise antennas. The 45 MHz antenna is a cowl mount, and for 155 MHz a glass mounted cellular look-alike antenna is employed. Figure 8 gives the maximum power densities for the handheld radio test in three locations. Finally, the last four tests were with the higher power, window mount style radar. These tests were performed with the radar antenna mounted at three inside locations and one outside.

CONCLUSIONS AND RECOMMENDATIONS

Table 1 lists the non-ionizing radiation (NIR) exposure limits, at the frequencies corresponding to the equipments measured, for the ANSI* and OSHA** standards.The ANSI standard applies to the general public; the OSHA standard to occupational exposures. We believe that both standards are relevant to OSP -- the occupational standard for OSP officers and personnel,

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* American National Standards Institute, ANSI C95.1 - 1982

** Occupational Safety and Health Administration (OSHA); Code of Federal Regulations (C.F.R.) Section 1910.97, "Nonionizing Radiation".

and the public standard for passengers and bystanders. However, this distinction is an assumption on our part and should be clarified with regulatory authorities.

TABLE 1. RADIATION LIMITS FOR FREQUENCIES OF INTEREST		
Limit (mW/cm^2)		
FREQUENCY	ANSI	OSHA
27.065 MHz	1.2	10
45.020 MHz	1.0	10
155.370 MHz	1.0	10
465.000 MHz	1.6	10
800.000 MHz	2.7	10
10.525 GHz	5.0	10

Although these standards specify definite limits, they should not be construed as representing a sharp boundary between a hazard and a non-hazard. They are *regulatory* limits, somewhat analogous to a speed limit. They are derived from a large body of experimental data (sometimes contradictory), some subjective judgement concerning the magnitude of an adequate safety factor, and some discernment of what constitutes an acceptable risk in light of the perceived benefits from utilization of the electromagnetic spectrum. In the latter case, the risks are biological and the benefits are (largely) economic, so there is a good deal of controversy concerning the proper trade-off. Wherever the appropriate exposure limit lies, radiological health experts agree that individuals having control over (their own or others') exposure to NIR should strive to keep the exposure "as low as reasonably achievable".* That is, the less the exposure, the lower the risk. Nevertheless, compliance with the appropriate regulatory limit is required of all users of electromagnetic energy. For the reasons cited above, the measurements reported here can be discussed only with respect to the exposure limit standards and not in terms of biological hazards.

Analysis of the data shows that, as expected, the 465 MHz vehicular repeater, 27 MHz CB, and 800 MHz Cellular Telephone are sufficiently low in power to be of little concern. It was always possible to exceed the ANSI limit at 45 MHz while standing at the antenna mounting location. In two vehicles with cages, high power densities were observed at points in the passenger compartment at 45 MHz. Similar effects were observed at 155 MHz; the cages apparently enhancing coupling within the vehicle.

The 25 mW radars currently in use never exceeded ANSI or OSHA limits even with the probe placed in close proximity with the antenna. Under normal use with

---

* abbreviated as ALARA, in the technical literature.

the antenna aimed forward or at 45 degrees, there was no measurable radiation, and no observable leakage from the sides and back of the antenna. With the antenna aimed rearward, exposure would result if someone looked closely at the antenna lens or leaned over, blocking the radar with their chest. It must be remembered that the radar antenna is highly directional and does not produce coupling effects as seemed to occur with the VHF equipment. There was no appreciable radiation measured outside the vehicles at 10.525 GHz, even with the radar antenna aimed forward. Very different results were produced by the 120 mW, window mount radar. As Figures 10-12 indicate, it was possible to experience levels in excess of 10 mW/cm^2 at any of the three internal mount locations. What is particularly of concern about these locations is that the point of maximum signal is at passenger head level. This practice should be reconsidered and it is suggested that, if window mount radar antennas are used, they should be mounted externally at all times. It is understood that these units are no longer employed by OSP.

Finally, the handheld radio tests showed levels to be considered, but it is difficult to determine if a concern is present. The levels were below ANSI standards in both the belt mounted and face positions.

Little is known about long-term effects of electromagnetic radiation exposure, and some parties contend that the current hazard limits are not very well founded. Until more is known, awareness of potential hazards can reduce fear, and education for those working around electromagnetic sources is a responsibility that must be shared. There are, according to ANSI and OSHA standards, some potential areas of concern in and around OSP vehicles. Some could be reduced with a different vehicle configuration, as in the case of cage coupling effects. Others can only be dealt with through education and awareness, as in the case of the antennas for the high power VHF radios. Keep in mind, however, that although some of the measured power densities around these antennas exceed the Standards, the real safety and health problem is long-term, continued exposure.

We appreciate the opportunity to work with OSP, and look forward to providing further assistance, if needed. Please call me at 614-424-6175 with any questions that you may have.

Sincerely,

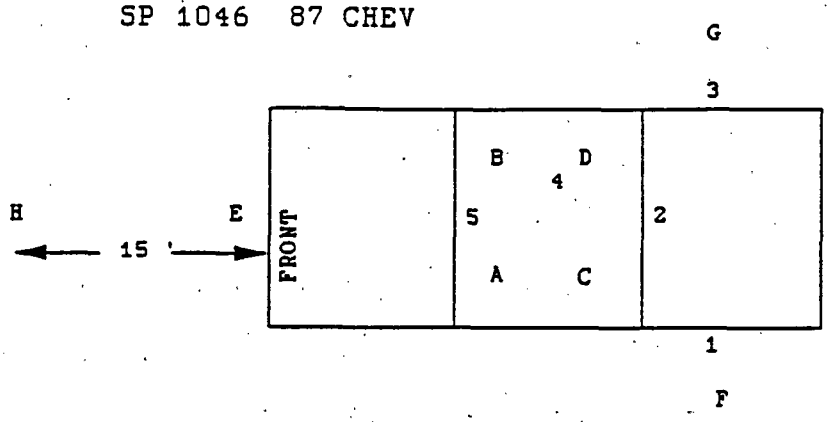
Robert T. Whitacre
Research Scientist
Electromagnetics Section

RTW:rrr

Attachments (Figures 1-12)

cc: Major Don Mack
State Highway Patrol
660 E. Main Street
Columbus, OH 43205

SP 1046 87 CHEV



ANTENNA LOCATIONS:

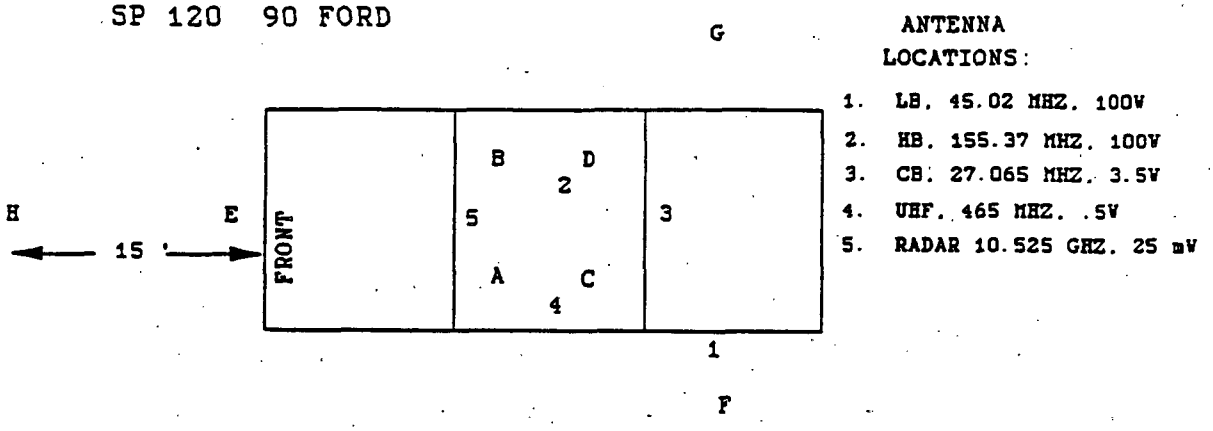
1. LB. 45.02 MHZ. 100V
2. CB. 27.065 MHZ. 3.5V
3. HB. 155.37 MHZ. 100V
4. UHF. 465 MHZ. .5V
5. RADAR 10.525 GHZ. 25 mV

	POWER DENSITY (mW/cm^2)								
	POSITION								
DEVICE	A	A,L*	B	C	D	E	F	G	H
45.02 MHz	0.40	0.05	0.40	0.30	0.50	0.50	10.0	0.5	.4
155.37 MHz	0.08	0.02	0.07	1.70	1.00	0.04	00.4	4.0	--
10.525 GHz** Facing Rear	--	--	--	--	--				
27.065 MHz	--	--	--	0.03	0.02				
465.00 MHz	--	--	--	--	--				
10.525 GHz Facing Forward	--	--	--	--	--	0.20			--
10.525 GHz ≈45° Right	--	--	--	--	--				

*A,L=Driver's Leg.
**Maximum=1.0 mW/cm^2 with probe 12" from radar antenna.

FIGURE 1. POWER DENSITY DATA FOR VEHICLE SP1046

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

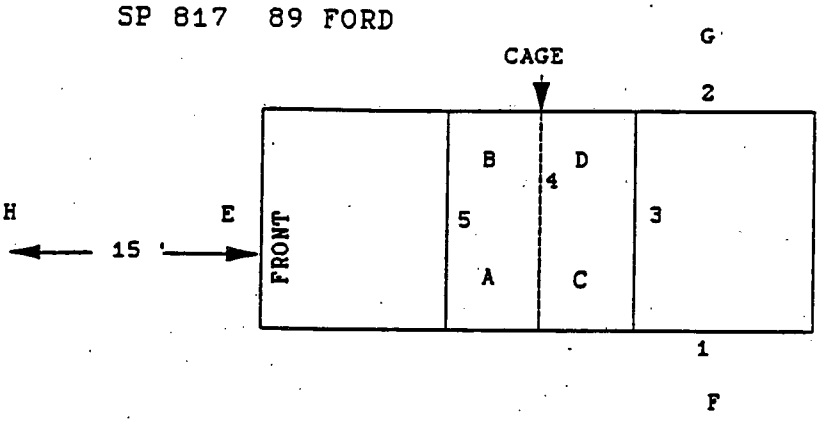


	POWER DENSITY (mW/cm^2)								
	POSITION								
DEVICE	A	A,L	B	C	D	E	F	G	H
45.02 MHz	0.6	0.12	0.20	0.50	0.20	1.00	5.0	3.0	.08
155.37 MHz	0.3	0.03	0.04	0.17	0.05	0.15	0.3	1.0	.01
27.065 MHz	--	--	--	0.04	0.10				
465.00 MHz	--	--	--	--	--				
10.525 GHz* Rear Facing	--	--	--	--	--				
10.525 GHz Forward Facing	--	--	--	--	--	--			
10.525 GHz 45° Right	--	--	--	--	--				
*Max in front .7 at 12". Max in back .2.									

FIGURE 2. POWER DENSITY DATA FOR VEHICLE SP120 (NOTE LOCATION OF 155 MHz ANTENNA.)

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 817 89 FORD



ANTENNA LOCATIONS:

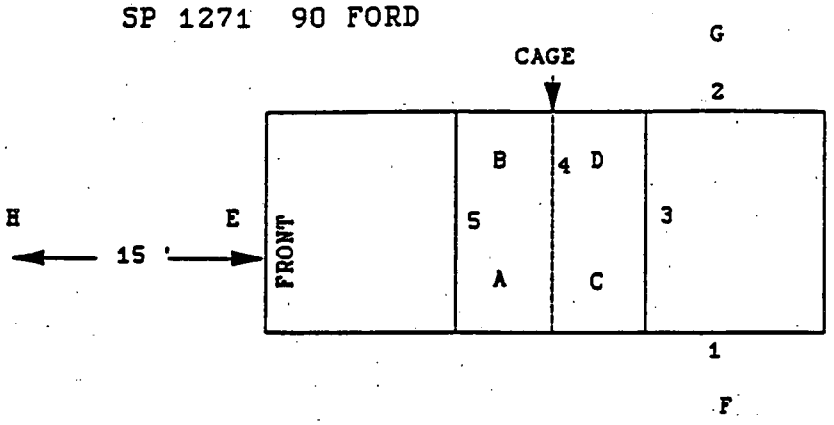
1. LB. 45.02 MHZ. 100W
2. HB. 155.37 MHZ. 100W
3. CB. 27.065 MHZ. 3.5W
4. UHF. 465 MHZ. .5W
5. RADAR 10.525 GHZ. 25 mW

	POWER DENSITY (mW/cm^2)								
	POSITION								
DEVICE	A	A,L	B	C	D	E	F	G	H
45.02 MHz	3.00	.06	.25	0.30	.20	.12	5.0	0.7	.04
155.37 MHz	0.03	.03	.04	1.20	.50	.10	0.3	4.0	.03
27.065 MHz	--	--	--	0.03	.04				
465.00 MHz	--	--	--	--	--				
10.525 GHz Rear*	--	--	--	--	--				
10.525 GHz Front	--	--	--			--			
10.525 GHz 45°	--	--	--						
*Max 1.0 at 6".									

FIGURE 3. POWER DENSITY MEASUREMENTS FOR VEHICLE SP817 (NOTE PRESENCE OF CAGE.)

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 1271 90 FORD



ANTENNA LOCATIONS:

1. LB. 45.02 MHZ. 100W
2. HB. 155.37 MHZ. 100W
3. CB.27.065 MHZ. 3.5W
4. UHF. 465 MHZ. .5W
5. RADAR 10.525 GHZ. 25 mW

ANOTHER VEHICLE PARKED ABOUT 10 FT IN FRONT OF THIS VEHICLE (POSITION H IS BESIDE FRONT VEHICLE DRIVER'S DOOR)

	POWER DENSITY (mW/cm^2)								
	POSITION								
DEVICE	A	A,L	B	C	D	E	F	G	H
45.02 MHz	.40	.12	1.00	0.7	.5	.20	4.0	0.6	.09
155.37 MHz	.05	.01	0.01	1.0	.5	.05	0.2	4.0	.01
27.065 MHz	--	.005	0.01	0.2	.9				
465.00 MHz	--	--	--	--	--				
10.525 GHz Rear*	--	--	--	--	--				
10.525 GHz Front	--	--	--	--	--	--			
10.525 GHz 45°	--	--	--						
*Max=.06 at 6".									

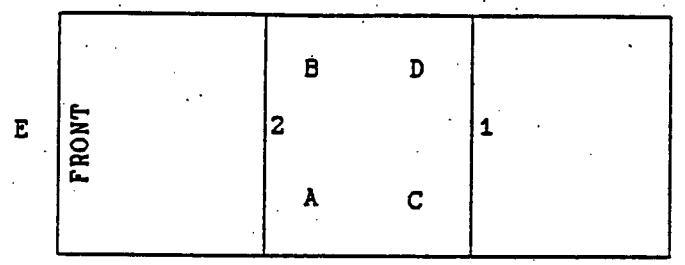
FIGURE 4. POWER DENSITY MEASUREMENTS FOR VEHICLE SP1271 (NOTE PRESENCE OF VEHICLE, SIMULATING TYPICAL TRAFFIC STOP.)

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 297 90 FORD

ANTENNA LOCATIONS:

1. CELLPHONE, 1W
2. RADAR, 10.525 GHZ, 60+mW



	POWER DENSITY (mW/cm^2)					
	POSITION					
DEVICE	A	A,L	B	C	D	E
10.525 GHz Rear Facing*	--	--	--	--	--	
10.525 GHz Front Facing	--	--	--	--	--	
Cellphone**	--	--	--	.03	.03	

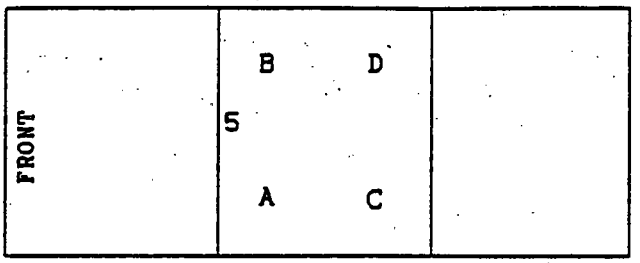
*Max=1.4 at 6". beam is wider
**Max=0.3 at center rear window.

FIGURE 5. POWER DENSITY MEASUREMENTS FOR VEHICLE SP297

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 842 RADAR ONLY

ANTENNA LOCATIONS:



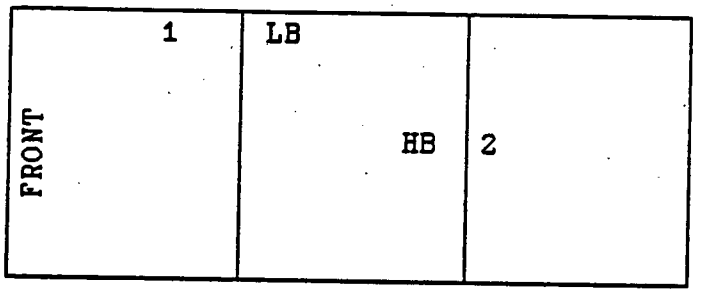
5. RADAR, 10.525 GHZ, 25 mV

	POWER DENSITY (mW/cm^2)			
	POSITION			
DEVICE	A	B	C	D
10.525 GHz Rear Facing*	--	--	--	--
10.525 GHz Front	--	--	--	--
Radar 45°	--	--	--	--
*Max=.8 at 6".				

FIGURE 6. POWER DENSITY MEASUREMENTS FOR VEHICLE SP842

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 765



ANTENNA
LOCATIONS:

1. LB, 45.02 MHZ, 100W
2. HB, 155.37 MHZ, 100W

MAX READINGS:
45.02 MHZ: 0.8 mW/cm^2
155.37 MHZ: 1.8 mW/cm^2

FIGURE 7. POWER DENSITY MEASUREMENTS FOR VEHICLE SP765 (THIS VEHICLE EMPLOYS DISGUISE ANTENNAS.)

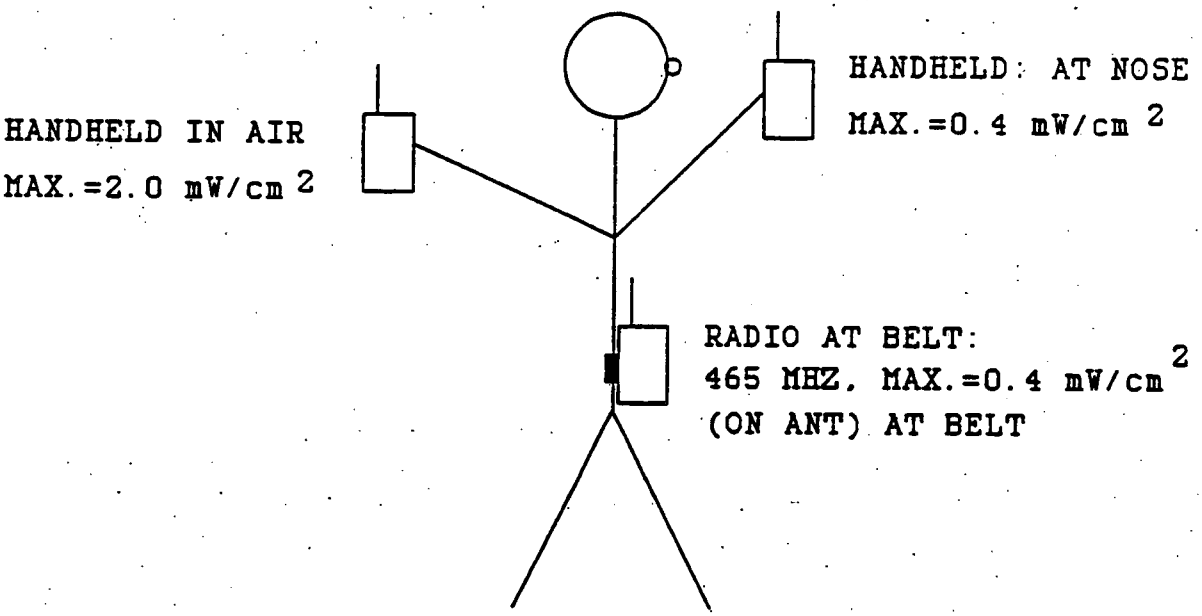
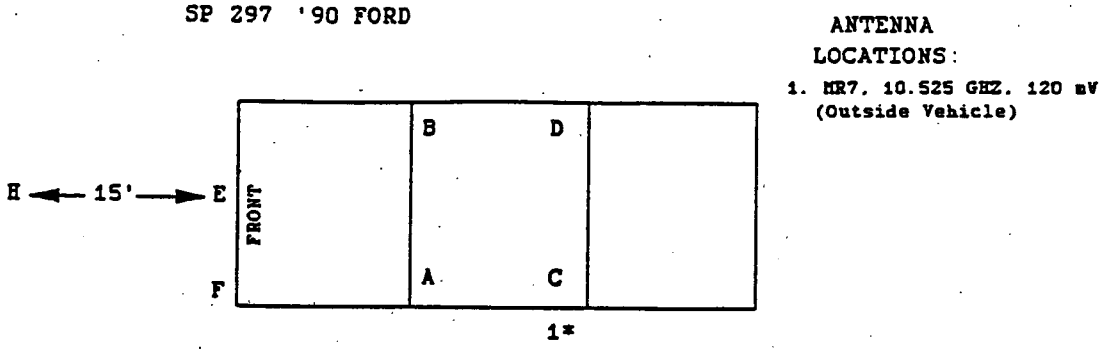


FIGURE 8. HANDHELD RADIO TESTS AT 465.00 MHz



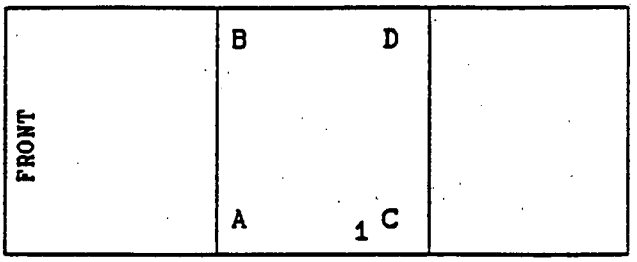
	POWER DENSITY (mW/cm^2)						
	POSITION						
DEVICE	A	B	C	D	E	H	F
10.525 GHz Forward Facing	--	--	--	--	--	--	4
10.525 GHz Rear Facing	--	--	--	--	--	--	--

FIGURE 9. POWER DENSITY USING HIGH POWER (120 mW) RADAR

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 297 '90 FORD

ANTENNA
LOCATIONS:
1. MR7, 10.525 GHZ, 120 mW
(Inside Vehicle)



	POWER DENSITY (mW/cm^2)			
	POSITION			
DEVICE	A	B	C	D
10.525 GHz Forward	16	--	--	--
10.525 GHz Rearward	--	--	--	--

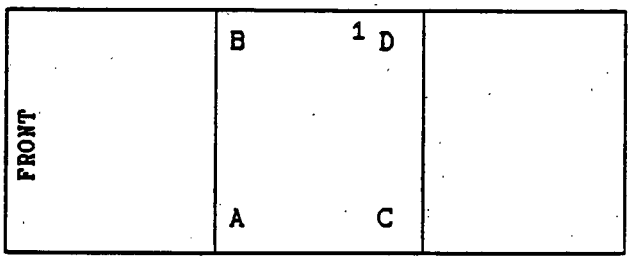
FIGURE 10. POWER DENSITY USING HIGH POWER (120 mW) RADAR

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 297 '90 FORD

ANTENNA LOCATIONS:

1. MR7. 10.525 GHZ. 120 mW (Inside Vehicle)



	POWER DENSITY (mW/cm^2)			
	POSITION			
DEVICE	A	B	C	D
10.525 GHz Forward	--	50	--	--
10.525 GHz Rearward	--	--	--	--

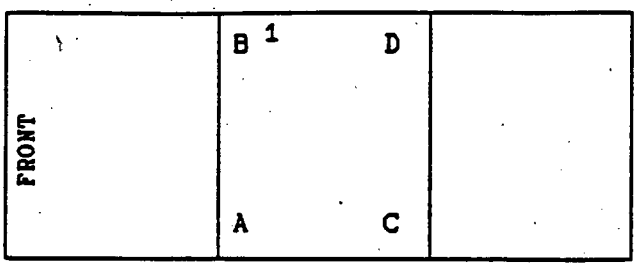
FIGURE 11. POWER DENSITY USING HIGH POWER (120 mW) RADAR

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

SP 297 '90 FORD

ANTENNA LOCATIONS:

1. MR7, 10.525 GHZ, 120 mW (Inside Vehicle)



	POWER DENSITY (mW/cm^2)			
	POSITION			
DEVICE	A	B	C	D
10.525 GHz Forward	--	--	--	--
10.525 GHz Rearward	--	--	--	40

FIGURE 12. POWER DENSITY USING HIGH POWER (120 mW) RADAR

If a "--" appears, this denotes a level that was below the sensitivity of the probe. If the space is left blank, a measurement at that location was not taken for that source.

BWC STATE OF OHIO WORKERS' COMPENSATION
30 W. Spring Street Columbus, Ohio 43266-0561

DIVISION OF SAFETY & HYGIENE

February 21, 1991

Major Don Mack
State Highway Patrol
660 East Main Street
Columbus, Ohio 43205

Dear Major Mack:

I was asked to assess radiation hazard risks associated with the use of communications and radar equipment from electromagnetic (non-ionizing radiation) measurements made by Battelle on December 13, 1990 and reported January 18, 1991.

After my initial phone conversation with you, Robert Whitacre, Research Scientist, Battelle, was contacted on February 15, 1991 in order to clarify some of the information presented.

Additionally, Mr. Ruggera, Engineer, Center for Devices and Radiological Health, (CDRH), Rockville, Maryland, (301) 443-3840; and Marcy Mathews, Health Physicist, Ohio Department of Health, Radiological Section (644-2727) were contacted to assist in the interpretation. Dr. Mays Swicord and Mr. Jack Monahan, both of the Division of Life Sciences, (CDRH) at (301) 443-7153 and 7192 respectively were referred to me. The CDRH is an excellent source of information concerning electromagnetic radiation.

CONCLUSIONS:

1. The 465 MHz vehicular repeater (UHF), the 27 MHz CB, and the 800 MHz Cellular telephone were of sufficiently low power to be of little concern. The K55 radar (with dash-mounted antennae), currently in use never exceeded ANSI or OSHA limits.

2. The handheld radios were not of concern, even with one result that exceeded ANSI. The ANSI standard has exclusion criterion to the protection guides in relation to fields from low power devices such as hand-held, mobile, and marine radio transceivers. These devices may emit localized fields exceeding the protection guides, but will result in a significantly lower rate of energy absorption than allowed for the whole body average. **Thus exposure to fields emitted by devices operating at 1 GHz or lower and at less than 7 watts output power would not be restricted.**

3. During normal transmission, the coupling effects from the cages should not cause excessive exposure. You stated that 2-way radios usually transmit for 30 seconds maximum. Even in cases where the radio is on for four to ten minutes, transmittance is not continuous during this time, and would not equal more than 5 minutes of cumulative transmittance. ANSI and OSHA standards are based upon 6 minute exposures.

4. If window mount radar antennas are used, they should be mounted externally at all times. The MR7 units are designed for outside. You stated that some officers have used them inside in inclement weather. During the monitoring, excessive levels were measured when they were mounted inside the vehicle. Even though MR7s are not currently in use by the OSP, if they are used in the future, external mounting would be necessary.

5. There is the potential for a localized effect (i.e. burn) from holding onto an antenna. This should never be done.

You asked the following questions during our February 15th conversation:

Q: Can there be a cumulative effect over a working lifetime to the levels measured?

A: Mr. Ruggera stated that presently no one knows the answer to this question. There are no obvious cumulative effects. Studies and debates are ongoing. The standards are based on the EM radiation's ability to raise the body's temperature above the normal metabolic rate. In the Battelle report the conclusion is made, "Keep in mind, however, that although some of the measured densities around these antennas exceed the Standards, the real safety and health problem is long-term, continued exposure." Mr. Ruggera stated that this conclusion is inaccurate. He knows of no epidemiological studies that prove or disprove health problems from long-term, continued exposure.

Q: Should the ANSI or the OSHA standards be followed?

A: ANSI should be followed because it is more conservative. However, there is not a great difference between the two standards. Both are low level radiation. Mr. Ruggera stated that the ANSI standard is frequency dependent and was determined based upon the average man's height and resonance of the body in the 10 to 100 MHz region.

Q: Why are European standards more conservative than are OSHA and ANSI?

A: In general, they are not more conservative when identical parameters are stipulated (e.g. same distance from source for measurements.) Mr. Ruggera stated that interference standards of course are lower than health hazard standards. He said generally the scientific community worldwide is in agreement on the health hazard standards. There are isolated cases where European standards are more stringent. He mentioned a Russian standard. When researchers from CDRH questioned the Russian scientists, it was discovered they could not meet their own standards.

Q: All of the Battelle study results were taken with one device on at a time. You stated that it is quite possible for the radar unit to be on during an entire 8 hour shift. What are the resulting exposures with concurrent radio and radar usage?

A: Mr. Ruggera stated that the power density results as measured in mW/cm^2 would be additive. However, in the 10 to 100 MHz region, the resonance effects in the body would also have to be considered and would increase.

Again, with normal radio usage, and with mounting the high power (MR7) radar antennas outside, no cumulative overexposures would be expected.

Q: You asked if there should be a concern for electronic technicians who repair radar equipment. They can be within 4 to 5 inches of an antenna for 30 minutes to an hour at a time for up to four or five hours per day.

A: Mr. Ruggera stated that further studies are needed of the repair shop area. There is a potential for concern with the practices described (i.e. long exposure times at close range.)

If you have further questions, please do not hesitate to call me (466-5563) or any of the three people from the CDRH referenced earlier in this report.

Respectfully submitted,

Beth Purcell

Beth Purcell, C.I.H.
Director of I.H. & Engineering

cc: J. Wasil, Superintendent
J. Fograscher, Dir. of Ergonomics & Safety