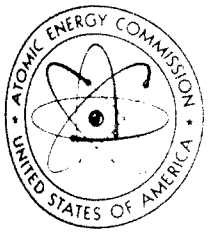
Proceedings of the Special Sessions on RADIATION TRANSPORT and BIOLOGICAL EFFECTS

held at Pittsburgh, Pennsylvania
November 1, 1966



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PROCEEDINGS OF THE SPECIAL SESSIONS ON RADIATION TRANSPORT AND BIOLOGICAL EFFECTS

Presented at the
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Sessions Chairmen:
R. E. Carter
and
J. I. Marcum

Proceedings edited by:
D. K. Trubey

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PREFACE

The proceedings of these sessions are being published as the result of the continuing policy of the American Nuclear Society Shielding Division to make available, by every possible means, information which is likely to be of value to its members. Deserving special recognition are the individual authors and their employers who devoted much time and effort not only to making the sessions in Pittsburgh a success but also in following through in a cooperative manner, making these proceedings possible.

Also deserving of special recognition are the following: the AEC Division of Technical Information Extension, who agreed to publish and distribute these proceedings; Eric Clarke, Technical Operations Research, who is Shielding Division Chairman and under whose general coordination these proceedings were prepared; J. R. Beyster, General Atomic, who as Shielding Division Program Chairman organized the sessions; and Mrs. Mildred Landay who graciously agreed to retype the manuscripts.

D. K. Trubey, Chairman
Publications Committee
ANS Shielding Division

Radiation Shielding Information Center
Oak Ridge National Laboratory
March 15, 1967

RADIATION TRANSPORT AND BIOLOGICAL EFFECTS

A SUMMARY OF SHIELDING DIVISION SPECIAL SESSION AT PITTSBURGH

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RADIATION TRANSPORT AND BIOLOGICAL EFFECTS

A Summary of Shielding Division Special Session at Pittsburgh

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The recent advances in using computer codes to solve radiation transport problems is causing many persons to think in terms of extending the techniques so that the radiation fields calculated can also be interpreted in terms of biological effects. The general awareness of this possibility provided the motivation for a special ANS session at Pittsburgh to bring together specialists in the area of radiation transport and biological effects for an interchange of ideas. This session, organized by the Shielding Division Program Chairman, J. R. Beyster of General Atomic, featured 12 speakers who either addressed problems of interpreting biological effects or reported developments in the use of computers to acquire detailed knowledge of energy deposition. This article will present first a synopsis of the problems of establishing criteria for radiation protection and then present summaries of papers reporting radiation transport calculations, followed by summaries of papers discussing biological effects.

RADIATION PROTECTION CRITERIA

Criteria for Radiation Protection; Permissible Dose to Critical Organs by Harald H. Rossi (Columbia University)

Dr. Rossi compared radiation protection criteria with highway speed limits. In both instances there is no absolutely safe limit. Any speed involves a certain risk, and the same must be assumed for any dose. Conversely, exceeding the limit does not necessarily involve a serious hazard, but it does tend to increase the risk.

It is sometimes argued that since knowledge of biological effects is incomplete, recommendations of permissible doses are questionable. This argument has only limited significance since detailed knowledge does not necessarily improve judgment.

The major factors which determine the biological effects of radiation are: dose magnitude, dose rate, radiation quality, volume irradiated, organ sensitivity, individual variability and age. In general the degree of effect depends on the magnitude

of these factors in a complex way. Some of the effects which are of particular importance to radiation protection are: leukemia and other cancers, aplastic anemia, cataract, aging, and genetic anomalies. Because of the complex interaction of the variables, extensive simplifications in the formulation of standards are necessary. For example, it is impractical to consider beam size in protection limits and so the maximum permissible dose is set for whole-body exposure which results in some "over-protection".

Perhaps the most important biophysical question in the formulation of protection standards concerns the existence of a threshold for radiation injury. There are certain indications that there should be no threshold for late effects due to chronic irradiation and it is altogether prudent to assume that risk is proportional to dose.

Since the permissible dose related to leukemia is rather small, this is among the most important limiting effects. This permissible dose is set at such a level that the leukemia incidence among radiation workers is almost indistinguishable from that of virtually unexposed populations.

Equal absorbed doses of various radiations can lead to different levels of effect, and it is consequently necessary to apply a weighting factor, known as a quality factor (QF), which allows for this difference. The quality factor is related to the linear energy transfer (LET) and in essence is equal to the -dE/dx of the charged particles. Its values are also selected conservatively for the most extreme case of low doses and dose rates in organ systems showing the largest quality effects. The product of the quality factor and the absorbed dose is the dose equivalent and its unit is the rem. Thus the term "maximum permissible dose" is, more precisely speaking, the maximum permissible dose equivalent.

The maximum lifetime doses permitted can only be received more or less uniformly during the professional life of a radiation worker. This is in part due to the requirement that the worker not use up his allowance before the end of his career. Another and even more important reason is to allow for maximum recovery and to avoid doses which are so high that the effect increases more than linearly. In addition, since children and adolescents may be expected to be more radiation sensitive, occupational exposure should not begin until an individual is 18 years old. All of these criteria are met by the requirements summarized in Table I, which limit the average annual dose increment to 5 rem after the age of 18. In cases where the accumulation limit has not been reached, an annual increment of 12 rem is permitted; however, this must again be divided more or less uniformly into at least four parts. Although various other conditions can exist, it is a common consequence of these rules that in areas fully occupied by radiation workers the maximum dose must be less than 10 mrem/week.

It is possible that under certain exposure conditions the dose received by the critical organ is comparatively low. This may occur in the case of external beams because of collimation or nonuniformity and in the case of internal emitters because of organ concentration. In such instances various other limits apply.

These and additional information may be found in Report 17 of the National Council on Radiation Protection (published as Handbook 59 of the National Bureau of Standards).

All of the limits mentioned thus far apply to the occupational exposure of radiation workers, i.e.,individuals who work in a controlled area under supervision of a radiation protection officer. For other members of the population the considerably lower limits shown in Table II are recommended. The reasons for the lower limits are that such exposures are involuntary, that they are more difficult to control, that the populations involved may be subject to other risks, and that they may include children. These reasons have prompted the adoption of a reduction factor of 10, making the maximum permissible dose equivalent for individual members of the general population 500 mrem/year.

TABLE I

BASIC LIMITS FOR OCCUPATIONAL RADIATION EXPOSURE

1.) The Dose Equivalent, D, to critical organs must at an age of N years meet the requirement:

$$D \leq 5\,(N - 18) \text{ rem} \quad .$$

2.) The Dose Equivalent received by critical organs in any 3-month period must be less than 3 rem.

There are higher limits for other organs or tissues.

TABLE II

LIMITS FOR NONOCCUPATIONAL EXPOSURE

1.) Individual (Somatic) Limit:

500 mrem/year, in addition to background and medical exposures.

2.) Population (Genetic) Limit:

An average of 14 rem in 30 years due to all radiations.

Fast Neutron Dose Weighting Factors for Manrated SNAP Reactors
by G. W. Spangler and C. A. Willis (Atomics International)

In spite of the unsolved problems, both biological and physical, related to energy deposition and organism response, protection criteria must be evaluated. For manned space missions, these authors find that short-term incapacitating effects from chronic, low-level exposure will not be limiting. Using carcinogenesis or life shortening as a basis gives the following criteria. For a 3-month mission

$$1 \geq \frac{D_n}{50} + \frac{D_\gamma}{200} \quad ,$$

where

D_n = fast-neutron dose, in rads,

D_γ = gamma-ray dose, in rads.

It is felt that additional experimentation is necessary, especially in connection with additivity questions.

TRANSPORT CALCULATIONS

Distribution of Dose and Dose Equivalent in an Anthropomorphic Phantom Resulting from Broad-Beam Sources of Monoenergetic Neutrons, by W. S. Snyder, J. A. Auxier, M. D. Brown, T. D. Jones, and R. T. Boughner (Oak Ridge National Laboratory)

These authors reported one aspect of a large program at ORNL to determine energy deposition in phantoms. They point out that any interpretation of neutron dose in terms of biological effects probably will need to take into account not merely a single value (for example, the maximum or midline dose) but will more likely need to consider the general pattern of dose in the body. Their Monte Carlo computer code made use of general geometry routines and a cross-section library developed at ORNL, as well as data compiled by the authors. Dose distributions as functions of LET and position were calculated in great detail.

Tissue Current-to-Dose Conversion Factors for Neutrons with Energies from 0.5 to 60 MeV by D. C. Irving, R. G. Alsmiller, Jr., and H. S. Moran

These authors also used the Monte Carlo method for energy deposition calculations. The high energies considered make their results particularly useful in the areas of accelerator and space shielding. Their results are for a 30-cm-thick slab. Although normalized to incident current, the results can be used with a known incident flux density by applying normalizing factors.

On Depth Dose Calculations in an Evaluation of Fallout Simulators and Fallout Field by R. L. French (Radiation Research Associates) and C. W. Garrett (Armed Forces Radiobiology Research Institute)

These authors also described detailed energy deposition in a phantom. The results here, however, were for real and simulated fallout gamma rays. The Monte Carlo method was again used, and the resulting distributions of dose due to a "compact simulator" were very similar to those from a real fallout field.

A New Technique for the Computer Representation of the Human Body by Philip Mittelman and Walter Guber (Mathematical Applications Group, Inc.)

This paper described a geometrical description technique with (a) minimum requirements of computer memory, (b) rapid tracking

of radiation through the configuration as required by Monte Carlo techniques, and (c) simplified geometrical input. Examples showing a simple body model built up from an elliptical cylinder, ellipsoids and a wedge, as well as a computer-produced description of a heart, served to illustrate the utility of the program.

Physical and Biological Dose Calculations for Proton and Alpha Fluxes Incident on a Shielded Man Model by C. W. Hill, K. M. Simpson, Jr., and W. B. Ritchie (Lockheed-Georgia)

A computer-oriented paper quite different from the preceding papers gave results for three types of man models: a sphere, two cylinders, and a reasonable facsimile of the human body. These results show significant differences between the various models. The calculation was a point kernel integration over solid angle which could account for detailed differences in the shielding with direction. The kernel was based on results obtained from detailed transport codes.

BIOLOGICAL EFFECTS

It is often the goal of the shield designer to calculate the radiation leakage in terms of dose equivalent. That is, the maximum permissible dose is specified in rem units and so some simple prescription is followed to convert flux density results to dose equivalent and yet often without the shield engineer appreciating the biological assumptions involved. The following papers were addressed to some of these matters.

Dose and Dose-Effect Relationship Modifying Factors in Predicting the Degree of Biological Response by V. P. Bond (Brookhaven National Laboratory)

Dr. Bond pointed out that the dose rate dependence is slight for most massive accidental exposures but rather depends on dose distribution on a macro scale. Dose rate becomes a major factor with exposure protracted over many months or years.

Radiation Responses of Man in the Intermediate Dosage Range by Wright H. Langham (Los Alamos Scientific Laboratory)

Dr. Langham considered higher rates than are usually associated with occupational radiation protection. For military or civil defense operations, emergency criticality situations, and possibly manned space exploration, it may be necessary to emphasize the amount of radiation a man can absorb and withstand. Most animal studies have concentrated on late effects (associated with low exposure levels) or lethal effects (associated with very high levels), leaving a dearth of information in the intermediate range. Much of this deficiency is due to a lack of suitable quantitative end points for evaluating the level, degree, or severity of effect. The somatic effects considered may be classified as follows:

1. Early effects (within 30 to 60 days)

    a. Skin erythema and desquamation
    b. Prodromal response
    c. Hematological depression
    d. Early lethality
    e. Decreased fertility and sterility

2. Late effects

    a. Permanent or delayed skin changes
    b. Increased incidence of cataracts
    c. Increased incidence of leukemia and other neoplastic diseases
    d. General life shortening

Each of these manifest a different response to varying degrees of exposure.

Cellular and Mammalian Radiation Effects and Their Interpretation in Relation to Manned Space Flight by Paul Todd (Pennsylvania State University)

A number of factors not normally considered as having effects on biological response were analyzed in this paper. Many of these factors act by affecting the reproductive capacity of stem cells in skin, bone marrow, and lymphatic tissues. Table III summarizes a number of interesting effects due to the interaction of certain agents and radiation.

TABLE III

Agent	Effect of Radiation on Response to Agent	Effect of Agent on Radiation Response
Radiation	Sensitizes	Sensitizes
Weightlessness	Additive with anemia and dehydration	Sensitizes chromosomes
Acceleration	Impairs vestibular function; weakens vascular and GI systems	Hypoxia may protect during exposure
Vibration	Sensitizes rodents	Sensitizes rodents
Heat	Additive?	Ultrasound sensitizes cells
Cold	Sensitizes rodents	Protects rodents
Excitement	Sensitizes GI tract?	Additive with intestinal cell loss?
Exercise	Reduces tolerance of rodents	Protects before; sensitizes after

Energy-Loss Distributions and Fractional Cell Lethality by Stanley B. Curtis (Lawrence Radiation Laboratory, Berkeley)

A potentially fruitful way of quantifying such biological effects is by using one or more inactivation cross sections. In this paper there was a discussion of these cross sections and their relation to particle energy loss, dE/dx. These cross sections are analogous to nuclear reaction cross sections in that they are the probability per unit flux density of the proliferative capacity of the cell being destroyed. Some of these cross sections have been measured by Paul Todd. This approach may become important in the future for the evaluation of the hazard from mixed-heavy-particle radiation environments when these or other suitable "malfunction" cross sections are available for critical or perhaps irreplaceable cells in the body and when accumulated damage over a long period, such as for extended flight, is of importance.

SUMMARY

A summarizing statement as follows was made by Dr. J. T. Brennan of the University of Pennsylvania

The most important thematic impression that emerges from having heard these 11 papers is a feeling that the finer complexities inherent in the biology of shielding are being recognized as proper targets for research. During the period 1945 to 1958, when atmospheric testing of nuclear weapons was going on, it gradually became evident that one could not continue to ignore the more difficult physical and biological parameters in radiation environments. At the beginning of that era, dose measurement devices were available which effectively integrated dose over 4π steradians. Similarly the exposed person, in his own way, often integrated dose over 4π steradians. For reasons of convenience it became the practice to hope that the instrument and the man would integrate in the same manner, making it unnecessary for the radiation hazard expert to investigate difficult problems such as the angular dependence of flux and energy spectrum. Similarly during the period 1952 to 1958, it became apparent that biological considerations such as dose rate and LET were more complex than one might have hoped, and existing knowledge was less adequate than had been thought for hazard evaluation purposes.

Throughout this session there was a suggestion that the advent of space radiation hazards has revived many of the same problems and has, in effect, given those concerned a second chance and a new reason for reattacking the more difficult parameters discussed above. Thus, we heard explained techniques for computer representation of the human body and techniques for the conversion of energy loss to cell lethality probability. Also, we heard a discussion of the need for a more refined evaluation of a depth dose pattern when one is considering sublethal doses.

These are the very questions that were unsolved in 1958 when the atmospheric testing of nuclear weapons came to a halt. It was most encouraging to note in these speakers a fresh appreciation of these problems, coupled with a renewed determination to

work toward their solution. The emergence of this attitude will be, in the long run, of greater importance than the degree of success which happens to have been achieved on any one facet at a given time.

Acknowledgement

The author is grateful for the cooperation of all the speakers of the session. Most of the material presented here was taken directly from summaries supplied by them. The assistance of Mrs. Lorraine Abbott is also gratefully acknowledged.

CRITERIA FOR RADIATION PROTECTION
PERMISSIBLE DOSE TO CRITICAL ORGANS

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CRITERIA FOR RADIATION PROTECTION
PERMISSIBLE DOSE TO CRITICAL ORGANS

Harald H. Rossi

Discussions of the principles of radiation protection frequently refer to the similarity between maximum permissible dose and highway speed limits. There are indeed a number of reasons why such a comparison is apt. Perhaps the most important of these relates to the fundamental fact that in both instances there is no absolutely safe limit. Any speed other than zero involves a certain risk, and the same must be assumed for any dose. Conversely, exceeding the limit does not necessarily involve a serious hazard; but a greater excess tends to increase the risk. There is thus in either situation no firm reason for the choice of a definite numerical value of the limit. The maximum permissible levels must be established by a judicious balancing of risk and benefit, but any values so chosen cannot be rigorously justified. Nevertheless, some numbers must be selected; and their arbitrary nature cannot be made an excuse for "speeding."

There are, however, also a number of important differences between the limits for highway speed and for radiation dose. In the case of radiation the hazard is almost exclusively controlled by the selection of the limit; and there is little, if any, opportunity for any analog to skillful driving. Furthermore, the number of individuals responsible for the determination of radiation levels is frequently much larger than that of those exposed to radiation, i.e., there is a much higher ratio of passengers to drivers. There is also less policing; some states still do not have legal radiation codes and even in those that do, inspection must be sporadic and enforcement is difficult.

All of these factors combine to indicate the need for a careful and conservative attitude, and there can be little doubt that the selection of maximum permissible doses involves far more thought and soul-searching than the selection of speed limits. As a result, it is much less risky to work in a radiation installation that adheres to radiation protection recommendations than to ride in a car that is driven in accordance with traffic regulations.

It is also apparent that the willingness to undergo personal exposure to radiation must involve an element of faith. This must not only be trust in the integrity and competence of those responsible for radiation protection but also confidence in the inherent sensibleness of the limits enforced by them. It is, therefore, desirable that the criteria for the selection of these limits be understood. Such an understanding is also particularly necessary on the part of shielding designers who must be assured of the validity of their efforts.

One of the most common criticisms levelled against those who select permissible radiation doses is that their recommendations must be questionable because knowledge of the biological effects of radiation is still incomplete. This argument has only limited significance. It should first be realized that knowledge, although it is a prime requisite for judgment, is not the only one; and that consequently increased knowledge does not necessarily improve the judgment. For example, the decision as to whether to raise the permissible dose to the general population by a certain amount is hardly made any easier by increasingly precise information on the relation between dose and the frequency of abnormal offspring. An approximate number is usually sufficient in view of such imponderables as progress, efficiency, etc., that also enter into the value judgment. Thus, the accuracy of information can often be inadequate for the objectives of radiobiology but sufficient for those of radiation protection.

Rather than to decry our ignorance of radiobiology, it would seem more pertinent to stress the fact that we already know far more about radiation effects than we can accommodate in a realistic set of protection regulations.

TABLE I

Major Biophysical Variables Influencing Radiation Effects

Dose
Volume Irradiated
Organ Sensitivity
Radiation Quality
Dose Rate
Individual Variability
Age

TABLE II

Major Late Effects of Chronic Irradiation

Leukemia
Other Cancers
Aplastic Anemia
Cataract
Aging
Genetic Anomalies

Table I contains a listing of major factors which determine the biological effect of radiation. It should be noted that in general the degree of effect depends on the magnitude of these factors in a complex way. In particular, in cases susceptible to numerical analysis the relation is usually not linear. Furthermore, there is a variable interaction between these factors. Thus, dose rate assumes different degrees of importance depending on radiation quality and individual organ sensitivity may change with age. All of these complicated relations and inter-relations are likely to be dissimilar for various biological effects. Some of these which are of particular importance to radiation protection are listed in Table II.

A consideration of our knowledge of the influence of the factors in Table I on the effects in Table II is impossible in the time available for this presentation. It is, however, also impossible in the formulation of practical radiation protection standards. Evidently extensive simplifications are necessary; and since most of the injuries listed in Table II are serious or fatal, any errors must be on the safe side. For example, it seems reasonable to assume that, other things being equal, the incidence of leukemia is - at least at low doses - proportional

to the amount of marrow exposed. Since it is impractical to consider beam size in protection limits, the maximum permissible dose must be set for whole body exposure. This, of course, results in "over-protection" when only a portion of the marrow is actually exposed.

Perhaps the most important biophysical question in the formulation of protection standards concerns the existence of a threshold for radiation injury. The only one of the effects listed in Table II which has not been demonstrated in man is genetic injury. However, it has been so extensively observed in experimental animals that its occurrence in man cannot be seriously questioned. Indeed, enough is known about its insidious nature to indicate that it would be very difficult to observe in first or second generation offspring, but that it should be very consequential in low level exposure of broad population segments. It also appears established that genetic effects are proportional to dose and independent of dose rate in the range of interest in radiation protection. By way of contrast the other effects in Table II have all been observed in heavily exposed individuals, but it is largely undecided whether they are subject to a threshold, i.e., a dose below which there is essentially zero effect. However, there are certain indications that there should be no threshold; and it is altogether prudent to assume that here also the risk is proportional to dose.

Since the risks are grave and since their probability cannot be reduced tc zero, the only alternative is to keep them at an acceptable level. The term "acceptable" refers here not only to other risks of everyday life but also to the normal incidence which in the case of all the effects listed in Table II can be caused by agents other than radiation. For example, leukemia happens to be a rather rare disease. Nevertheless, the permissible dose is set at such a level that it would be virtually impossible to detect even in a large population of radiation workers a leukemia incidence that is higher than that in populations which are virtually unexposed. Since this permissible dose turns out to be rather small, leukemia is among the most important limiting effects. The tissues that must be irradiated to cause leukemia, namely the blood-forming organs, are termed a critical organ.

If radiation workers were exposed to only one kind of radiation, permissible amounts would be expressed in terms of the absorbed dose which is the energy imparted by charged particle per unit mass of the irradiated tissue. However, equal absorbed doses of various radiations can lead to different levels of effect; and it is consequently necessary to apply a weighting factor known as the quality factor (QF) which allows for this difference. The quality factor is related to the linear energy transfer (LET; in essence equal to the $-dE/dx$ of the charged particles) and its values are again selected conservatively for the most extreme case of low doses and dose rates in organ systems showing the largest quality effects. The product of the quality factor and the absorbed dose is the dose equivalent and its unit is the rem. Thus what are termed "maximum permissible doses" are, more precisely speaking, maximum permissible dose equivalents.

It is evident that except in cases of local irradiation the exposure to radiation must be limited by the sensitivity of critical organs. As far as we know and to the extent to which sensitivities can be equated, it would appear that three organ systems have similarly high radiation sensitivity. These are the blood-forming organs, the reproductive organs, and the lens of the eye. If the whole body or any of these critical organs are irradiated, the most important dose limits apply.

The maximum doses permitted can only be received more or less uniformly during the professional life of a radiation worker. This is in part due to the requirement that the worker not use up his allowance before the end of his career. Another and even more important reason is the need to allow for maximum recovery and avoidance of doses which are so high that the effect increases more than linearly with dose. In addition, since children and adolescents may be expected to be more radiation sensitive, occupational exposure should not begin until an individual is 18 years old. All of these criteria are met by the requirements summarized in Table III. It will be seen that these limit the average annual dose increment to 5 rem after the age of 18. In cases where the accumulation limit has not been reached, an annual increment of 12 rem is permitted; however, this must again be divided more or less uniformly into at least 4 parts. Although various other conditions can exist, it is a common consequence of these rules that in areas fully occupied by radiation workers, the maximum dose must be less than 100 mrem in a week.

It is possible that under certain exposure conditions the dose received by the critical organ is comparatively low. This may occur in the case of external beams because of collimation or non-uniformity and in the case of internal emitters because of organ concentration. In such instances various other limits apply. These and additional information may be found in Report 17 of the National Council on Radiation Protection (published as Handbook 59 of the National Bureau of Standards).

All of the limits mentioned thus far apply to the occupational exposure of radiation workers, i.e., individuals who work in a controlled area under supervision of a radiation protection officer. For other members of the population the limits shown in Table IV are recommended. It will be noted that they are considerably lower. Some of the reasons for this are that such exposures are involuntary, that they are more difficult to control, that the populations involved may be subject to other risks, and that they may include children. These reasons have prompted the adoption of a reduction factor of 10 making the maximum permissible dose equivalent for individual members of the general population 500 millirem per year.

All of these permissible doses are additional to those which are more or less unavoidable. Depending on location, the natural background has been estimated to range from about 100 to over 300 millirem per year with a median near 150 mrem per year. The dose due to radiological procedures in medicine is of course quite variable, but the average is now believed to be in the vicinity of 50 millirem per year. The sum of such numbers is comparable

to the limit set for non-occupational exposure. Geneticists are nevertheless of the opinion that the limit for the entire population should still be somewhat lower than that allowed to individuals, and a dose equivalent of 14 rem per 30 years has been postulated as the maximum average population dose from all causes, including background, medical exposures, and all other sources of man-made radiation. It should be noted that with regard to genetic results, it makes little difference whether some dose D has been received by one individual or a dose D/10 has been received by ten individuals. However, the individual is protected against personal (somatic) effects by the limits for radiation workers and non-radiation workers given above.

NBS Handbook 59 is a very concise exposition of the basic principles of radiation protection, but it is nevertheless a document of some seventy pages. In the time available to me, I could cover only some of the most cardinal principles and recommendations; but I hope that despite its briefness this summary has given you an appreciation of the magnitude of the problems and of the basic soundness of the solutions.

TABLE III

Basic Limits for Occupational Radiation Exposure

1.) The Dose Equivalent, D, to critical organs must at an age of N years meet the requirement:

$$D \leq 5\,(N - 18) \text{ rem}$$

2.) The Dose Equivalent received by critical organs in any 3 month period must be less than 3 rem.

There are higher limits for other organs or tissues.

TABLE IV

Limits for Non-Occupational Exposure

1.) Individual (Somatic) Limit:

500 mrem per year in addition to background and medical exposures.

2.) Population (Genetic) Limit:

An average of 14 rem in 30 years due to all radiations.

FAST NEUTRON DOSE WEIGHTING FACTORS FOR MANRATED SNAP REACTORS

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FAST NEUTRON DOSE WEIGHTING FACTORS FOR MANRATED SNAP REACTORS

G. W. Spangler and C. A. Willis

Radiation protection considerations for space flight are different in many particulars from those for terrestrial operations. First, population genetic considerations are not limiting because few people will be astronauts. Second, irradiation over the individual's adult life need not be assumed because an astronaut will undertake only 1 or at most a few, missions. Third, the penalties, particularly weight penalties, from overconservatism in dose limits can be critical. Fourth, the fact that dose limits for space radiation based upon crew incapacitation* must be considered in establishing dose limits for reactor radiations. Fifth, the reactor radiation field characteristics (quantity, quality, direction, etc.) can be known with precision for each mission. Finally the space environment permits doses from a wide variety of types and energies of radiations not encountered in terrestrial operations. In summary, space use of reactors involves several unusual radiation protection considerations while requiring the use of minimal safety margins.

In the 1940's, H. M. Parker introduced the Relative Biological Effectiveness (RBE) concept to relate the effects of radiation of different quality and to provide a normalization factor for adding doses. The RBE is commonly defined by:

$$\text{RBE} = \frac{\text{Dose (rad) of 250 kVp x-rays required to produce a particular biological effect}}{\text{Dose (rad) of the radiation in question required to produce the same biological effect}}$$

Research has subsequently demonstrated that the RBE is not only a function of radiation quality [i.e., Linear Energy Transfer (LET)] it varies dramatically with the effect of interest; RBE is also a function of several other factors, including dose rate as well as total dose. Decreasing dose rate generally reduces the degree of effect of all kinds of radiations but this reduction of effect is most pronounced with low-LET radiation. As a consequence, the reduction of dose rate has the effect of increasing

*J. Billingham, D. Ewing and D. Robbins, "A Method of Evaluating Radiation Risk for Manned Space Flight", 14th Annual Meeting, Radiation Research Society, Coronado, California, February 13-16, 1966

the observed RBE for high LET radiation such as neutrons or protons. A wide range of RBE values is observed.

To eliminate ambiguity and simplify radiation control practice while ensuring adequate protection, the Quality Factor (QF) was recommended by the RBE Committee in 1962 to replace the "general" RBE values then in use. The LET-dependent QF is based on a mathematical extrapolation to zero dose rate for a particular effect. QF is defined only for total doses not exceeding the occupational exposure limits. The QF (or the National Bureau of Standards Handbook 59 RBE) can be averaged over the SNAP reactor neutron spectrum to give a weighting factor of about 6 for total fast neutron dose. However, as stated in the handbook and reiterated in the RBE Committee report, the present knowledge of biological effectiveness of radiations with different specific ionization does not warrant fine distinctions and the relationship between QF and LET cannot be regarded as having more than a very slight experimental basis. While the QF is convenient and legislatively required for normal terrestrial radiation control activities, it is neither suitable nor intended for other applications such as accident evaluations or radiation control in space.

An appropriate basis for the choice of fast neutron dose weighting factors in space applications is therefore not readily apparent.

Two possible sources for weighting factors do, however, warrant consideration: (1) existing directly applicable data and/or information from performance of experiments which duplicate exposure conditions of concern, or (2) extrapolations from the basic mechanisms of radiation effects so that dose information can be used directly in predicting the biological effects from the various combinations of radiations and exposure conditions. A review of existing information shows that experimental data relating to the biological effects from exposure conditions of concern are virtually nonexistent. Further, the mechanisms of radiation effects are not well enough understood to directly predict biological effects from exposure information, particularly when the additivity of various types of radiation is considered. Since no basis exists for a generally applicable weighting factor, a realistic choice can therefore only be made if the biological effects of interest are identified.

For crew incapacitation during the mission, the effects of interest are: (1) disturbances in the gastrointestinal tract (significant symptom is probably nausea), (2) cataract induction, and (3) skin erythema. The particular mode of exposure from a SNAP reactor can be characterized as a chronic, relatively low level exposure. Under these conditions the need for a dose limit to prevent serious gastrointestinal disturbances is obviated by the prevention of lethal doses. Similarly, the production of an incapacitating cataract during the mission would not be a consideration. With this chronic low level exposure mode, skin erythema would require many hundred rad, which would be unacceptable for the same reason. In summary, short term incapacitating effects from chronic exposure to low level radiation will not be

limiting.

From the preceding, it is clear that radiation from the reactor (alone) will not incapacitate the astronauts, but is not clear what effect the reactor radiation doses will have on resistance to incapacitating damage from solar flare radiation. Partial additivity has been experimentally demonstrated from some combinations of radiations and effects. Other experiments have shown beneficial effects from preirradiation. For gastrointestinal disturbances and skin effects there should be essentially complete recovery so the reactor radiation should have little effect on response to solar flares. Conversely, spontaneous remission of lens opacities is doubtful. While present data suggest that reactor radiation doses would not reduce resistance to solar flare radiation for short term effects, the additivity question has not been answered and further data is needed.

If the effect of chronic exposure on solar flare sensitivity is assumed negligible, crew incapacitation effects of chronic exposure cannot be limiting. There are complexities in using life span alteration as an objective. There are many mechanisms for radiation effects on life span and different dose levels are associated with each. Further, for small doses probabilistic factors must be considered. For lower doses and chronic exposure, carcinogenesis and unspecific "life shortening" are dominant considerations. The choice of a quantitative assurance of unaltered life span appears to be the logical basis for dose criteria.

Using carcinogenesis or life shortening (rather than crew incapacitation) as a basis gives the following criteria. If a 99% assurance of avoidance of carcinogenesis and less than approximately a one-year life span effect is acceptable for a three month mission, then the exposure limit would be about 50 rad fast neutrons or 200 rad gamma. For this mission the criteria is

$$1 \geq \frac{D_n}{50} + \frac{D_\gamma}{200}$$

Without further data complete additivity must be assumed. This criteria and the apparent weighting factor cannot be considered general because the neutron dose limit, the gamma dose limit, and the ratio of the two limits varies with changes in effects considered and with mission duration. For a limit based on probability of short term effects (which could be used for accident evaluation) a weighting factor of 2 or less appears appropriate. When long term effects are considered, neutron weighting appears warranted; but the weighting factor should be based on some acceptable damage or risk of damage at the pertinent dose rate.

From these analyses, it is evident that additional experimental investigations of these questions, particularly radiation additivity, are needed.

SNAP Reactors will be shielded to safe radiation levels; and by focusing attention on the effects of interest for each mission, shield weight can be minimized.

DISTRIBUTION OF DOSE AND DOSE EQUIVALENT IN AN ANTHROPOMORPHIC PHANTOM RESULTING FROM BROAD-BEAM SOURCES OF MONOENERGETIC NEUTRONS*

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ABSTRACT

A Monte-Carlo-type program has been coded for a digital computer to estimate dose in a tissue phantom from a variety of neutron sources with neutron energy not exceeding 14 meV. The program allows for elastic and inelastic scattering as well as for some 14 absorption interactions. The cross-section data are taken from BNL-325 or other literature as available, and remaining gaps in the cross-section data are filled by linear interpolation.

In some cases, information on the energies of secondary particles produced by an absorption interaction was not found in the literature and has been calculated so that both momentum and energy have been conserved in the interaction. The phantom consists of a right circular cylinder with a radius of 15 cm and a height of 60 cm and is thus a reasonable approximation of a human torso. It is considered to be homogeneous and composed of H, C, N, and O in the proportions indicated for standard man. With some rewriting of the source subroutine, the program may be used for a wide variety of sources, but only results for a monodirectional, monoenergetic, broad, lateral beam of neutrons are reported here. Results are obtained for a selection of energies ranging from thermal to 14 MeV. Over the range below 10 MeV, the maximum doses do not differ greatly from the maximum doses in slabs as given in NBS Handbook 63. However, a greater difference is found when the results for slabs and cylinders are compared at depths well below the irradiated surface. The variation of dose along a diameter perpendicular to the beam is found here and may be as much as a factor of 3 in some cases. In addition to giving dose in each of 150 volume elements of the phantom, the partition of these doses in each of 12 intervals of LET is obtained. Thus the quality of the radiation and the magnitude of the dose are provided for any desired portion of the phantom.

When the human body is irradiated unilaterally by neutrons, the resulting dose within the body generally varies appreciably in magnitude and in quality at different depths below the

irradiated surface. Bilateral or isotropic sources produce more nearly constant patterns of dose, but at some neutron energies there remains considerable variation in dose and dose equivalent in different regions of the body. Only at neutron energies of hundreds of Mev or more will the dose pattern be approximately constant within the body. Thus for neutrons of energy not exceeding 14 Mev--and this is the practical limit of the fission spectrum--neutron dose within the body can seldom, if ever, be adequately characterized by a single number. Any interpretation of neutron dose in terms of biological effects probably will need to take into account not merely a single value (for example, the maximum dose, or the midline dose), but will more likely need to consider the general pattern of dose within the body. The remainder of this paper is intended to illustrate this thesis by presenting quantitative results of recent studies of neutron dose in an anthropomorphic phantom. These studies have been the joint work of staff in the Health Physics Division, and have had extensive cooperation from the staff of the Mathematics Division, and the Neutron Physics Division of ORNL.

The basic program used in these studies is of straightforward, Monte Carlo type which, for the most part, follows the basic physical processes quite closely. The cross sections used for elastic and for inelastic scattering have been developed largely by the Neutron Physics Division and are in general use at ORNL. In addition, cross sections for 17 nuclear interactions have been programmed and used in the calculation. These include elastic neutron interactions of H, C, N, and O, inelastic interactions of C, N, and O, and n-α, n-p, and n-t reactions with N and O.(1-14)

There are many gaps in some of the measured cross sections, and these have been filled in largely by smoothly connecting the measured values. For example, the more important interactions with oxygen are probably those of the n-α_0 interaction (ground state) which shows the peaks in the region below 8 Mev and the n-α_2 reaction which dominates all of these processes at 14 Mev. In fact, the cross section of this N-α_2 interaction is about 12% of the total oxygen cross section at 14 Mev. These interactions are also of special importance because of the high LET values at which the dose from these α particles is deposited. Similar cross section data were used for the other interactions.

Some compromises with physical reality were necessary to keep within the capacity of the computer--an IBM-360, Model 75. Thus only an average energy was used for the alpha particles, neutrons, protons, tritons, and recoils produced by these nuclear interactions and by the inelastic scattering processes. This average energy was computed so that both momentum and energy were conserved for the interactions. The formula used for this average energy is shown as follows:

$$\bar{E}_2 = \frac{2 M_1 M_2 E_1}{(M_2 + M_3)^2} + \frac{M_3 Q + E_1 (M_3 - M_1)}{M_2 + M_3}$$

where

M_1 = mass of incident particle,

M_2 = mass of reaction product with energy E_2 ,

M_3 = mass of reaction product with energy $E_3 = E_1 + Q - E_2$,

Q = Q value for reaction,

E_1 = energy of incident particle,

$\bar{E}_2$ = average energy of reaction product with mass M_2 .

Figure 1 shows the linear energy transfer, or LET, of the various charged particles considered in this calculation. As you are aware, the biological effectiveness of the dose is thought to be influenced by the LET at which the dose has been deposited, and quality factors which depend on LET have been recommended by ICRP and NCRP [15] to convert dose in rad to dose equivalent in rem. Thus it is of some interest to know not merely the dose in rad but also what fraction of the dose was delivered in various ranges of LET so that the dose equivalent can be obtained. It is easy to see that if a charged particle, say, a proton, is produced with energy E, the energy absorbed with LET less than a value L_i is given by

$$\Delta_i E = \mathrm{Min}(E, a_i) + \mathrm{Max}(0, E - b_i)$$

where a_i and b_i are the proton energies at which the proton LET equals L_i. That is, if we consider the line on the figure where LET = L_i and if this line crosses the proton curve at proton energies a_i and b_i, then the formula gives the total energy deposited below a_i and above b_i, i.e., where LET is less than L_i.

Using this device, the fraction of dose deposited in 12 intervals of LET was estimated for each volume element where dose was recorded. Many of these intervals and quality factors are those directly recommended by ICRP and NCRP, [15] but we did add some additional intervals in order to obtain a better idea of the distribution of dose with LET, and the quality factor was linearly interpolated for these additional intervals from the recommended values.

Figure 2 shows the subdivision of the phantom into subregions for evaluation of dose. All cases calculated to date have been for broad beams of monoenergetic neutrons entering along the bisector of the angle of region 17. The phantom is a right circular cylinder of dimensions nearly those of the torso of a husky male, i.e., height 60 cm, radius 15 cm. The composition is homogeneous, consisting only of the four elements H, C, N, and O in the proportions typical of soft tissue with a density of 1. The energy carried by charged particles produced by an interaction within one of these subregions is assumed to be absorbed in that region. Photons are produced as a result of inelastic scattering or as a result of some capture processes, and a sample of such

photons are run on a separate program to estimate the dose due to these photons.

Figure 3 shows the distribution of dose with LET for neutrons of energies 2.5 Mev, 5 Mev, and 14 Mev. These are for volume elements at the irradiated surface, at the center, and at the greatest depth within the phantom. As you see, the distributions are quite different. Figure 4 shows the variation in LET for neutrons of energy 1 Mev at three different depths below the irradiated surface, namely, on the irradiated surface, near the center, and on the far side of the phantom. It is clear that the quality of the radiation varies greatly with distance from the irradiated surface. Many other examples of this variation in quality could be given. Since LET may plausibly be one of the parameters influencing the biological response, it seems clear that data of the type just shown are desirable if a close correlation of dose within a large animal and biological effects is attempted. One of the reasons for undertaking these studies is to provide data of this kind.

Figure 5 shows the dosimetric pattern within the central layer of the phantom for irradiation by neutrons of 14 Mev and 1 Mev. The quality factors have been used here as described earlier to obtain dose in rem. Of course, the dose in rad also is available. Figure 6 shows the similar data for neutrons of energies 1 kev and 0.025 ev.

The next three figures present the unsmoothed data in depth for neutrons of energies 2.5 Mev, 0.5 Mev and 0.1 kev. From these you can judge the general statistical accuracy we obtain with 10,000 neutron case histories as well as the surprisingly little variation we see from one tier to another. Figure 10 shows the trend of dose down the center of the phantom for neutron energies 10 kev, 5 Mev, and 14 Mev. The dose from capture gamma rays and the dose from neutron interactions are shown separately. Of course, each point represents an average dose over a wedge-shaped region as shown earlier. Here, again, the quality of the radiation will vary with both depth and neutron energy and so does the magnitude of the dose.

We turn now to a very practical situation, that of the determination of the dose pattern within the body for a practical situation. In 1958 a criticality incident occurred at one of the Oak Ridge plants, and five men were exposed to levels above 100 rad. Doses ranged from 236 to 365 rad, as estimated by Hurst, Ritchie, and Emerson.[16] Actually these values must be considered as the maximum doses within the bodies of these individuals. Less than a year later, a criticality incident occurred at Vinca in Yugoslavia, and essentially the same group of health physicists estimated doses to these individuals.[17] The six individuals with high doses were estimated to have received from 207 to 436 rad. The range of doses in the case of the Yugoslavs included the range of doses estimated for the Oak Ridge cases and extended upward beyond the Oak Ridge doses by about 20%, which does not seem to be a very marked increase in dose. Yet many have felt that the symptoms exhibited by the Yugoslavs were more severe than one might expect from the difference in dose. Perhaps this feeling is in part a reflection of the fact that one of the Yugoslavs

died. We cannot resolve this question at present, but we can, perhaps, use it to illustrate something of the complexity of the problem. The next three figures show the distribution of dose with LET in these two cases at depths of 1.5 cm, 15 cm, and 28.5 cm. These distributions have been obtained by using only the neutron spectra from the two sources as estimated and measured by the team of health physicists who "mocked up" each of the sources. Thus dose from photons incident on the body or phantom must be added to the doses shown in the lowest interval of LET, i.e., 0 to 3.5 kev/μ. Except for this lowest interval of LET, the distribution would not be changed due to this neglect of a part of the dose. However, our calculations use this spectrum only as a broad-beam, monodirectional source and thus do not take into account the effect of movements of the exposed individuals. Thus these results should be considered as qualitative evidence, and there might be some averaging of dose in different portions of the body due to the movements of the individual. Nevertheless, it is apparent that the radiation quality is markedly different in the two cases and the data on the dose levels within the body indicate that in the Oak Ridge cases some portions of the trunk received much less dose than the portion where the maximum occurred. This maximum would correspond to the estimated dose. You can see, too, how different the dose pattern would be if the individual had turned and had received half the exposure from opposite sides of his body, or the same dose was received in terms of Na activation of the blood but resulted from an isotropic source rather when it was an unidirectional source. This merely illustrates again that a single number does not serve to represent adequately the dose pattern within the body and that if any close evaluation of biological effects is undertaken, more information is needed.

The last figure gives the dose to a mouse-sized phantom and to a rat-sized phantom exposed to a beam of neutrons with the mass of the rat being about 8 times the mass of the mouse. The distributions of dose with LET in the two cases are shown for neutrons of three different energies. The dose is estimated for the volume element nearest the front surface, and the LET distribution of the dose is shown as a cumulative so that the value on the right represents the total dose. In all cases the mouse received more than the rat, and yet, intuitively, we feel the buildup must be greater in the rat than in the mouse. However, the computer gives the higher dose to the mouse. The computer is not wrong, and neither is our intuition; in changing the phantom size, we have also changed the dimensions of the volume elements. Thus the dose for the rat is averaged over a depth which is about twice as great as in the case of the mouse. At first one might feel that this is a poor way to present data, but after some thought about the matter we decided it was better to keep the volume elements in a fixed relationship to the whole phantom. In comparing biological effects seen in mouse, rat, and man, it may well be a poor way of looking at dose to compare only surface doses or only doses at a depth of 1 cm in each animal. A certain dose at one centimeter below the irradiated surface may have quite different significance for a mouse, a rat, and a man merely because of the different anatomical structures one finds at such a depth in the three cases. Perhaps, as a first approximation, one would do better to compare doses, not at the same

depth, but at corresponding depths at which the organs of concern are found. This scaling of the volume elements in proportion to the phantom is a first crude approximation to this and is mentioned here to emphasize that in extrapolating animal data on biological effects to infer results concerning human exposure we must consider the dose patterns within each of the bodies--and it may very well be quite different in magnitude and in quality in comparing a man with a mouse! But our thesis remains that for a really adequate assessment of exposure and for interpretation in terms of hazard or biological effects, one needs the entire dose pattern found within the body. We hope these studies will illustrate this thesis and that the much more extensive body of data we are producing will provide a more adequate basis for assessment and interpretation in terms of the biological effects.

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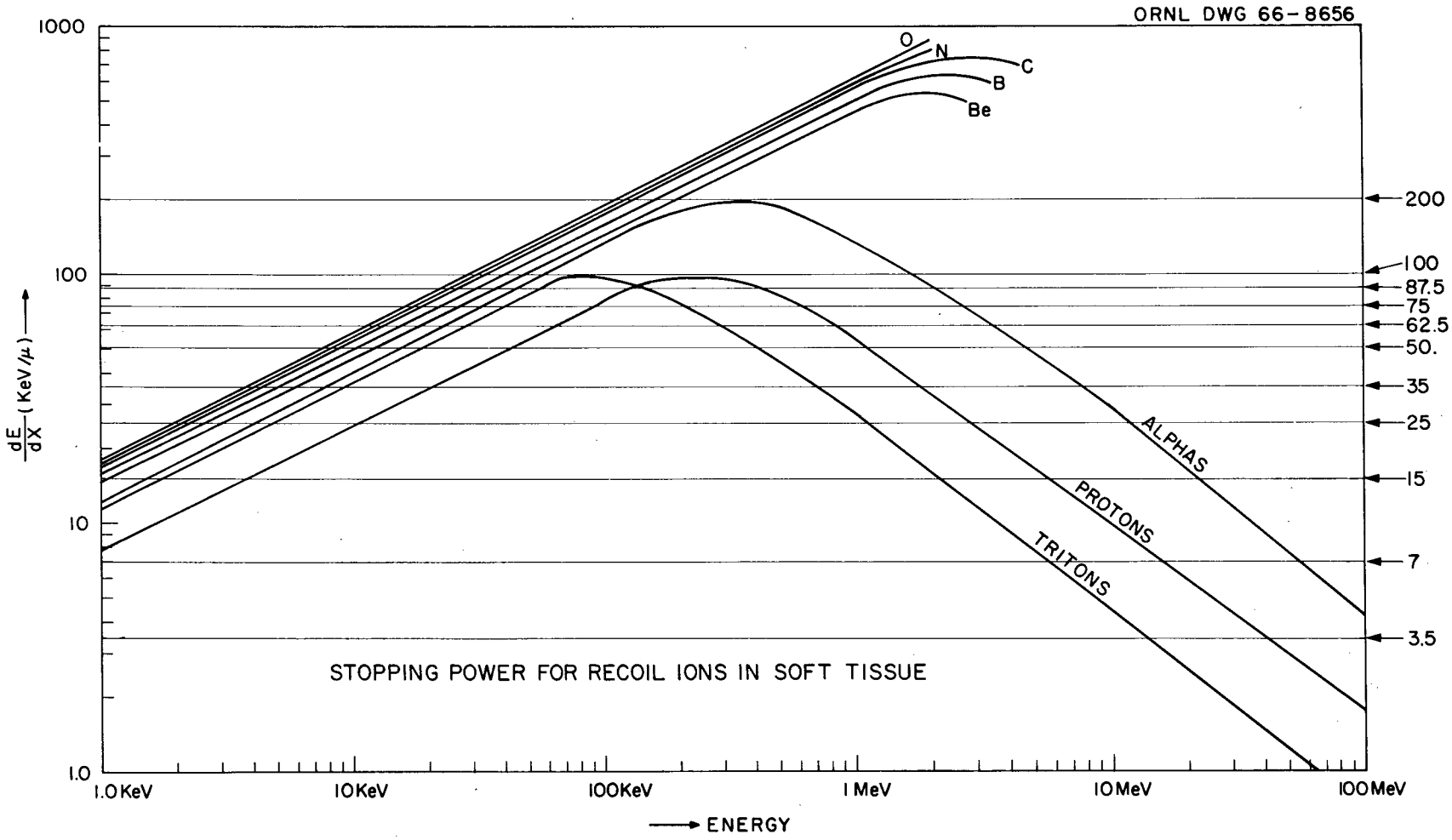

Fig. 1. Stopping Power for Recoil Ions in Soft Tissue as a Function of Particle Energy.

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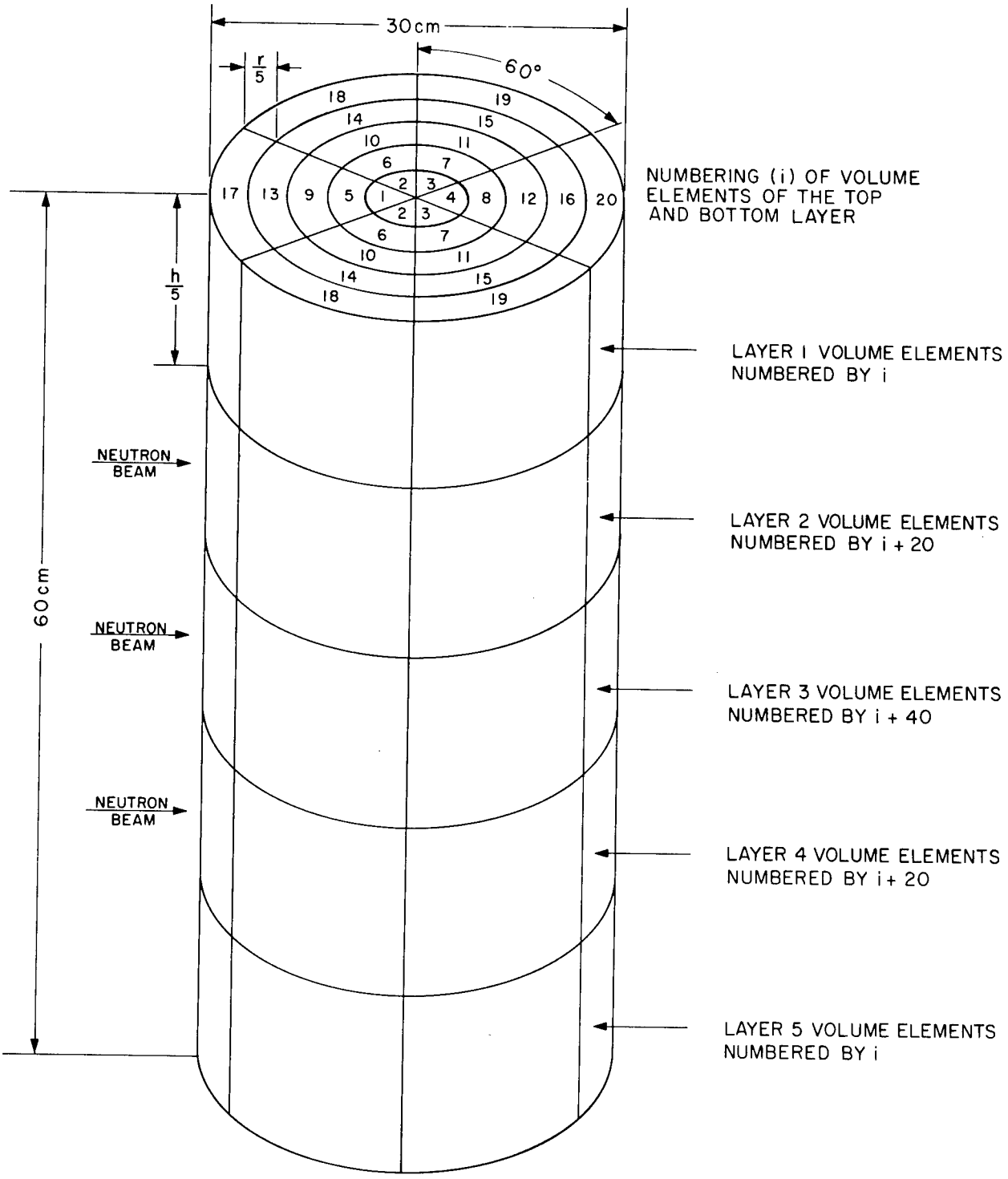

Fig. 2. Cylindrical Phantom Used in Dose and Dose-Equivalent Calculations.

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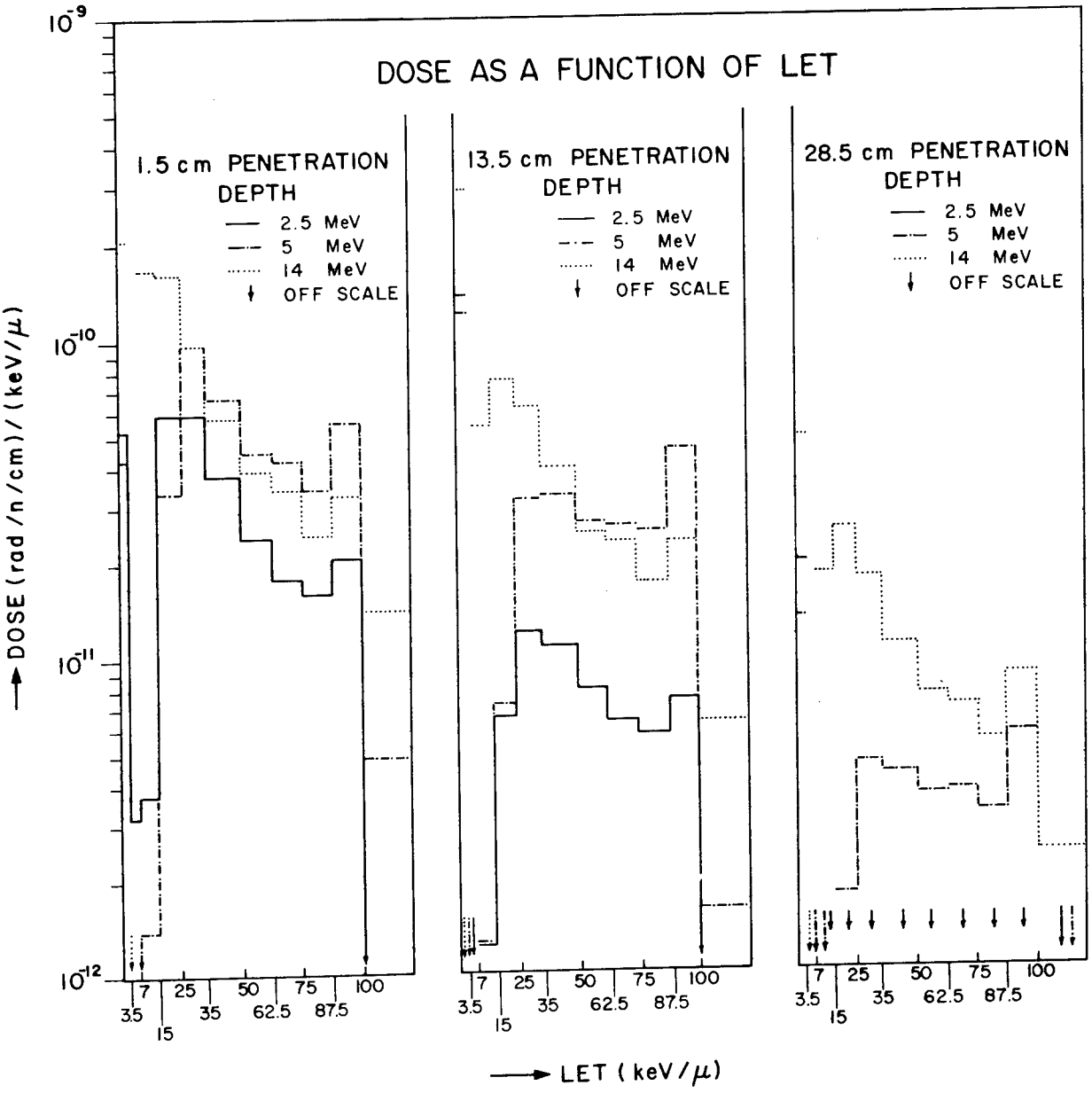


Fig. 3. Dose as a Function of LET for Penetration Depths of 1.5 cm, 13.5 cm, 28.5 cm, and Neutron Source Energies of 2.5 MeV, 5 MeV, and 14 MeV.

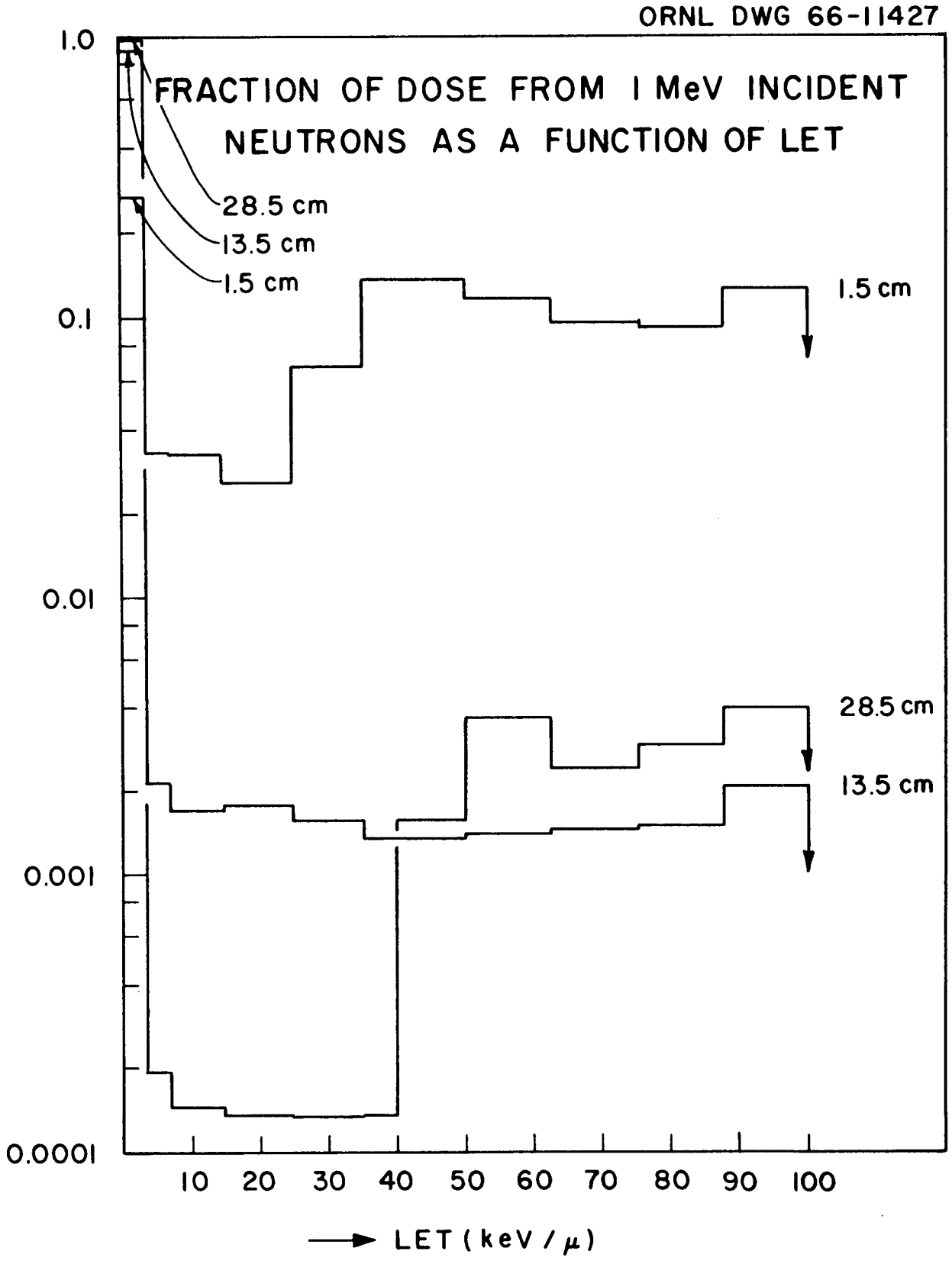


Fig. 4. Fraction of Dose from 1 MeV Incident Neutrons as a Function of LET.

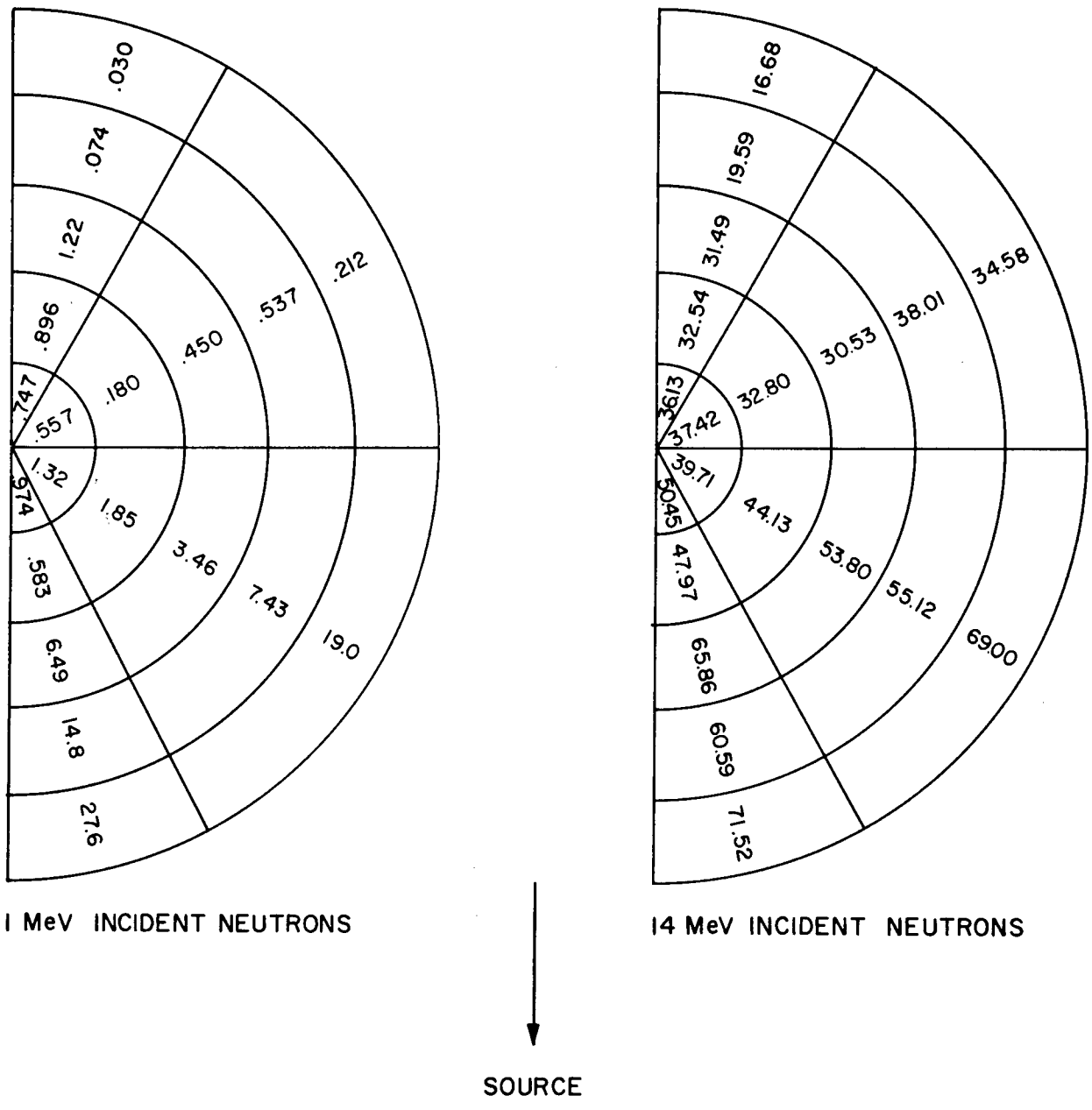


Fig. 5. Dose-Equivalent Distributions in the Middle Tier of the Cylindrical Phantom for Neutron Source Energies of 0.025 eV, 1 keV, 1 MeV, and 14 MeV.

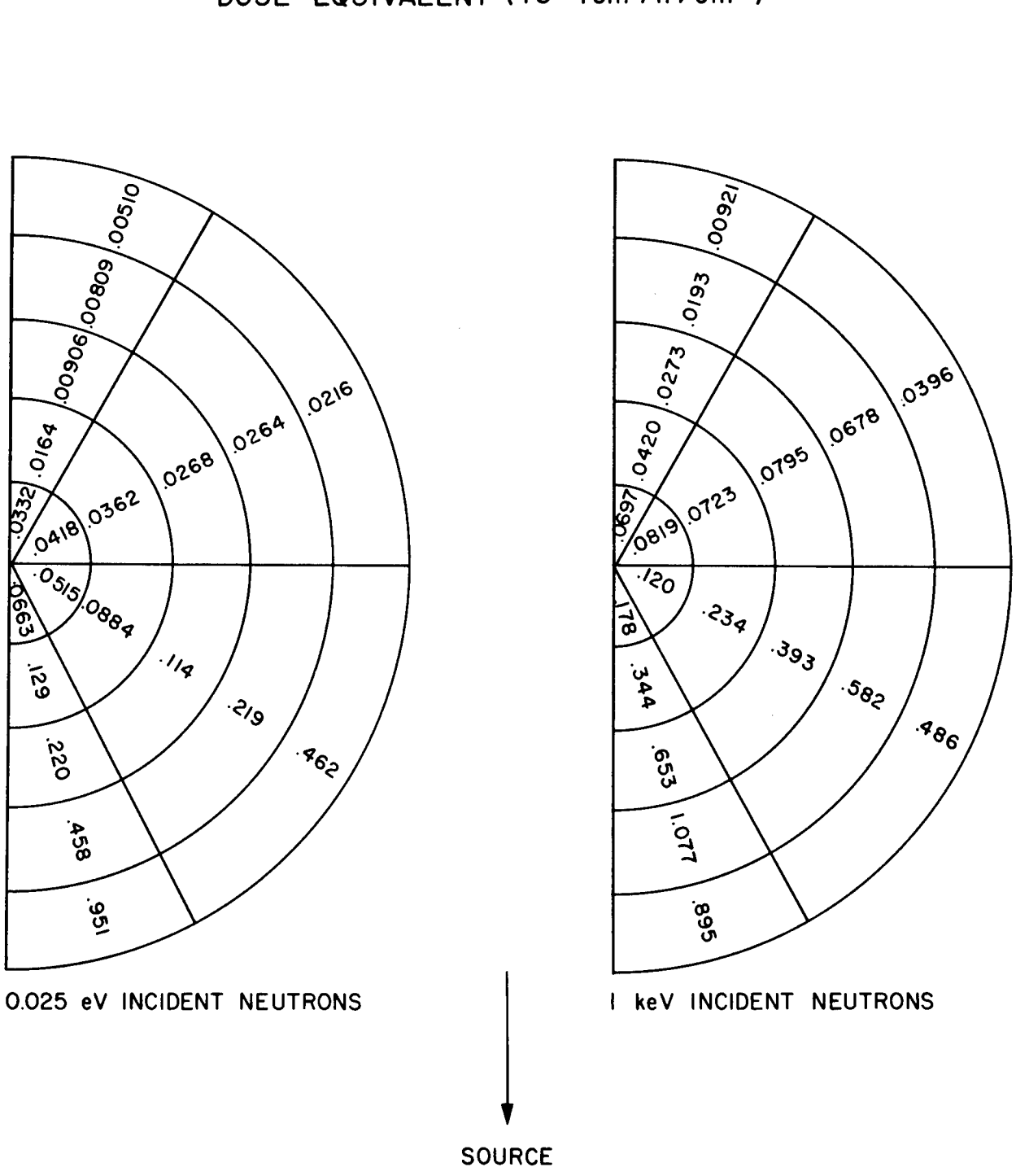


Fig. 6. Dose-Equivalent Distributions in the Middle Tier of the Cylindrical Phantom for Neutron Source Energies of 0.025 eV, 1 keV, 1 MeV, and 14 MeV.

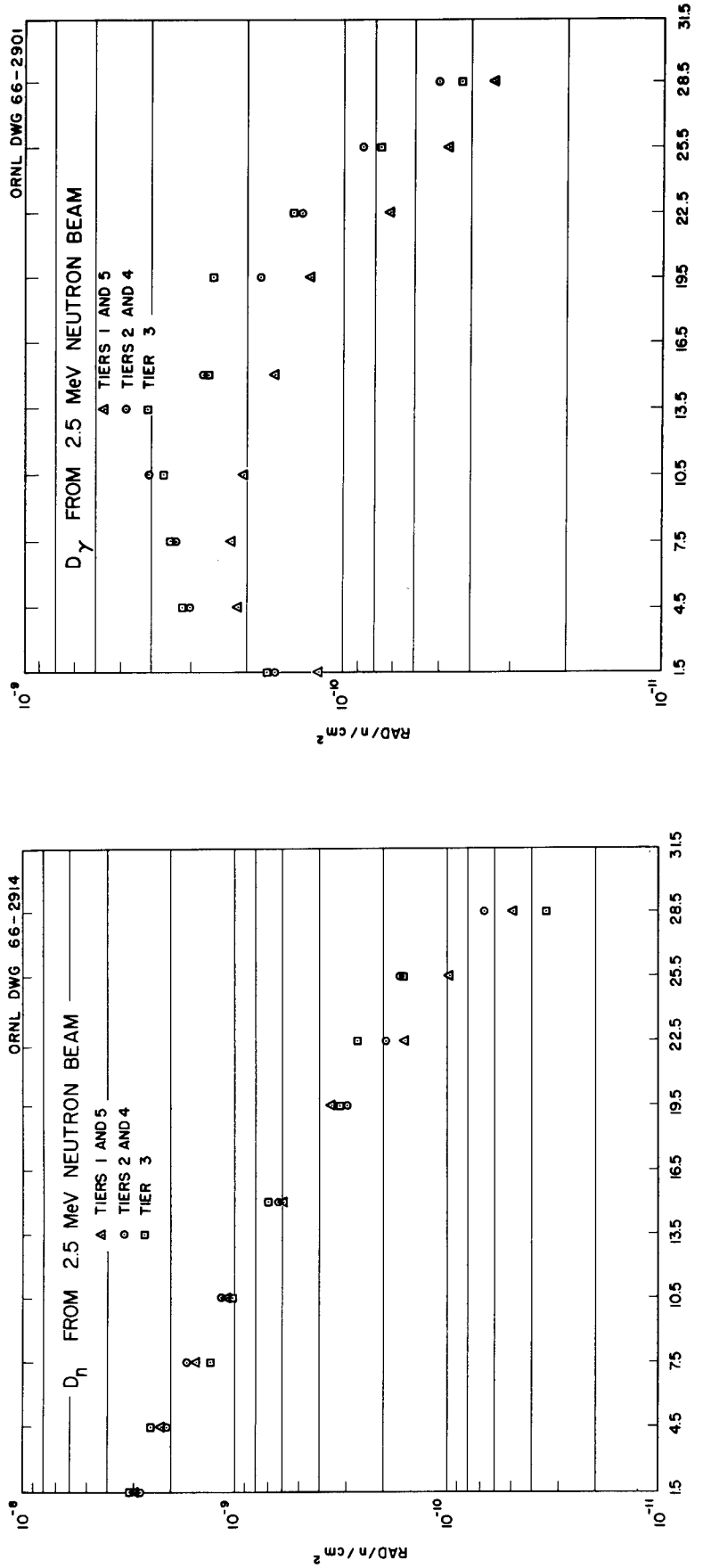
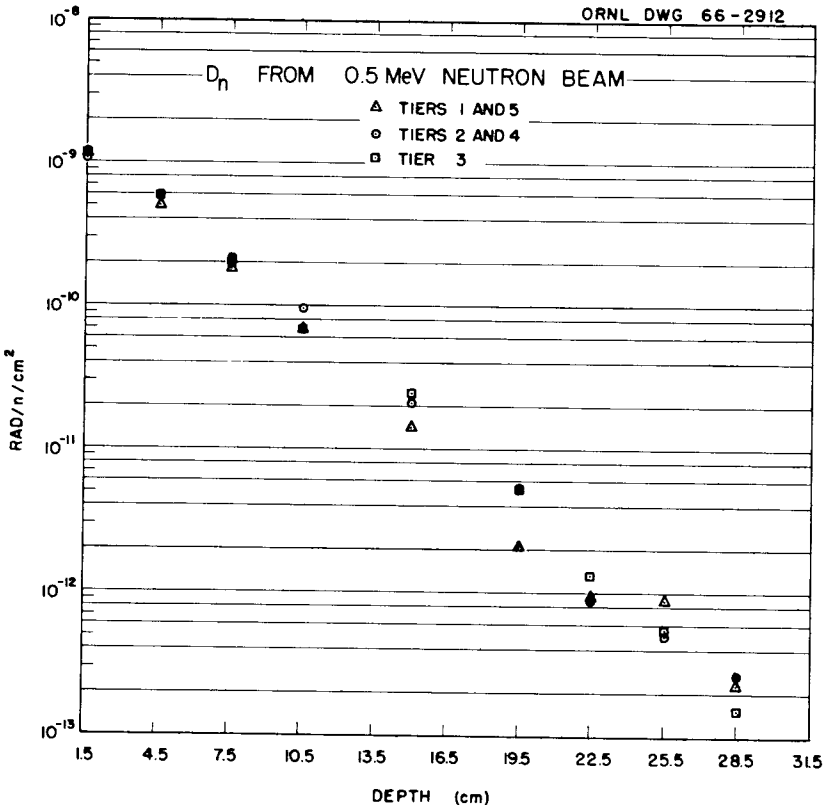


Fig. 7. D_n and D_γ as a Function of Penetration Depth for Source Energies of 0.1 keV, 0.5 MeV, and 2.5 MeV.



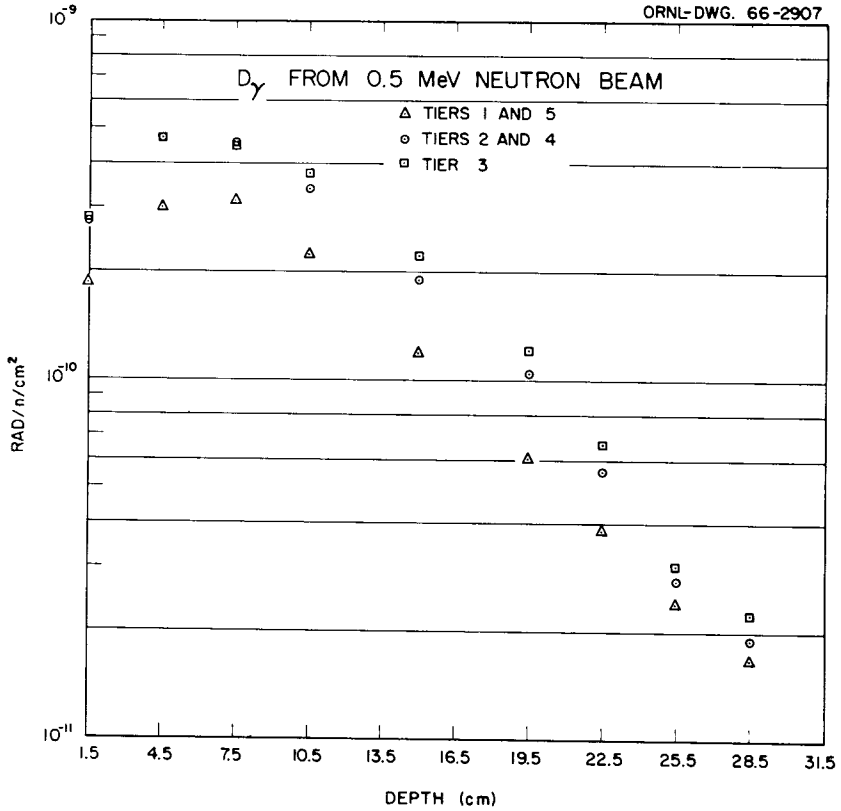
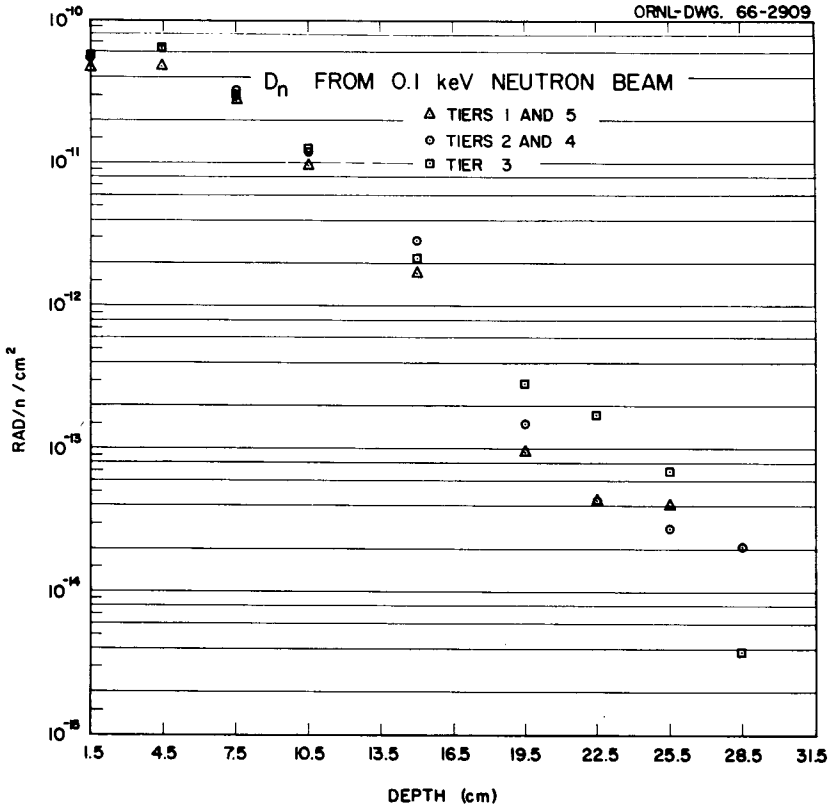


Fig. 8. D_n and D_γ as a Function of Penetration Depth for Source Energies of 0.1 keV, 0.5 MeV, and 2.5 MeV.



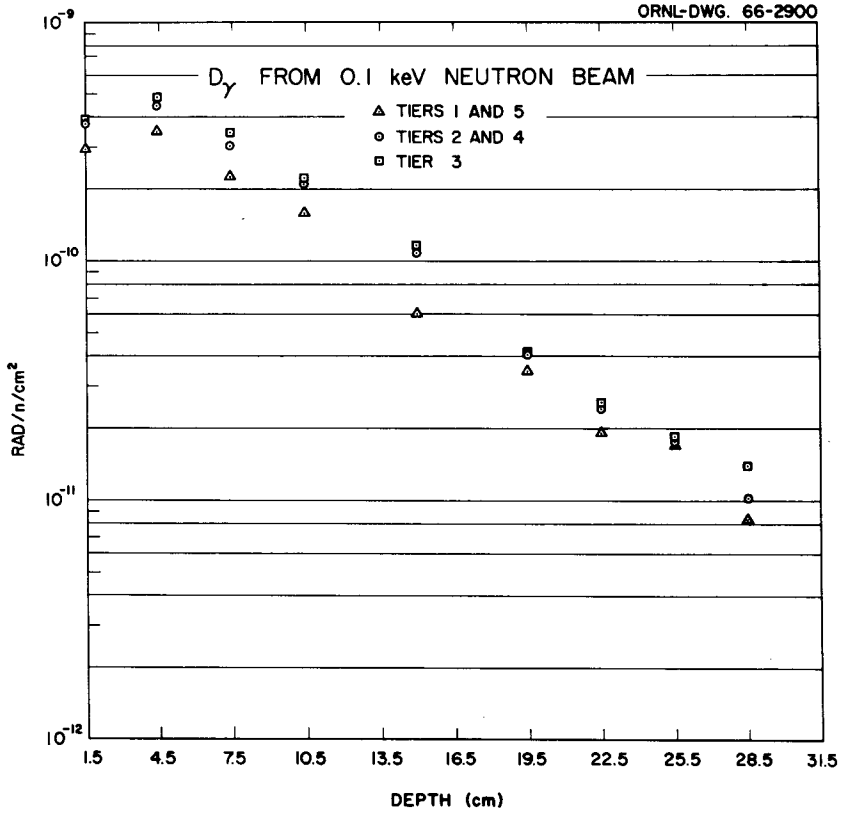


Fig. 9. D_n and D_γ as a Function of Penetration Depth for Source Energies of 0.1 keV, 0.5 MeV, and 2.5 MeV.

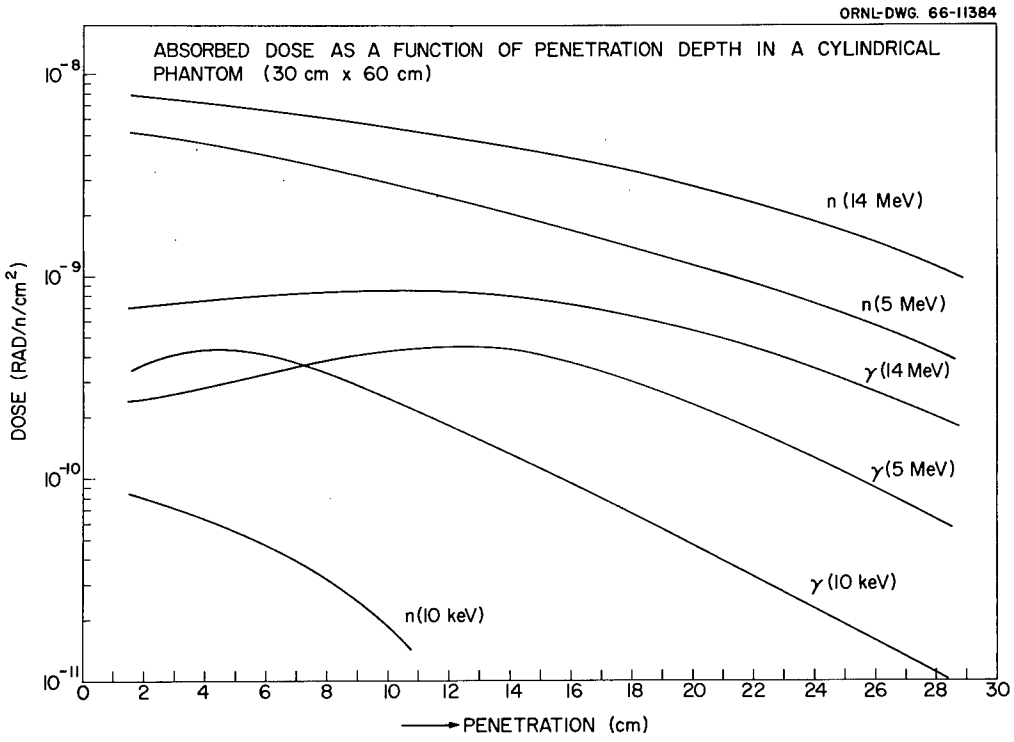


Fig. 10. Dose as a Function of Penetration Depth in a Cylindrical Phantom.

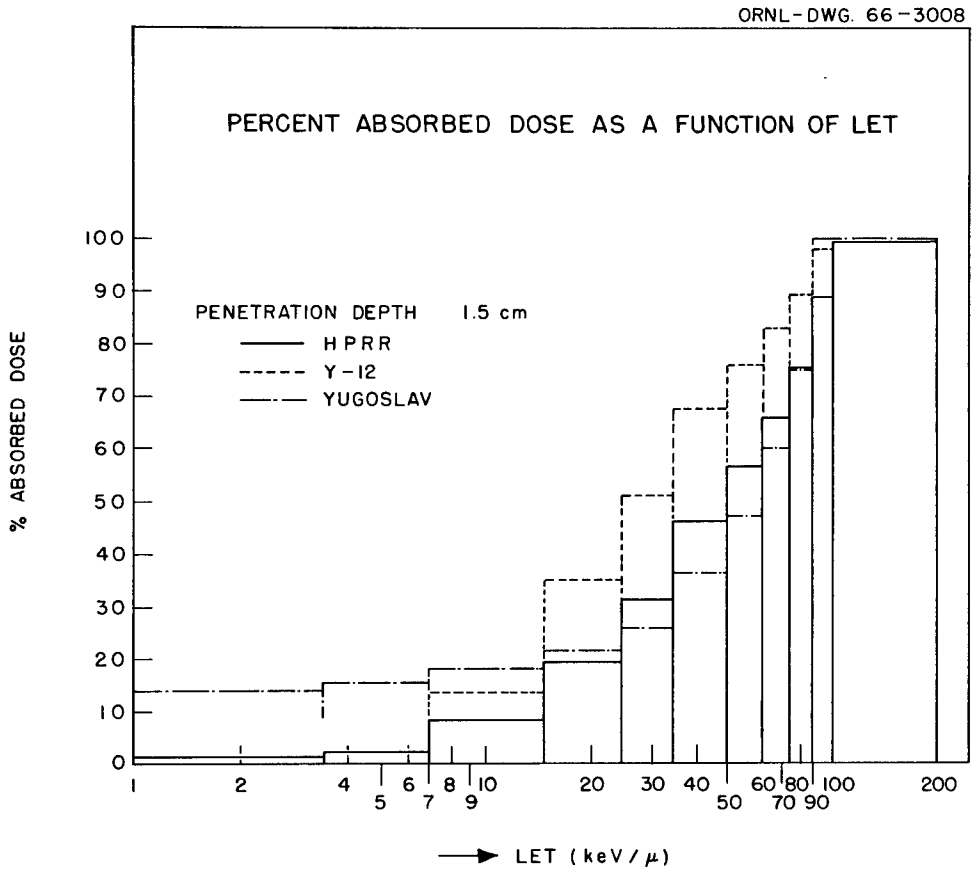


Fig. 11. Percent Dose as a Function of LET for Penetration Depths of 1.5 cm, 15 cm, and 28.5 cm.

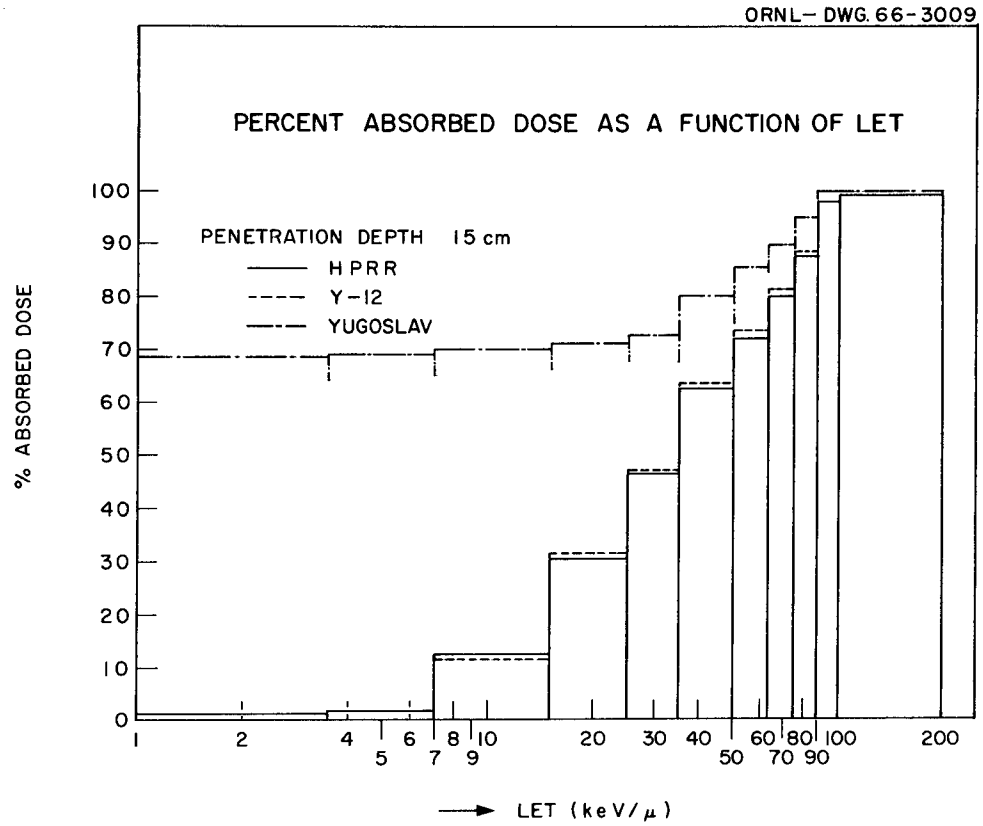


Fig. 12. Percent Dose as a Function of LET for Penetration Depths of 1.5 cm, 15 cm, and 28.5 cm.

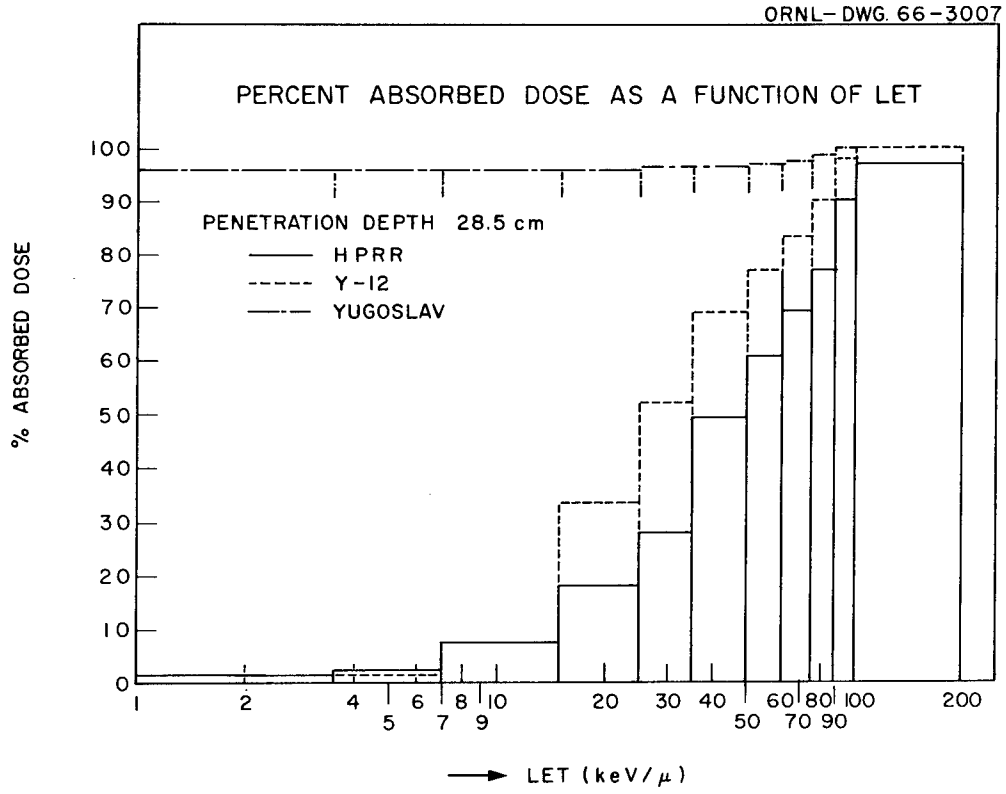


Fig. 13. Percent Dose as a Function of LET for Penetration Depths of 1.5 cm, 15 cm, and 28.5 cm.

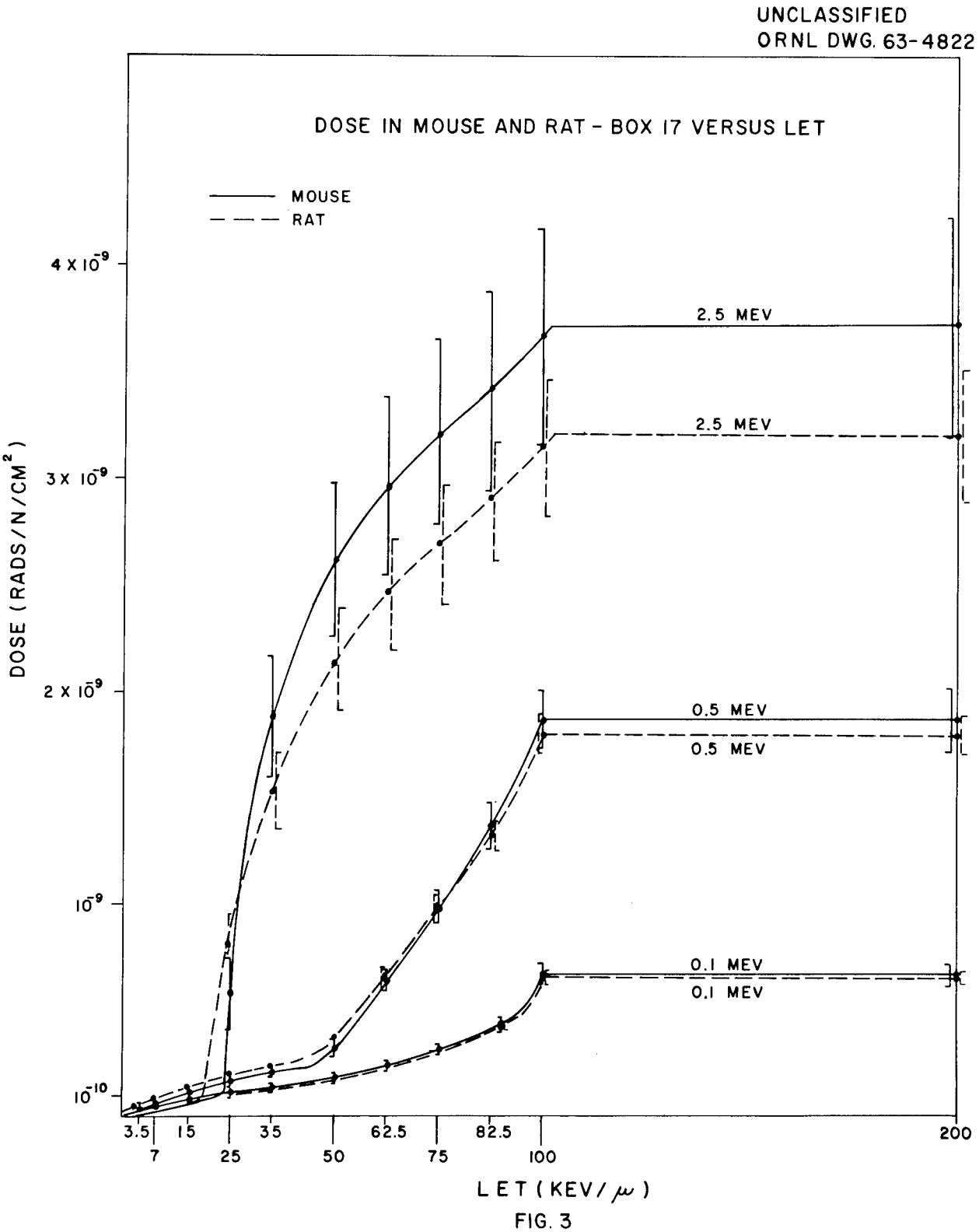


FIG. 3

Dig. 14. Dose as a Function of LET in a Mouse- and Rat-Sized Phantom for Source Energies of 0.1 MeV, 0.5 MeV, and 2.5 MeV--Volume Element 17.

TISSUE CURRENT-TO-DOSE CONVERSION FACTORS FOR NEUTRONS WITH ENERGIES FROM 0.5 TO 60 MeV*

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and H. S. Moran

To assist in the evaluation of the hazard associated with exposure to high-energy neutrons, a Monte Carlo computer program was used to calculate the energy deposition as a function of depth in a 30-cm-thick infinite slab of tissue resulting from neutrons incident on the slab at energies up to 60 MeV. The program treated nonelastic and elastic interactions, including evaporation processes and nuclear recoils. Cases of both normal and isotropic incidence were calculated for neutrons of 0.5, 2, 10, 18, 30, and 60 MeV. From these data, current-to-dose conversion factors were extracted for the average whole-body dose, the dose at a 5-cm depth, and the maximum dose. A set of quality factors (QF's) was adopted for transforming absorbed dose to dose equivalent.

Results for the absorbed dose and the dose equivalent at a depth of 5-cm are given in Figs. 1 and 2.

*This is the abstract of a paper to be published in Nuclear Instruments and Methods.

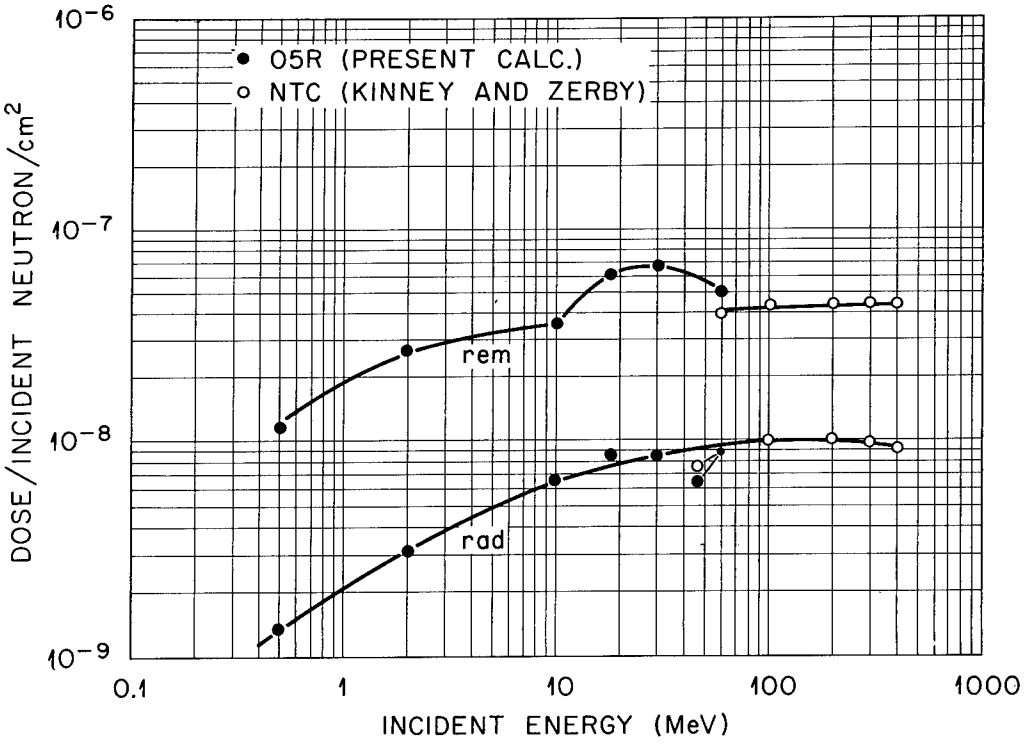

Fig. 1. Dose at 5-cm-Depth Due to Isotropically Incident Neutrons.

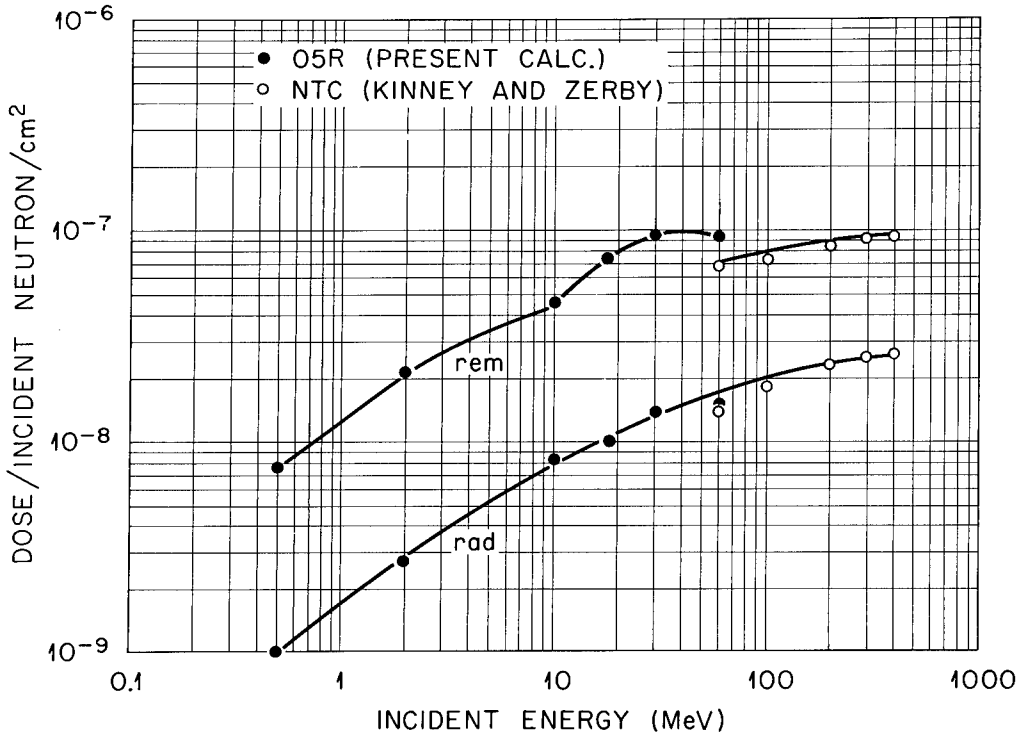

Fig. 2. Dose at 5-cm-Depth Due to Normally Incident Neutrons.

ON DEPTH-DOSE CALCULATIONS IN AN EVALUATION OF FALLOUT SIMULATORS AND FALLOUT FIELDS

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ON DEPTH-DOSE CALCULATIONS IN AN EVALUATION OF FALLOUT SIMULATORS AND FALLOUT FIELDS

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R. L. French

To properly study the radiobiology of fallout, one requires a knowledge of the properties of the radiation fields present. Also, since actual fields are presently non-existent, the biologist studying the effects of exposure to fallout requires a laboratory-produced radiation field for experimental use. It thus is of importance to study the characteristics of these radiation fields, both to assist the radiobiologist in estimating the biological effects resulting from a fallout exposure and to evaluate the degree of simulation achieved in existing or proposed laboratory simulators.

To characterize such fields, it has been the custom to calculate or measure differential and total doses* and flux densities in the "free fields" (unperturbed by the absorbing biological specimen) from which, for example, "protection factors"** are obtained. Such free-field data enable one to draw qualitative comparisons between simulators and the fields they attempt to reproduce and between shielded and unshielded situations. However, of greater interest to the radiobiologist are the spatial and spectral distributions of the dose deposited in biological material placed in these fields of interest.

In a series of calculations, we have studied several real and simulated fallout situations. Initial studies[1,2] characterized the free fields at specified detector points in typical geometries by describing the photon angle and energy distributions at each. Some of the geometries considered are shown in Figure 1. In the first, a detector point was placed three feet over an infinite, isotropic and uniformly distributed fallout source. A detector was also located at the axial midpoint of a 4 ft. dia. by 5 ft. deep foxhole placed in the fallout field. In the second a 60-Co isotropic point source elevated three feet above the ground was

---

*Actually, kerma or exposure are the quantities most often determined. In fallout fields, the differences in the numerical values of kerma and absorbed dose are slight; to be consistent with the terminology of the referenced work, we use the term "dose" throughout.

**The "protection factor" of a shield is usually defined as the ratio of the unshielded free-field dose to the shielded free-field dose. Often its reciprocal, the "transmission factor" is used.

positioned 200 ft. from a detector also placed three feet above the ground. The third was a sophisticated conceptual device called a "compact simulator". It consisted of a three-isotope (60-Co,137-Cs,144-Ce) disc source, a similar ring source placed three feet above the disc, and an overhead scattering slab of water. The detector point was at the midpoint of the 120 ft. dia. device.

To learn more about the properties of these fields, calculations were recently performed[3,4] using the free-field results as source data to compute the axial and radial depth-dose distributions in homogeneous, tissue-equivalent cylindrical phantoms whose midpoints were placed at the detector points shown in Figure 1. Doses were calculated at points placed along the vertical axis and along a midplane diameter of the 30cm. dia. by 60cm high phantom.

The COHORT Monte Carlo procedure was used to perform the phantom computations. Details of the calculational procedures will not be described here, but are available in References 3 and 4. The results are given in terms of "dose fractions" which are plotted as functions of position along the phantom's midplane diameter (radial distributions) and vertical axis (axial distributions). The "dose fraction" is the ratio of the dose at the detector points in the phantom to that at the location (phantom midpoint) of the free-field detector with no phantom present. Figure 2 shows the results for the "above-ground" geometries. For the fallout field, fallout ages of 1.12 hrs., 23.8 hrs., 4.52 days and 9.82 days were examined; the first two with precision, the second two with more approximate methods. The results were found to be identical for all ages within the accuracy of the calculations.

For the fallout field models, it is seen that the radial distributions are relatively flat, with the midline dose 80 percent of that at the lateral surface, which in turn was 81 percent of the calculated free-field dose. The axial distributions were found to fall off quite linearly with increasing height, closely following the free-field falloff. The distributions in the 60-Co point source simulator, while not differing greatly from the real field patterns, showed greater differences toward the end and lateral surfaces than those of the compact simulator which are seen to be very similar to the real field results.

Comparison of the three geometries illustrates how depth dose calculations aid in evaluating the effectiveness of a simulator. Although there are differences in the dose spectrum (fallout has significant photon energies ranging up to 4 MeV whereas both simulators have a maximum energy of 1.33 MeV), it is seen that both devices quite well simulate the depth-dose patterns of the fallout field. Assuming other important parameters such as dose and dose rate are properly controlled, they could be expected to produce similar biological effects.

In Figure 3 are shown the distributions for the phantom placed in a foxhole. Also shown for comparison are the above-ground fallout field dose patterns. It is seen that the radial distribution is quite similar in shape to the above -ground cases, however the axial distribution is completely different from that

found above the ground. (It should be recalled that the ordinate scale on Figure 3 is the ratio of the dose in the phantom to that in a free field at the center of the foxhole. Since the upper part of the phantom was in a significantly higher incident flux region, ratios above 1 were obtained.)

The foxhole case illustrates the problem in choosing protection (or transmission) factors. A value presently in use for foxholes[5] and based on free-field dose ratios is 10. However, as Figure 3 shows, the very non-uniform axial distribution in the foxhole is far different from that above ground. One cannot expect that the protection afforded by the foxhole may be measured solely in terms of a single free-field dose ratio. Also to be considered is the nature of the depth-dose patterns, for they affect the biological damage.

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2. French, R. L., "A Comparative Study of Radioactive Source Arrangements for Simulating Fallout Gamma Radiation Fields," Armed Forces Radiobiology Research Institute Report CR65-2 (1965); originally published as Radiation Research Associates Report RRA-T45 (1964).

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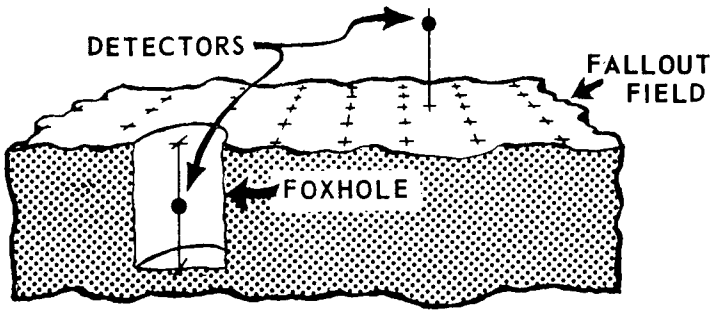
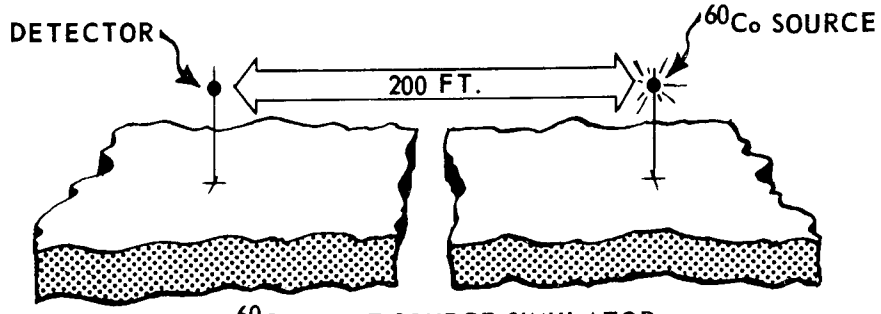
4. French, R. L., Tompkins, K. W. and Garrett, C. W., "Gamma-Ray Dose Distributions in a Phantom Exposed to Fallout and Simulated Fallout", Armed Forces Radiobiology Research Institute Report CR67-1, prepared as RRA-T71 by Radiation Research Associates, Fort Worth, Texas (1 Feb. 1967).

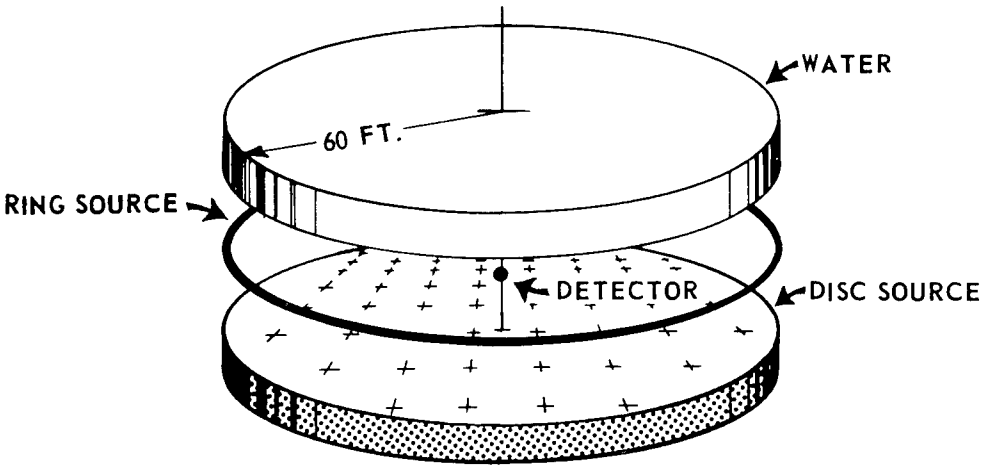
5. "Operational Aspects of Radiological Defense" (UNCL), Department of the Army Field Manual FM3-12, Pg. 56, (Jan.1963).



A. FALLOUT FIELD

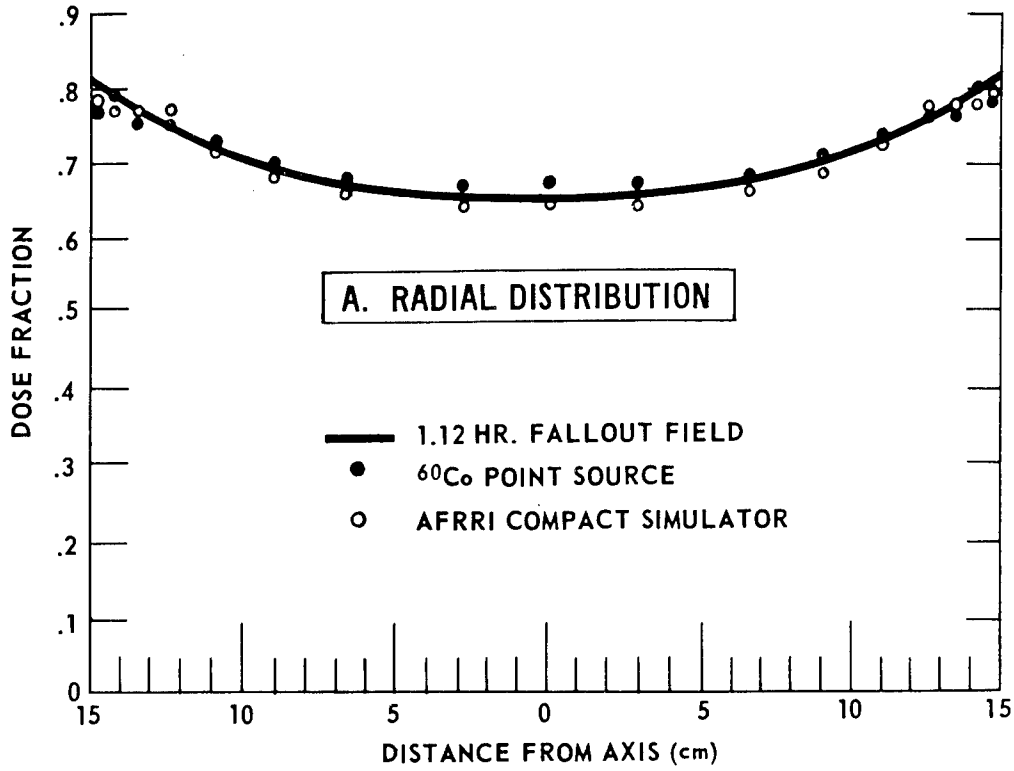


B. ^{60}Co POINT SOURCE SIMULATOR



C. AFRRI COMPACT SIMULATOR

FIG. 1 FALLOUT AND SIMULATOR GEOMETRIES



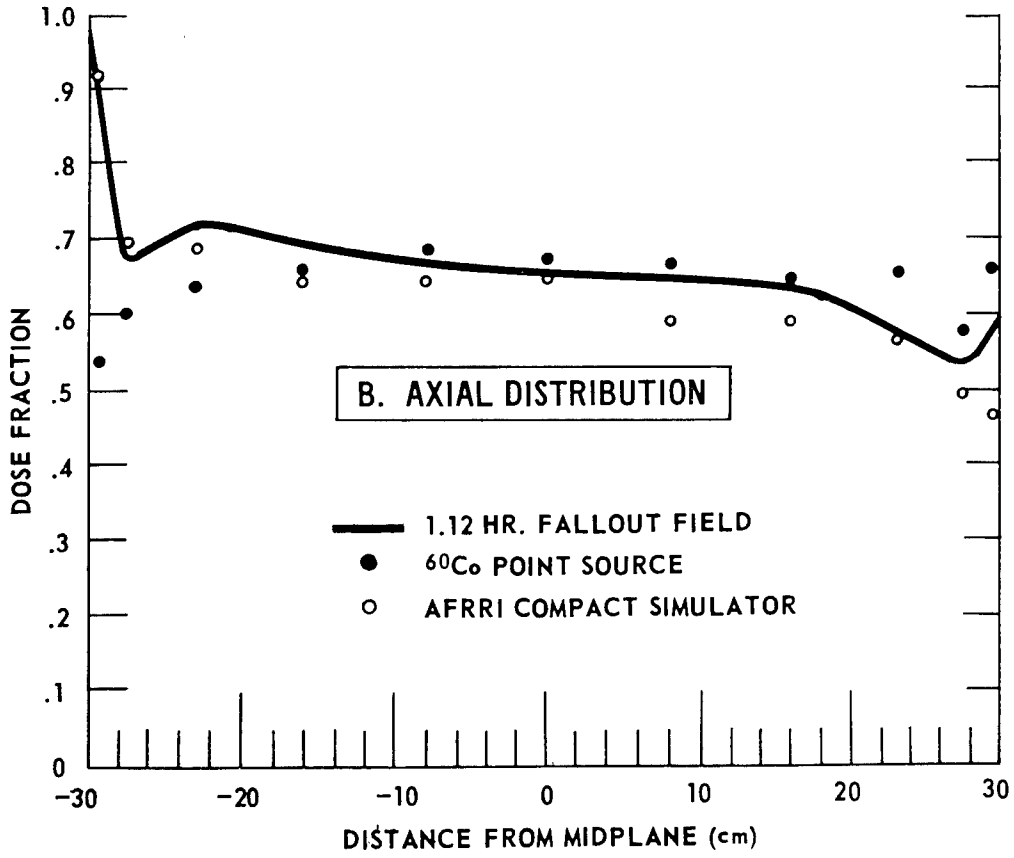
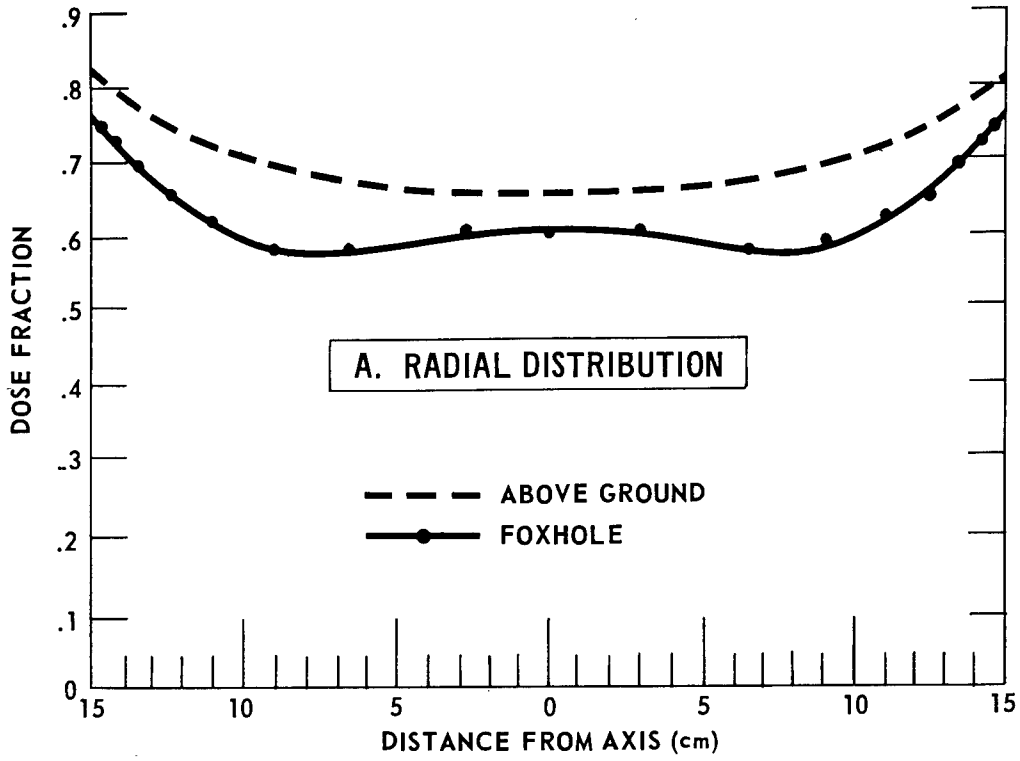


FIG. 2 ABOVE GROUND DEPTH DOSE DISTRIBUTIONS



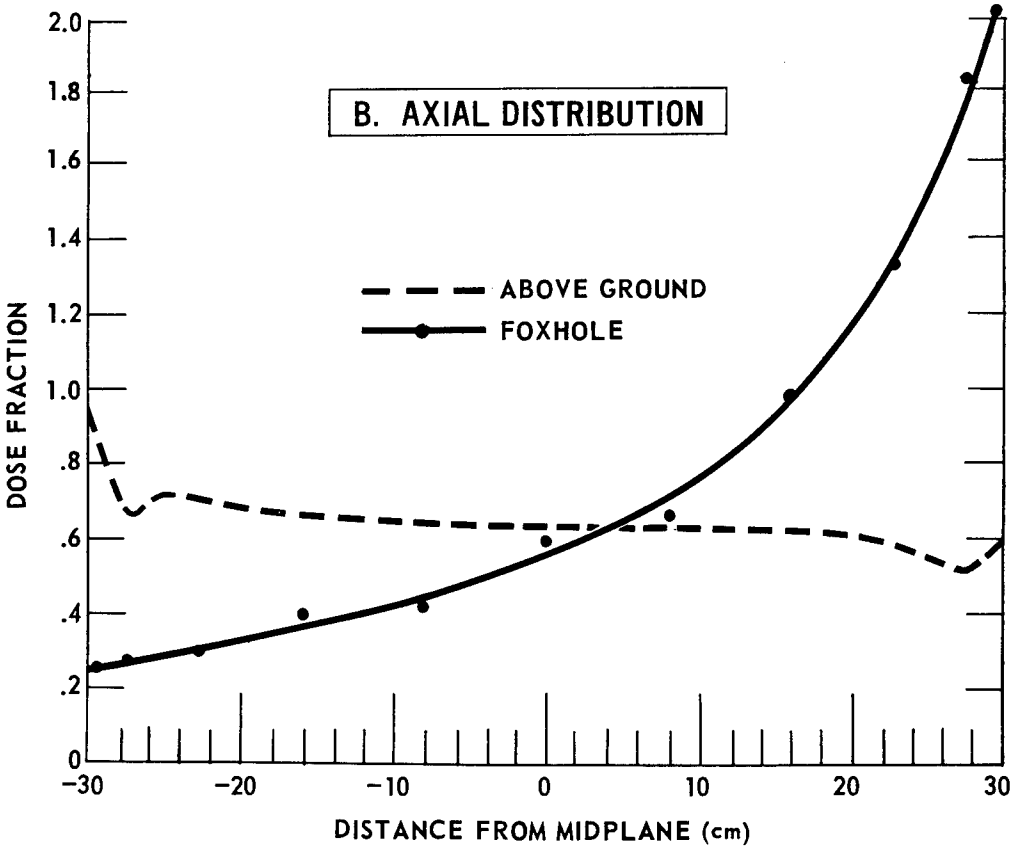


FIG. 3 FOXHOLE DEPTH DOSE DISTRIBUTIONS

A NEW TECHNIQUE FOR THE COMPUTER REPRESENTATION OF THE HUMAN BODY

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A NEW TECHNIQUE FOR THE COMPUTER REPRESENTATION OF THE HUMAN BODY

Phillip S. Mittelman & Walter Guber

A detailed three dimensional computer model of the human body with its internal organs, skeleton and structure associated with a radiation transport code would be of considerable value to those concerned with medical radiation studies.

It would permit the evaluation of the dose at specific organs of the body when exposed to radiation sources such as fallout fields, weapons and therapeutic devices. The effect of local body shielding could be accurately determined.

For therapeutic situations an optimum irradiation plan could be developed so as to maximize the dose at the desired target while minimizing the exposure of sensitive regions.

For isotope sources placed internal to the body, one could predict either the radiation levels at other internal locations or the radiation field at points exterior to the body.

Since the same techniques for geometrical description could be used to set up animal body geometries, intercomparison could be made of radiation doses at selected organs assuming both are exposed to a given source. The large body of data on animal irradiation could more accurately be related to the study of human radiation exposure.

The technique for such a geometrical description has been developed* and is being programmed under contract with the Aberdeen Proving Grounds. It will be incorporated into the UNC-SAM 2 Monte Carlo program.

The basic technique for describing a physical region involves the addition and subtraction of figures bounded by planes or quadric surfaces. A simple example will demonstrate the power of the method.

Consider a sphere and a cylinder labeled 1 & 2 respectively as in Figure 1.

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*In an abstract titled "A General Geometry Shielding Code for Space Protons" by R. Madey, D. Levine, F. Schwamb and F. Sisavie; Trans. AMS 7 1 64 (Pg.19) the use of a geometrical description technique similar to that presented here is mentioned.

A variety of figures can be produced from these two.

```
Region A = 1 + 2        (points common to both)
Region B = 1 - 2        (all points in 1 but not in 2)
Region C = or 1 or 2    (points in either 1 or 2)
```

Ray tracing is simple and rapid with this approach. Figure 2 shows a box containing a sphere intersected by an ellipsoid. One first defines the three surfaces, 1,2,3,. One can define Volumes A and B and C as follows:

```
Volume A = 2 - 1
       B = 1
       C = 3 - 1 - 2
```

The program establishes a set of entering and leaving tables which define the regions one may enter if a given surface is entered or left.

The tables are formed by scanning the region descriptions and, for any region described by a surface with a plus sign that region is placed in the entering table for that surface. Any region described by a surface preceded by a - sign is placed in the leaving table for that surface. If an (or) is present the region goes into both the entering and leaving tables.

Consider now a ray starting in C and heading as shown in Figure 2. Region C is defined by three surfaces 3,1 & 2. Tests are made to find which surfaces will be hit and the distance to each hit surface. In this instance all three will be hit but 2 is hit first. The distance along the ray from 0 to surface 2 is assigned to region C and a test is made to find the region being entered. Since the ray is entering surface 2, its entering table is examined. Only region A can be entered through 2. Thus, the ray is now in A. From the description of A it is seen that surfaces 1 & 2 are used to describe A. These surfaces must then be checked to see which is hit first. In this case 1 is hit first. Upon entering surface 1 it can be seen that only region B is being entered. Since B is described in terms of surface 1 only, then the distance to surface 1 is computed. On leaving surface 1 the table shows that regions A or C might be entered. Tests are performed on the surfaces involved in the descriptions of regions A & C. In this case only the definition of C is satisfied. Therefore the ray has emerged into C. Tests are performed on surfaces 1,2,3 which describe region C. The only intersection found is with surface 3. The ray is then transmitted into the next box.

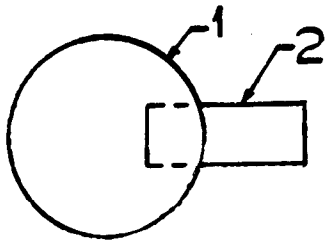
As an example of how this technique is used to describe the human body, we have constructed two partial representations. These are not intended to be complete or accurate but merely to illustrate the application of the geometrical description technique for the human body. Figure 3 illustrated the outer surface of a simple body model built up from an elliptical cylinder, ellipsoids, and a wedge.

Figure 4 shows the basic figures used to produce the body shown in Figure 3.

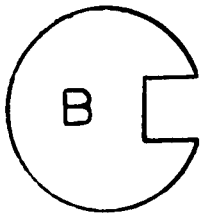
Each of the body organs can be described and located in its proper position in the body. We have prepared a description of the heart. Figure (5) shows a cross section of this model as displayed on the computer printer. This picture was prepared by sending a number of parallel rays through the model - all rays lying in the same plane. The printer produces a point when the ray passes from one region of the heart to another. Such figures can be prepared for any plane through the heart model.

It should be noted that the individual component described as Heart can be located at any desired point inside the body model. Scale changes are also easily made.

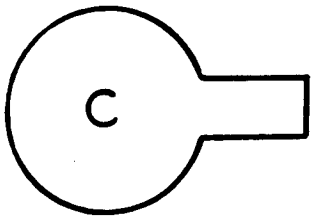
The use of this body description technique in association with the UNC-SAM II Monte Carlo program will permit dosage calculations to be made with a very high degree of accuracy.



A = +1 +2

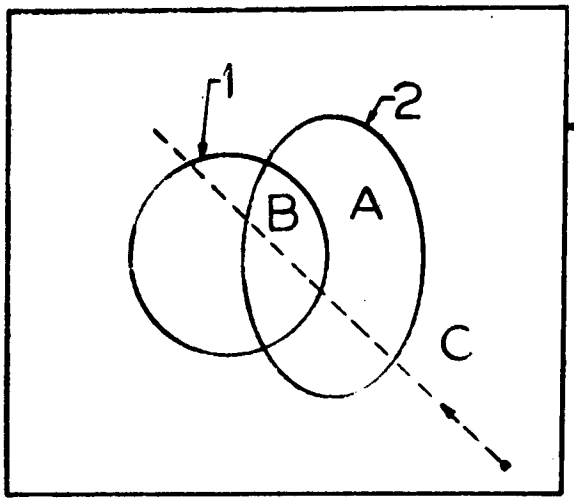


B = +1 -2



C = OR 1 OR 2

Fig. 1. Region Produced by Combinations of a Sphere and a Cylinder.



REGION DESCRIPTION

A = +2 -1

B = +1

C = +3 -1 -2

SURFACE	ENTERING	LEAVING
1	B	A OR C
2	A	C
3	C	SPECIAL

Fig. 2. Ray Tracing.

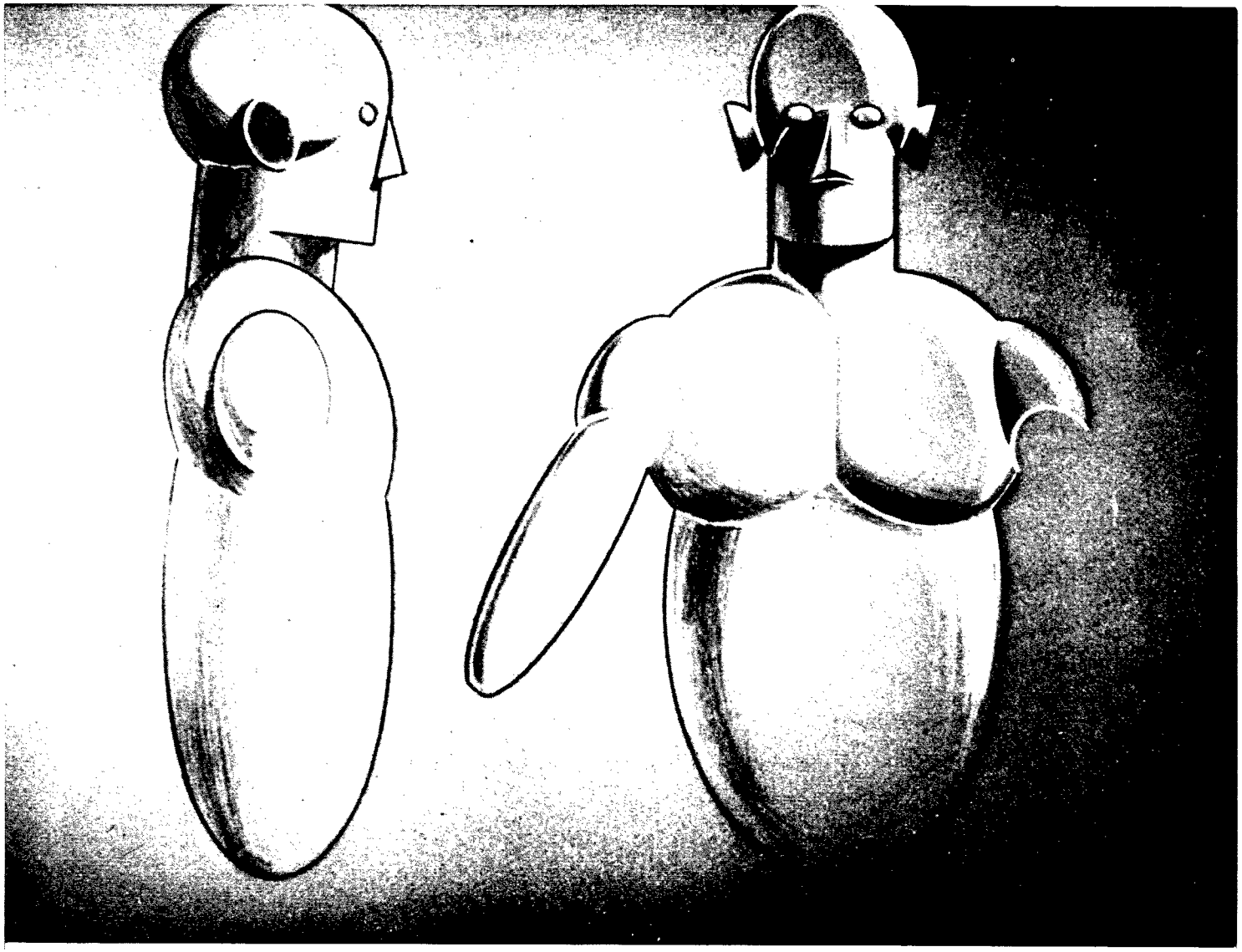


Fig. 3. Outer Space of Simple Man Model.

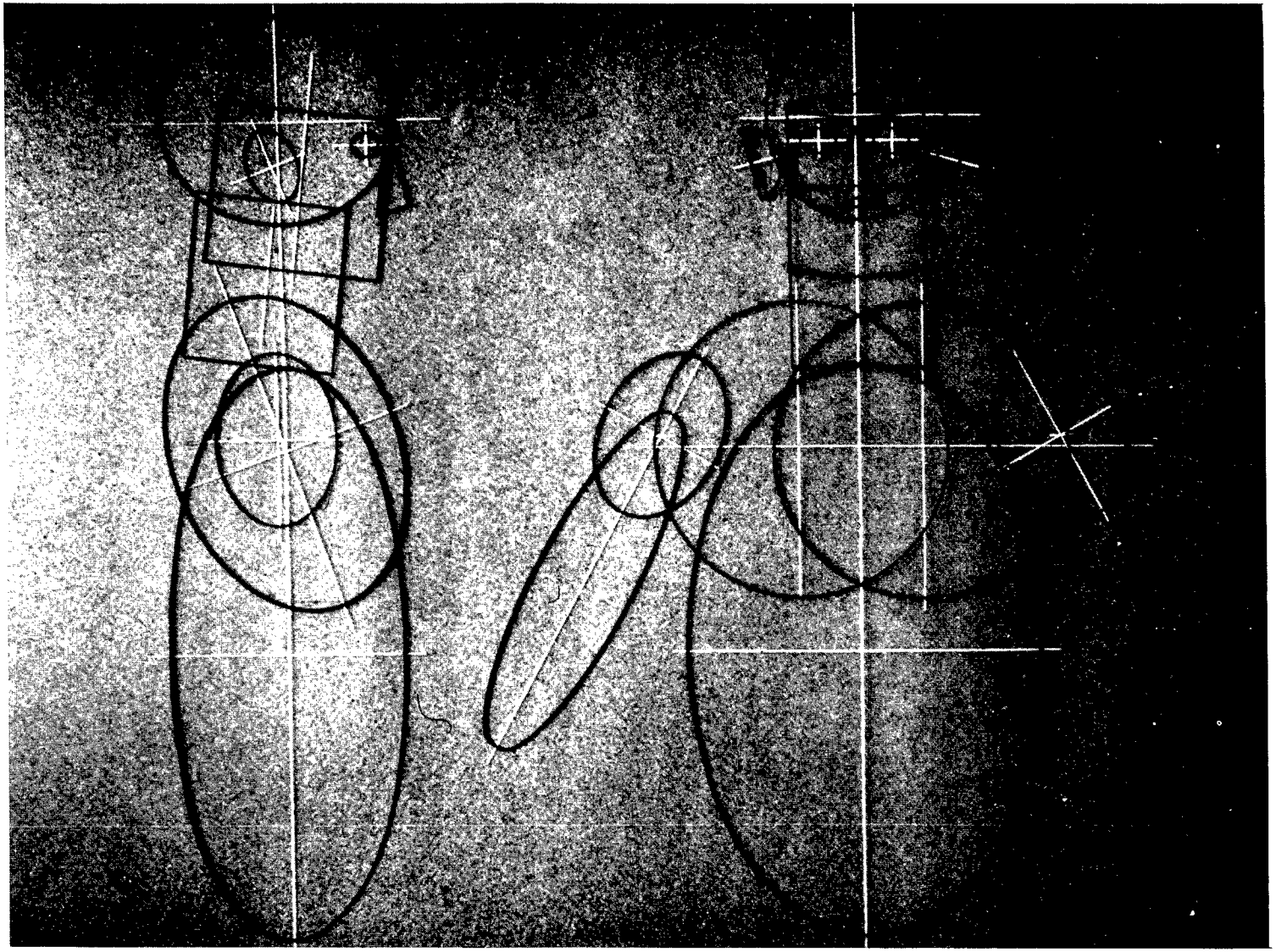


Fig. 4. Basic Surfaces Used to Construct Man Model.

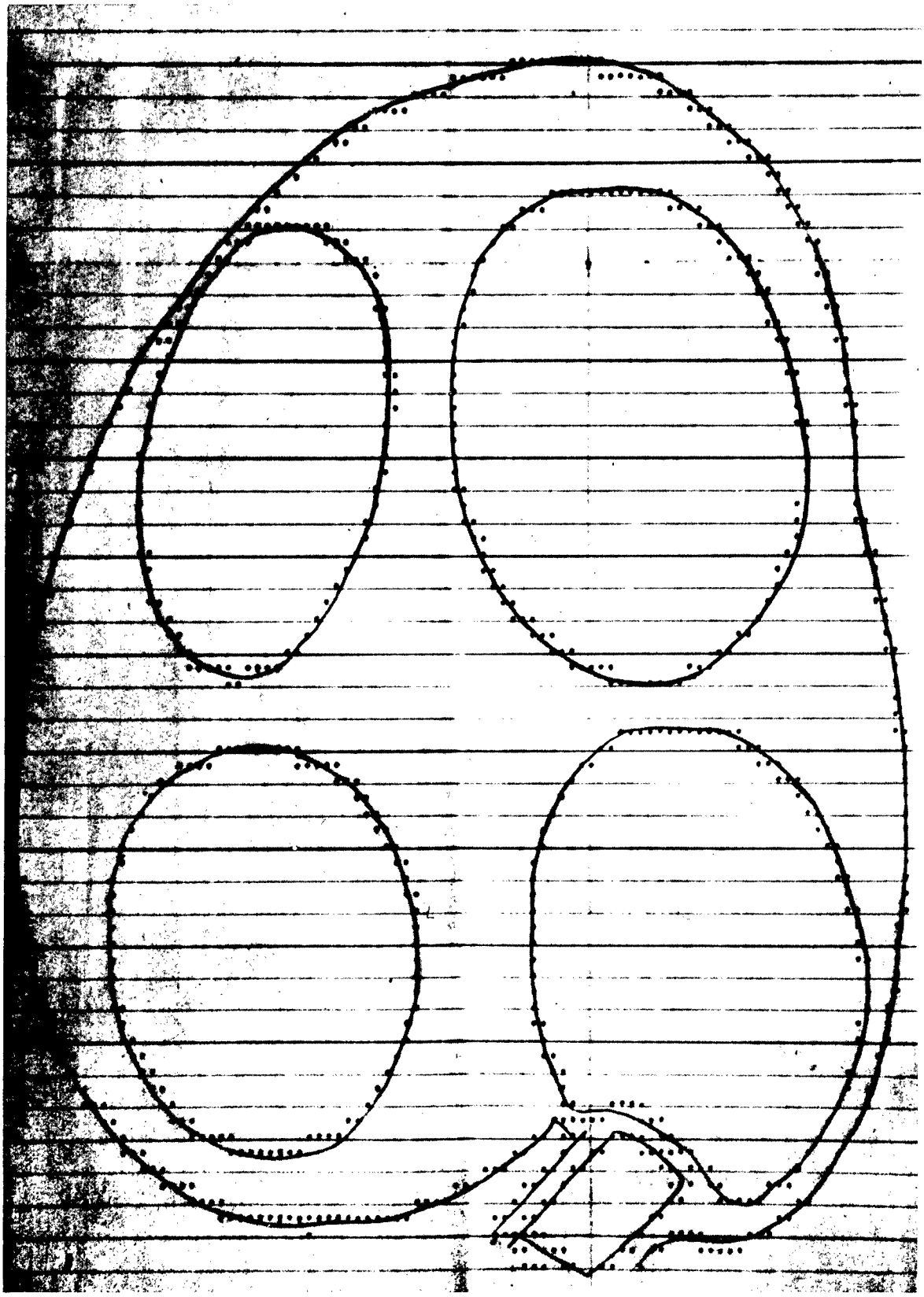


Fig. 5. Cross Section of Heart Model Produced by Computer.

PHYSICAL AND BIOLOGICAL DOSE CALCULATIONS FOR PROTON AND ALPHA FLUXES INCIDENT ON A SHIELDED MAN MODEL

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INTRODUCTION

The assessment of hazards associated with space radiations has received considerable attention during the past seven or eight years. The interface between radiation transport and biological effects was not seriously examined during the first part of this period. Yet, the diversity of radiation types and energies, combined with the frequently steep dose-depth relationship, often makes this interface more critical for space radiation shielding than for reactor and weapons shielding. One reason for this neglect is the major uncertainties in other parts of the calculation. For example, the continued mapping of the trapped radiation belts in a parametric fashion permits the prediction of orbital radiation exposures to within a factor of two to ten, generally, depending upon the region of the magnetosphere involved, and upon the time period. Current estimates based on the life history of solar flux events have reduced the uncertainty in long range prediction to perhaps an order of magnitude or two. With the probabilistic models coming into vogue, the uncertainty may be ascribed to either the radiation environment or to the risk level assigned.

The transport of protons through simple shield configurations is in a more satisfactory state. The production and attenuation of secondary radiation arising from inelastic nuclear collisions is reasonably well known. Physical dose may usually be computed to within 30% for simple shield configurations up to 20 grams per square centimeter in thickness.

This paper will not presume to estimate the uncertainty in correlating physical dose with biological effects.

Other uncertainties may arise in the "interface" areas between source definition and radiation transport, or between radiation transport and biological effect. An example of the first interface is the interaction between source anisotropy and transport calculations. The second interface - between radiation transport and biological effect - is one subject of the present session. This paper will examine one aspect of the interface: that is, the influence of performing transport calculations in detailed phantom

geometries on dose to specific organs.

COMPUTATIONAL MODEL

A computational model is selected for illustration purposes. Details of the model are given below.

RADIATION ENVIRONMENT

The radiation exposure arising from solar flux events during a 1986-1987 Mars expedition is estimated with the aid of FLARE, a Monte Carlo computer code. The FLARE code uses data derived from observations of solar flux events during solar activity cycle 19. In essence, the mission is "flown" a large number of times on the computer. The probability of an event is sampled for each day of the mission, and proton and alpha flux spectra are accumulated over each mission history. After a specified number of histories are processed, a table of flux level versus frequency of occurrence is formed. Figure 1 shows the predicted radiation environment for the mission under consideration. The solid lines represent proton spectra at the 0.1, 1.0, and 10.0 percent probability levels, while the dashed lines represent alpha spectra at the same probability levels. Note that a factor of ten uncertainty in the 1.0 percent curve could shift the spectra up to near the 0.1 percent curves, or down to near the 10.0 percent curves.

RADIATION TRANSPORT

The transport of heavy charged particles, ignoring nuclear collisions, is easy to calculate providing range, energy, and direction straggling effects may be neglected. The latter effects may be important for mono-energetic beams, but are only one or two percent effects for the continuous spectra considered here. Some difficulties arise when the production and attenuation of secondary radiations are included in the calculation.

The treatment of proton-induced secondary radiations is fairly well advanced. A large body of experimental and Monte Carlo data is available, and the validity of straight-ahead or isotropic assumptions has been checked in some instances. A number of codes are available to treat proton transport problems in simple geometries.

The treatment of alpha-induced secondaries is in a more primitive state. Relatively little experimental data is available, and the Monte Carlo method has not yet been applied to the intranuclear cascade problem because of lack of understanding of collective nuclear forces. No alpha transport code which treats secondaries in detail is available. The DOSE CODE, used in this study, is the only published code which attemots to include an estimate of alpha-induced secondaries. The estimate is based upon ratios of alpha and proton radiochemical data which is an indirect measure of secondary yields.

The detailed treatment of charged particle transport becomes expensive for complex geometries. Here, a point kernel method is usually employed; that is, an array of vectors emanating from a

receiver point samples the shield configuration. A transport calculation is performed for each vector and the results, weighted by solid angle, are summed.

DOSE CALCULATION

Physical dose is calculated in a standard way using the charged particle stopping power. A dose buildup factor derived from a detailed transport code provides for dose due to secondaries.

Biological dose, nominally in rem, is obtained with the quality factors recommended by the ICRP. It should be noted that these quality factors are intended to be upper limits on RBE for long term biological effects. These values are probably too large for computing acute effects due to short term exposures.

PHANTOM GEOMETRY

Three types of phantom geometry are examined in the present study. Figure 2 illustrates these phantoms. The first phantom is simply a sphere of tissue with unit specific density. The radius is 27 centimeters. A skin dose detector is located 7 milligrams per square centimeter within the surface. An eye lens detector is located 3 millimeters within the surface. A bloodforming organ (BFO) detector is located 5 centimeters within the surface. Other detectors are taken along a radius to permit midline dose calculations.

The second phantom is constructed from two truncated, elliptic cylinders. The top cylinder, 32 centimeters high, contains an eye lens detector 3 millimeters within the surface at the appropriate site. The bottom cylinder represents the trunk of the body. It is 67 centimeters high, 37 centimeters wide, and 22 centimeters thick. Midline dose detectors are located on the minor axis line which runs through the navel. The BFO detectors are placed 5 centimeters below the surface, one on the major axis and one on the minor axis.

The third phantom is a more detailed simulation of the human body. It is 6 feet high and scaled appropriately. The eyeballs are recessed in eye sockets to simulate the extra self-shielding found in practice. Lungs of suitable shape and size are located in the chest cavity. Lung tissue is assigned a density of 0.33 grams per cubic centimeter. Cylindrical bones with density 1.2 are placed in the right arm and leg of the phantom. Midline detectors are located on a minor axis through the navel. Three BFO detectors are located in the bone marrow of the right arm; one near the shoulder, one at the elbow, and one near the wrist. Three additional BFO detectors are located in the bone marrow of the right leg; one near the hip, one at the knee, and one near the ankle.

SHIELD CONFIGURATION

The shield configuration is simplified to permit the effects of phantom geometry to be examined without confusing detail due to shield structure. Each phantom is surrounded by an aluminum shell shield with mass thickness 5 grams per square centimeter. Dose

within the spherical cavity can vary by a factor of two or more due to the so-called "focussing factor". Here, the radius of the shield is set to 50 feet in order that dose variation in the region occupied by the phantom is less than 0.1 percent with the phantom absent.

DOSE COMPARISONS

Figure 3 compares midline dose in rem for the three selected radiation level probabilities and for each of the three phantoms. Remember that midline doses are taken along the 27 centimeter radius of the sphere, and from the navel to the center of the cylindrical and detailed phantoms.

Midline doses for the three phantoms are essentially equal for depths up to 5 or 7 centimeters. At this point, the elliptic cross sections of the cylindrical and detailed phantoms lead to reduced self-shielding and higher doses in comparison with the spherical phantom. The doses are 20 to 50 percent high for phantoms 2 and 3 near their center, compared to the spherical phantom. This difference would be considerably smaller for a 15 centimeter radius sphere. The presence of arms in the detailed phantom is negligible for this particular midline dose curve. It is interesting that increasing lung tissue density from 0.33 to unity has negligible effect on the detailed phantom midline dose shown.

Figure 4 shows rem and rad dose for the spherical man model. The top line of each table represents free field dose; that is, no shield and no phantom. The second line represents what is usually termed a point dose within a shield. Occasionally, this is termed "skin dose", and, in a sense, represents a fourth phantom in this study. Several years ago our sophistication in these calculations increased to the point where we realized that self-shielding would often reduce the skin dose below point dose by a factor of two. These numbers confirm the hypothesis in this situation to within 6 percent. The last two lines in each table show that the eye dose, 3 millimeters below the surface, is about 25 percent lower than the skin dose for this thin shield case. The skin and eye rem doses are 30 to 50 percent larger than the rad doses.

Figure 5, again for the spherical phantom, shows the relative importance of the proton and alpha components of the 0.1 percent spectra. A comparison of the first and third columns shows that the alpha rad dose is 2 to 10 percent of the proton rad dose. A comparison of the second and fourth columns shows that the alpha rem dose is 8 to 50 percent of the proton rem dose.

These data are typical of thin shield results in that proton rem dose is 15 percent larger than proton rad dose, and alpha rem dose is 4 or 5 times larger than alpha rad dose.

Figure 6 compares skin and eye dose for the three phantoms. The skin dose varies only a few percent between phantoms. The eye dose varies less than 20 percent between phantoms.

Thus far, no justification for the necessity of a detailed phantom has been presented. However, it is usually possible to find such a requirement if a diligent search is made. The present case is no exception.

Figure 7 shows dose to the bloodforming organs for each of the cases studied. The spherical phantom has one BFO detector. The cylindrical detector has two; one near the front and one near the side of the trunk. Note that doses to these detectors bracket the dose to the first phantom BFO detector. The BFO doses in the detailed phantom are shown in order from the top of the arm down to the ankle. In this case, the dose variation is as large as a factor of 6.

CONCLUSIONS

It is a little dangerous to draw generalizations from a limited study such as the present one. The fact that the spectrum is fairly soft and the shield thin should emphasize differences between the several phantoms. Nevertheless, these phantoms yield very similar results for all save the widely-distributed BFO's. It is possible that a suitably chosen set of spherical phantoms could have matched the BFO response as well. Alternatively, the midline dose-versus-depth curves might be used to allow for varying self-shielding.

In summary, it appears that spherical man models may safely be used to estimate dose to skin, eyes, midline and possibly bloodforming organs in parametric studies with fairly uniform shields. However, it is very likely that detailed phantoms must be used in conjunction with realistic configurations where shielding is usually not uniform. Detailed phantoms will also be necessary in correlating dosimeter response with dose to specific organs following an exposure.

FIGURE 1

PROTON and ALPHA FLUXES

Mars Mission

450 Days

1986-1987

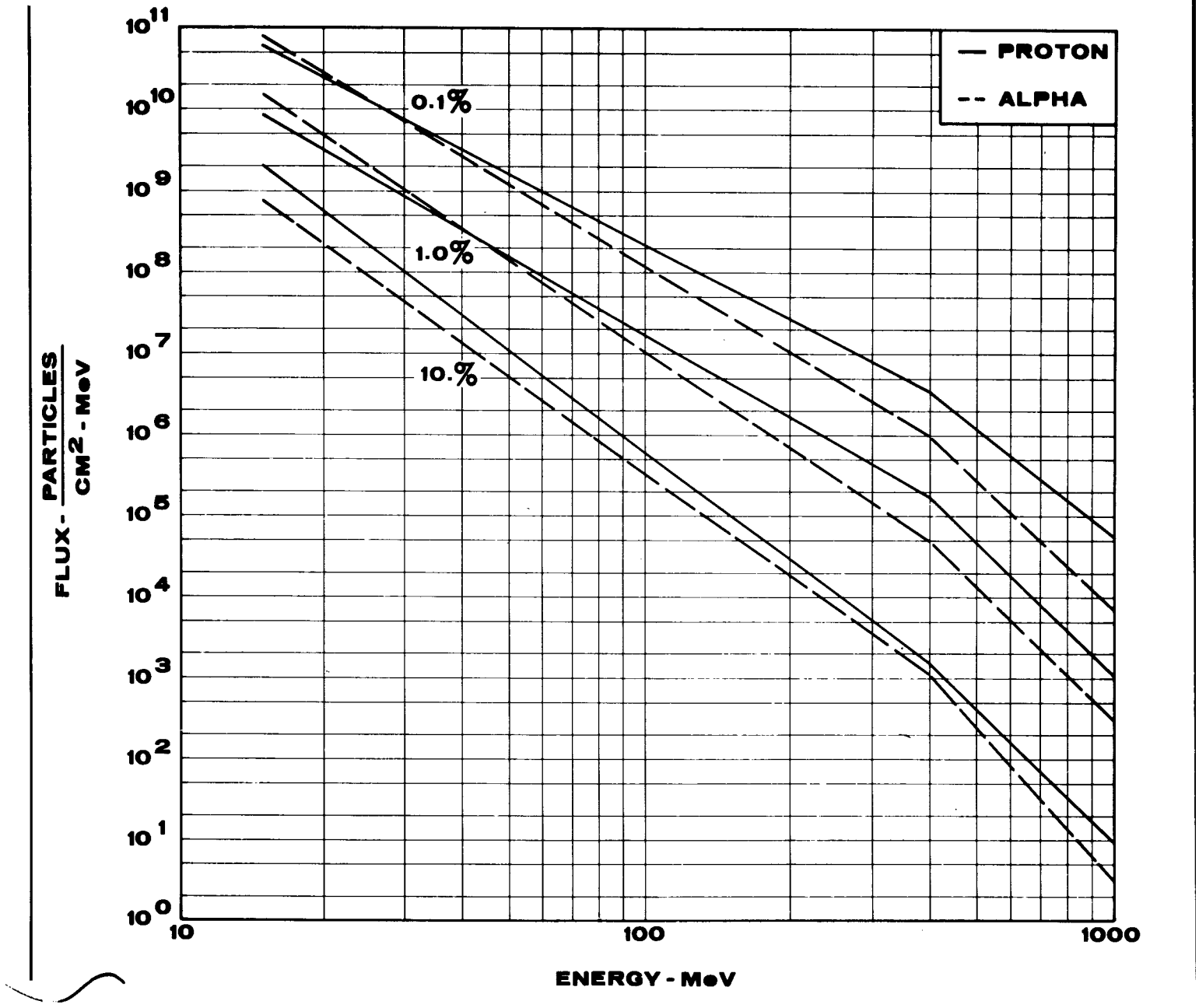

FIGURE 2

PHANTOM GEOMETRIES

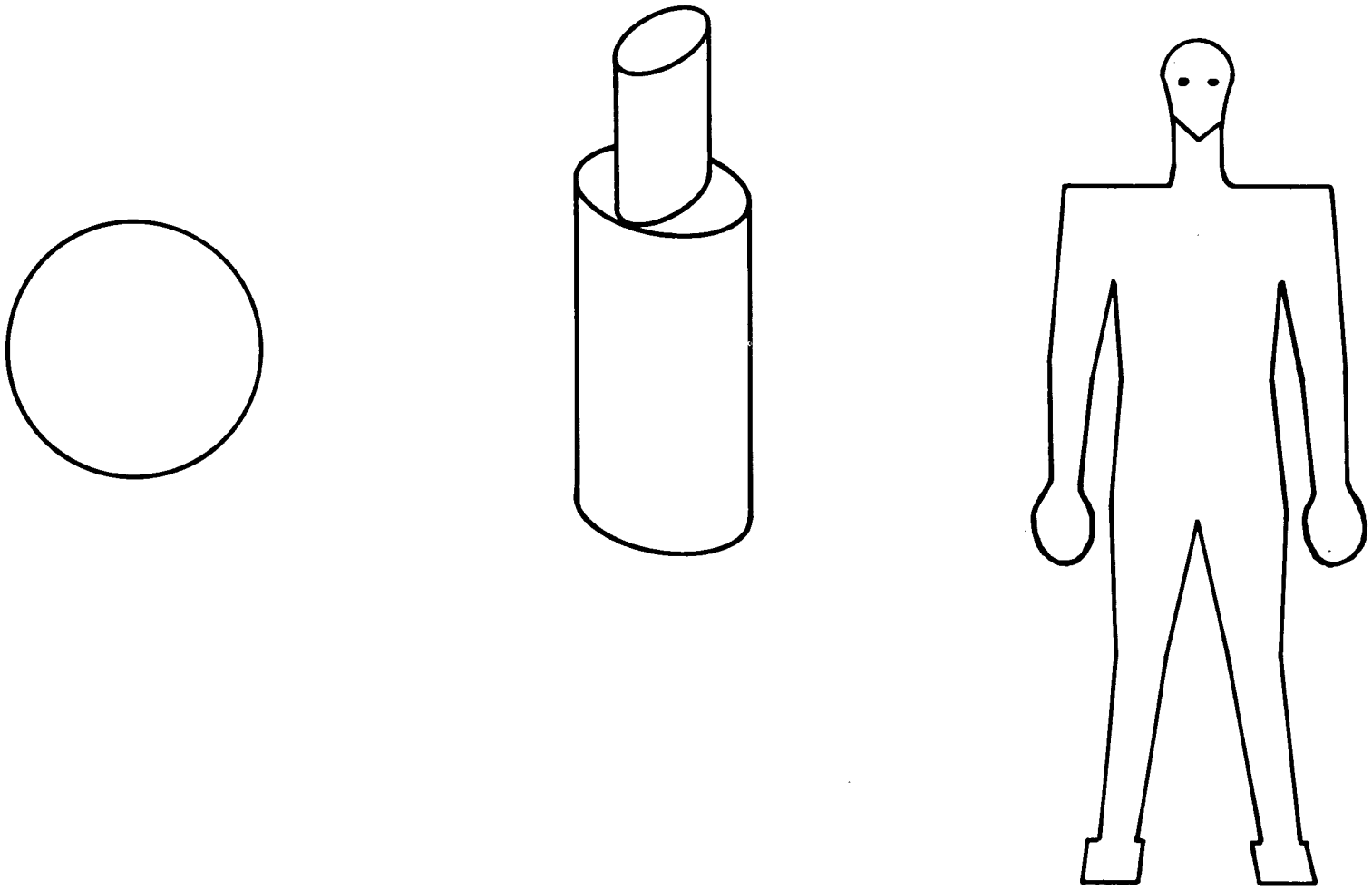

FIGURE 3

PHANTOM

MIDLINE

DOSE

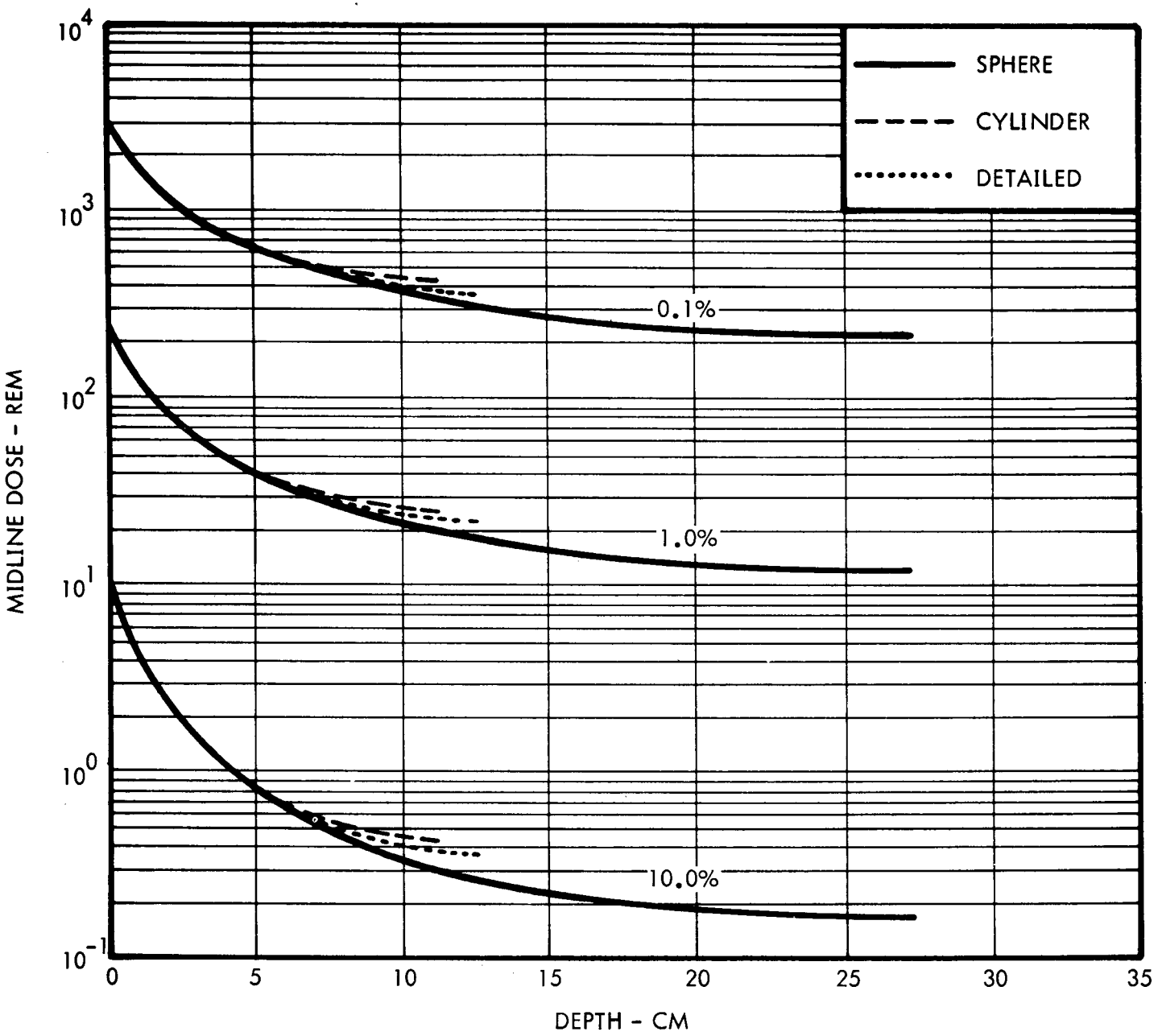

FIGURE 4

TOTAL RAD DOSE

	0.1%	1.0%	10%
NO SHIELD	2.38 + 6	3.89 + 5	5.97 + 3
AL SHIELD	6169.	494.	20.6
SKIN	3273.	258.	10.4
EYE	2444.	178.	6.9

TOTAL REM DOSE

NO SHIELD	1.50 + 7	2.54 + 6	1.85 + 4
AL SHIELD	9411.	699.	26.1
SKIN	4919.	362.	13.2
EYE	3556.	255.	8.7

FIGURE 5

SPHERICAL MAN MODEL

0.1% SPECTRUM

	PROTON		ALPHA	
	RAD	REM	RAD	REM
NO SHIELD	1.71 + 5	1.95 + 5	2.21 + 6	1.48 + 7
AL SHIELD	5582.	6329.	587.	3082.
SKIN	2975.	3329.	298.	1551.
EYE	2236.	2524.	208.	1032.
10 CM	348.	385.	8.	30.

FIGURE 6

TOTAL REM DOSE

	SPHERE	CYLINDER	DETAILED
	0.1% SPECTRUM		
SKIN	4919.	4849.	5082.
EYE	3556.	4092.	3726.
10 CM	414.	490.	454.
	1.0% SPECTRUM		
SKIN	362.	357.	371.
EYE	255.	293.	266.
10 CM	25.	30.	28.

FIGURE 7

BFO TOTAL REM DOSE

		0.1%	1.0%	10%
SPHERE		744.	48.	1.0
CYLINDER		723.	46.	.9
		947.	61.	1.4
DETAILED	ARM	859.	55.	1.2
		1512.	100.	2.6
		2356.	161.	4.7
	LEG	677.	43.	.8
		1404.	92.	2.3
		1636.	108.	2.7

DOSE AND DOSE-EFFECT RELATIONSHIP MODIFYING FACTORS IN PREDICTING THE DEGREE OF BIOLOGICAL RESPONSE

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DOSE AND DOSE-EFFECT RELATIONSHIP MODIFYING FACTORS IN PREDICTING THE DEGREE OF BIOLOGICAL RESPONSE

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Although absorbed dose in tissue is the cardinal parameter determining the degree of biological response, additional factors must be considered. These include principally the dose rate, the macro-distribution of energy deposition (depth-dose pattern), and the micro distribution of energy deposition (radiation quality, or LET). The relative importance of these factors will depend on the nature of the biological response under consideration (e.g., early effects of massive exposure such as mortality, or late effects such as leukemia production). Most accidental massive exposures occur over a period of seconds to hours; and in this dose rate range the dependence of biological effect on dose rate is slight. Likewise the RBE for high-LET radiations encountered practically, mainly neutrons, is close to unity at least for the bone marrow syndrome. The chief modifying factor for use in determining the early effects of massive exposure is then the degree of inhomogeneity of dose distribution on a macro scale. Non-homogeneous dose distribution can be taken into account as far as the bone marrow syndrome is concerned by means of a distribution effectiveness factor that is calculable on the basis of dose distribution, bone marrow stem cell distribution, and the marrow stem cell survival rate which is exponential with dose.

With exposure protracted over months to years, long-term effects become of cardinal importance. Dose rate then becomes a major factor not only in regard to the expected degree of effect for low-LET radiations, but in determining the RBE of high-LET radiations as well. Although the integral dose per gram or the mean dose has no direct usefulness in predicting early mortality, it may be of value in determining the probability of cancer induction at least at relatively low values of total dose. If the cancer induction dose-effect curve is of the linear, no-threshold type, then the probability of cancer induction in an organ may be related to the mean dose per cell, $\Sigma m_i D_i / \Sigma m_i$, where m_i represents the mass of the cell i and D_i represents the absorbed dose to that cell and the expression is summed over the entire organ. This expression is conceptually equivalent to gram-rads/gram. There is evidence, however, that at least some net dose-cancer induction curves pass through a maximum at higher doses, perhaps as the result of interaction of the cancer-induction and a cell killing or other functions. In these dose regions the average dose would not represent a useful parameter for prediction of cancer rates.

RADIATION RESPONSES OF MAN IN THE INTERMEDIATE DOSAGE RANGE

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RADIATION RESPONSES OF MAN IN THE INTERMEDIATE DOSAGE RANGE

Wright H. Langham

INTRODUCTION

In conventional occupational radiation protection, emphasis has rightfully been placed on maximum protection of the individual. Emphasis on maximum protection has led to establishment of maximum permissible exposure levels based on statistical probability of occurrence of late or delayed responses, primarily genetic mutations, general life-shortening, and increased incidence of leukemia and other neoplastic disease. Radiation protection guides for occupational exposure are based on recommendations of the National Committee on Radiation Protection (NCRP), which recommends that radiation exposure to the whole body, head and trunk, active blood-forming organs, gonads, or ocular lens, accumulated at any age, shall not exceed 5 rems multiplied by the number of years beyond age 18 and that exposure in any 13 consecutive weeks shall not exceed 3 rems. Written as an expression,

$$MPL = 5\ (N - 18)\ \text{rems},$$

where MPL is the maximum permissible accumulated exposure in rems and N is the individual's age in years. Not only is the total accumulated exposure regulated but the accumulation rate as well. There are possible circumstances, however, in which total exposure and accumulation rate cannot be controlled, and radiation risk may not be confined to the probability of late or delayed responses only. Examples of such possible circumstances are military and civil defense operations, emergency criticality situations, and possibly manned space exploration. Under the prevailing conditions, it may be necessary to emphasize the amount of radiation man can absorb and withstand rather than maximum protection of the individual.

Knowledge of man's response to radiation exposure is confined largely to extrapolation from animal observations and to occasional radiation accidents and exposures of patients for medical purposes. Animal studies have concentrated heavily on late or delayed effects (with emphasis on occupational exposure levels and exposure levels for the general population) and on lethal and supralethal effects of high doses and high dose rates. Emphasis in the latter case has stemmed from military interest in radiation exposures required for production of early incapacitation or debilitation. For the situations mentioned previously, there is a need for more information on radiation damage of animals and man in the dosage range above the maximum occupational exposure limit

up to the LD_{50}. Dearth of information over this dosage range results largely from a lack of suitable quantitative end points for evaluating the level, degree, or severity of effect. An animal exposed to one-half an LD_{50} dose either dies (in which case death automatically becomes the end point) or survives (in which case the end point becomes the probability of expression of a late or delayed effect). There is no satisfactory quantitative method for evaluating or expressing the current radiation status of an animal or his ability to tolerate additional exposure. If it is not feasible to control exposure rate, it is necessary to consider the probability of occurrence of both early and late radiation effects when considering man's response in the intermediate dosage range.

CLASSIFICATION OF RADIATION EFFECTS

Radiation effects may be divided into two general categories: somatic and genetic. Somatic effects are those manifested directly by the irradiated individual, in contrast to genetic effects which show up only in his progeny. Only the former are considered here. Somatic effects may be divided further into early and late. Early effects are taken arbitrarily as those occurring within 30 to 60 days after exposure to relatively high doses ($> \sim 50$ rads) delivered at relatively high dose rates (several rads/hr). Late effects are those occurring only after many months or many years. Late effects may result from a single high-intensity exposure or from gradual dose accumulation at low dose rate over protracted periods. They are assumed to be nonthreshold phenomena and probabilistic functions of the total dose. Although it is possible to avoid manifestation of early radiation responses by dose protraction, it is not possible to avoid actuarial risk of late effects. In keeping with the above discussion, the more significant radiation effects may be classified as follows:

I. Somatic Effects

1. Early effects

   a. Skin erythema and desquamation
   b. Prodromal response
   c. Hematological depression
   d. Early lethality
   e. Decreased fertility and sterility

2. Late effects

   a. Permanent or delayed skin changes
   b. Increased incidence of cataract
   c. Increased incidence of leukemia and other neoplastic disease
   d. General life shortening

II. Genetic Effects

A rather extensive survey of reports of pertinent animal studies, accident cases, and clinical observations (not referenced for purposes of this brief summary) gives some basis for gross estimates of absorbed doses (at the anatomical site or region of

interest) of high-intensity penetrating x or gamma radiation required to produce approximately a 50 percent level of early responses in an irradiated population and some indication of the probability of risk of late manifestations of radiation damage.

EARLY EFFECTS

Skin Erythema and Desquamation: Controlled observations on human skin and clinical experiences suggest that prompt doses of 550 to 650 rads of 200- to 250-kVp x rays to small areas, measured at the depth of the basal cell layer ($\sim$0.1 mm depth), may produce mild transient erythema in $\sim$ 50 percent of cases. The dose required is highly dependent on dose rate and skin texture. With increasing dose, erythema may progress to dry and then to moist desquamation. The 50 percent moist desquamating dose appears to be about 2000 rads.

Prodromal Reaction: The prodromal symptom complex (consisting of anorexia, fatigue, nausea, vomiting, and diarrhea) is the earliest manifestation of high-intensity radiation exposure. Signs and symptoms may appear within 2 to 6 hours after exposure and persist for 1 to 2 days, depending on dose. With the exception of diarrhea (which seems to be prominent only in the lethal dose range), the 50 percent effective dose for production of prodromal signs and symptoms appears to be in the dose range of 100 to 200 rads measured at the approximate midline of the body. The epigastric region of the body is the most sensitive. The mechanism of action of radiation in producing the prodromal response is not thoroughly understood. Psychological factors are important in individual responses, making any dose-response relationship highly conjectural.

Hematological Depression: Significant high-intensity exposure of the bone marrow is reflected by a drop in count of the formed elements of the circulating blood, primarily lymphocytes, neutrophils, and platelets. Day-to-day fluctuation in counts in both the normal and pathological state precludes the use of blood counts as an indication of the level of radiation damage or exposure of the individual. Absorbed doses at the average depth of the bone marrow (assumed to be 5 cm) of 100 to 200 rads of x or gamma radiation may be associated with mean blood count depressions in the population of about 50 percent of normal at the nadir. The time of maximum depression is dose-dependent. Blood count progression as a function of time after exposure may be of considerable prognostic value but is of little value as a biological dosimeter.

Early Lethality: Early lethality is an augmentation of the hematological radiation syndrome with increasing dose. It begins with the prodromal reaction followed by hematological depression, which terminates in fever, infection, and death in about 2 to 8 weeks if the bone marrow fails to recover. The LD_{50} for man is not known. The various estimates (based entirely on indirect methods or judgments) of the LD_{50} for man range from about 200 to 700 rads or R. Much of the apparent disagreement in estimates stems from vagueness or unwillingness to specify conditions including specific definition of dose or exposure. Currently (in the opinion of the author), the best estimate of the whole-body

absorbed dose of high-intensity penetrating x or gamma radiation for the LD_{50} in man is about 300 rads, measured at the approximate midline of the body.

Decreased Fertility and Sterility: The germinal epithelium appears to be the most radiosensitive tissue in the body. Radiation damage to the germinal epithelium in the male is reflected by a drop in sperm count some 7 to 8 weeks after exposure. As little as 15 to 20 rads of high-intensity x irradiation may cause a drop in sperm count to about 50 percent of normal, and 100 rads may cause azoospermia and sterility of limited duration in some individuals. Doses of ~600 rads or greater are required to produce permanent sterility. Libido is unaffected by sterilizing doses. Because of wide day-to-day fluctuations in both normal and pathological states, sperm counts are of little value as an indicator of degree or extent of radiation damage or exposure.

LATE OR DELAYED EFFECTS

Permanent or Late Skin Changes: Minimal permanent or late skin changes accompany all high-intensity radiation doses that produce an early moist desquamation reaction. These minimal changes usually consist of slight change in skin texture, telangiectasia, and slight pigmentary manifestations. With increasing dose, these changes may progress to necrosis, ulceration, and skin cancer. The dose of high-intensity x irradiation (to small areas) for production of about a 50 percent probability of necrosis appears to be about 2500 to 3000 rads. The required dose may be approximately doubled when exposure is protracted or fractionated over a period of several weeks. The higher the accumulated dose, the greater the probability that the permanent or late changes produced may progress to the cancerous stage.

Late Changes in the Ocular Lens: Radiation exposure to the eyes results in development of opacities in the ocular lens which may progress to the stage of a cataract and impair vision. Retrospective observations of a few hundred patients that received exposure to the eyes suggest that the effective dose of high-intensity x irradiation for production of minimal opacities is about 200 rads, measured at the depth of the front surface of the lens (~ 3 mm). A single dose of about 750 to 800 rads may be associated with a probability approaching unity for production of permanent lens changes. The higher the dose, the greater the probability that the changes will progress to the stage of a cataract and the shorter the latent period prior to development. A 50 percent incidence of lens damage may occur with single doses of 400 to 500 rads. Some lessening of effect may result from dose protraction or fractionation over periods of several weeks. The lens of the eye seems quite sensitive to radiation quality (LET), and fission neutrons appear as much as 10 times as effective per rad as x or gamma radiation.

Increased Incidence of Leukemia and Other Neoplastic Disease:

Radiation exposure of the total body or a large fraction of the bone marrow is known to increase the probability of developing leukemia and probably (although not proven) other neoplastic conditions. Observations of the Japanese atomic bomb survivors and

people exposed for medical purposes suggest that the increase in leukemia incidence is proportional to accumulated dose (above 100 to 300 rads) and that the best estimate of risk from penetrating x- or gamma-ray exposure is about 1 to 2 chances/10^6 man-years/rad for protracted or fractionated exposure and 3 to 4 chances/10^6 man-years/rad for single high-intensity exposure. Nothing presently can be said about specific dose-response relationships for other types of cancer, although some information is beginning to accumulate from the observations of the Atomic Bomb Casualty Commission.

General Life Shortening: Whole-body radiation exposure produces a statistical decrease in after-expectation of life which appears proportional to the accumulated dose. Retrospective studies of medical radiologists and observations of irradiated animal populations suggest that the life-shortening risk in man from whole-body protracted or fractionated x-ray exposure may be of the order of 2 to 3 days/rad measured at the approximate mid-line of the body. For single high-intensity exposure, the risk may be higher by a factor of ~ 3.

GENERAL COMMENTS

The previous discussion of dose-response relationships in man is based on exposures to penetrating x or gamma radiation and is subject to modification by a number of exposure factors and conditions. Among the most important modifying factors are radiation quality (LET), dose protraction (either by low dose rate or dose fractionation), and nonhomogeneities in dose distribution (both topical and in depth). Furthermore, nothing has been said about doses corresponding to response probabilities above and below the 50 percent level. Although one may hazard an estimate of the mean response dose, there is little information on which to predict the variance or distribution of sensitivities that may exist in the population. The variance of the population is a very important parameter in any quantitative risk prediction or evaluation.

CELLULAR AND MAMMALIAN RADIATION EFFECTS AND THEIR INTERPRETATION IN RELATION TO MANNED SPACE FLIGHT

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CELLULAR AND MAMMALIAN RADIATION EFFECTS AND THEIR INTERPRETATION IN RELATION TO MANNED SPACE FLIGHT

Paul Todd

If major concern involves the response of man in the intermediate dose range (above recommended limits but sublethal), then the so-called "early effects" are among the most relevant to the completion of an extended mission. Among the important early effects on man are skin erythema, prodromal (immediate) gastrointestinal response, and hematological depression. Although the etiology of the prodromal response is poorly understood, it is generally agreed that the responses of skin, bone marrow, and lymphatic tissues are due to the inhibition of the reproductive capacity of stem cells in these tissues. It thus becomes relevant to examine some of the factors which modify the effects of ionizing radiations upon the reproductive capacities of human cells. The reproductive capacity of human cells is retained in tissue culture over many years and can be assayed by the ability of single cells to reproduce and develop into "colonies" visible to the naked eye. With this assay, the following modifications of the inhibition of reproduction by ionizing radiation have been found:

(1) The removal of oxygen reduces by a factor of 3 the effective x or gamma ray dose.

(2) Cysteamine, a radioprotective drug, in maximum tolerated concentrations also reduces by a factor of about 3 the effective x or gamma ray dose.

(3) Doses spaced more than about 2 hours apart are reduced in effectiveness compared to acute exposure, due to the cells' rapid repair of sublethal injury.

(4) For the same reason reduced dose rates result in reduced inactivation.

(5) Ultrasonication of irradiated cells results in increased chromosome breakage and reduced survival.

(6) Chromosome deletions produced by beta radiation are produced at a greater frequency in the gravity-free state.

(7) Particulate radiations with a higher rate of energy loss inactivate cells more effectively.

(8) Particulate radiations do not appear to differ from

x rays in their dependence upon the atomic number of the material surrounding the cells.

(9) Prior irradiation results in occasional debilitated cells which produce radiation-sensitive progeny which are eventually outgrown by normal surviving cells.

A few examples of the *in vivo* counterparts of these effects have been reported. The protective effect of anoxia and sulfur-containing drugs has been demonstrated in the cells of rodents. Dose-rate reduction and fractionation studies indicate that the early effects of gamma rays are characterized by a certain amount of irreparable injury, immediate recovery of sublethal cellular injury, and tissue repopulation by surviving cells. Neutrons and alpha particles are decidedly more effective in producing skin lesions than are x or gamma rays. However, particulate radiations appear to produce gastrointestinal effects in rodents in preference to hematological effects. Hypothermia appears to protect rodents from radiation effects -- possibly due to the resulting hypoxia. The cellular responses in the intact organism are complicated by hormones and biochemical processes occurring in other than "critical" organs -- possibly indicated by the observations that exercise before exposure slightly protects rodents, but exercise after sensitizes.

Some physiological effects of radiation on mammals probably deserve consideration with respect to mission accomplishment: impairment of vestibular function in rabbits has been observed at doses below 500 rads, and the tolerance of rodents to cold and exercise is impaired by x ray doses in the intermediate range.

It must be kept in mind that radiation exposures in the space flight condition may not be equivalent to those on earth, in the sense that the astronaut's body must be prepared to tolerate a variety of additional stresses not commonly encountered in everyday life, and, furthermore, conditions prior to and following exposure are not likely to be at all similar to ground-level conditions. Tolerance dose magnitudes may have to be adjusted accordingly. Combined stresses may work in a variety of ways:

(1) A stress applied before or after exposure exaggerates the radiation response.

(2) A stress applied at any time simply adds to the radiation stress.

(3) An agent counteracts the effects of radiation.

(4) Radiation reduces tolerance to the additional stress.

A limited number of such effects have been recorded, mostly on physiology or viability of laboratory rodents. Hypothermia prior to exposure protects rodents from radiation lethality as does prior exercise (category 3). Intense exercise afterward enhances radiation sensitivity of rodents (category 1). Vibration and radiation appear to reduce tolerance to each other (categories 1 and 4). As noted above, radiation reduces tolerance to cold and exercise (category 4).

Aside from known observations on animals, there are other agents worthy of speculative consideration. For example, will the effect of emotional excitement (associated with increased heart rates in astronauts) in the gastrointestinal system add to the effect of "intermediate" doses of ionizing radiation? Will the observed decreased blood count encountered in long missions add to the effect of radiation on the blood-forming organs? In the unlikely event of radiation exposure during intense acceleration, will the hypoxic tissues be spared? If temperature control is lost in containment suits, will the effect of hyperthermia add to the "fever" associated with radiation sickness? Will weightlessness have the same effect on radiation response as it has on cells outside the body? Will exposure during hyperthermia (if that is what ultrasound produces) result in enhanced cellular effects?

An attempt is made to summarize all of these considerations in the following table:

AGENT	EFFECT OF RADIATION ON RESPONSE TO AGENT	EFFECT OF AGENT ON RADIATION RESPONSE
Radiation	Sensitizes	Sensitizes
Weightlessness	Additive with anemia and dehydration	Sensitizes chromosomes
Acceleration	Impairs vestibular function. Weakens vascular & GI systems	Hypoxia may protect during exposure
Vibration	Sensitizes rodents	Sensitizes rodents
Heat	Additive?	Ultrasound sensitizes cells
Cold	Sensitizes rodents	Protects rodents
Excitement	Sensitizes GI tract?	Additive with intestinal cell loss?
Exercise	Reduces tolerance of rodents	Protects before; sensitizes after

ENERGY-LOSS DISTRIBUTIONS AND FRACTIONAL CELL LETHALITY*

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ENERGY-LOSS DISTRIBUTIONS AND FRACTIONAL CELL LETHALITY

Stanley B. Curtis

ABSTRACT

In the evaluation of the hazard from a given radiation environment, various factors other than the absorbed dose play an important role in determining the biological response. One of these is the quality of the radiation, that is, the dE/dx of the particles depositing the dose. It is convenient, especially when dealing with charged particles heavier than electrons, to display the dose at a point as a function of dE/dx. This function is called an energy-loss or dE/dx distribution. Such a representation allows an evaluation of the importance of the various dE/dx components that comprise the dose. In particular, the high dE/dx components are of interest because it has been shown that, in general, high dE/dx radiation is more effective--that is, has a higher RBE (Relative Biological Effectiveness) in producing biological damage than low dE/dx radiation. Examples are given of dE/dx distributions due to two typical solar-particle events in free space with different spectral shapes and under different shielding thicknesses. The case of a steep spectrum under thick shielding shows the proton component dominating, while the case of a flatter spectrum under thin shielding shows the helium-ion component to be slightly more important than the proton component.

A potentially fruitful way of quantifying the biological effects of a given environment is by using the inactivation cross section. This experimentally determinable quantity is equal to the probability per unit flux of a cell being inactivated, and is analogous, in this sense, to a nuclear scattering or interaction cross section. It appears to be a function of dE/dx, but does not depend on the type of heavy particle producing the dE/dx. Unfortunately,

few mammalian inactivation cross sections have been experimentally determined to date. Todd has measured the inhibition of the proliferative capacity of human kidney cells in vitro and has shown that the resulting damage may be interpreted as being caused by two distinct damage mechanisms--an irreversible single-hit mechanism dominating at high dE/dx, and a reversible multi-hit mechanism dominating at low dE/dx. As an illustration, the cross sections from these experiments have been used to calculate the numbers of inactivation hits/cell for two sample radiation environments involving protons, helium ions, and heavier components: the galactic cosmic radiation in free space under 0.2 g/cm^2 water shielding and a large solar-particle event in free space. Presented in terms of the ratio of inactivation hits/cell of the heavy components to that of the protons, the results show: (1) For the galactic cosmic radiation, the very heavy components (Fe-Ni ions) cause one and a half times as much damage as protons under 0.2 g/cm^2 shielding. (2) For the solar-particle event, the helium-ion contribution is slightly less than the proton contribution, but is the same order of magnitude and remains so even at large shielding thicknesses. The heavier-component contribution is down by an order of magnitude from that of the helium ions, and drops off much more steeply with increasing shielding thickness.

The fractional number of cells inactivated or fractional cell lethality (FCL) can be calculated if the numbers of lethal hits/cell are known from both the reversible and irreversible damage mechanisms. It turns out that irreversible damage dominates for the solar events chosen for illustration. FCL values were calculated for two points inside the body at the waist of a seated astronaut for several large solar-particle events of the last solar cycle, taking into account the body self-shielding. The results show that up to 7% of the cells would have been inactivated 4 cm inside the body at the waist behind 1 g/cm^2 of vehicular shielding in the largest event.

Such calculations as this may help in the future for the evaluation of the hazard from mixed-heavy-particle radiation environments when inactivation cross sections or other suitable "malfunction" cross sections are available for more critical and perhaps irreplaceable cells in the body and when accumulated damage over a long period, such as for extended space flight, is of importance.

INTRODUCTION

The problem of evaluating the hazard from a given radiation environment can be very complex. In the first place, the differing interactions of the various types of radiation make the analysis difficult. Secondly, the shielding of the human body itself provides an added complication in the determination of the particle flux to reach a point deep within the body. Finally, the ultimate biological effect depends not only on the amount of energy deposited by the particles per unit volume (i.e., the absorbed dose) but also on such quantities as the dose rate and the ionizing power (dE/dx of the particles. We consider here only one aspect of the problem: the dependence of the biological effect on the dE/dx of the particles depositing the dose. All material presented in this paper except that on the galactic cosmic rays has been published elsewhere.[1,2]

It has been clearly demonstrated[3,4] that the relative biological effectiveness (RBE) of radiation from charged particles in mammalian systems depends on the rate of energy loss of the particles, that is, on their dE/dx. Other parameters (such as the amount of energy deposited in a finite sensitive volume) may ultimately be used to describe the quality of the radiation from the biological standpoint;[5] but until more is known about the effects of microscopic distributions of energy in specific biological systems of interest, it appears reasonable to continue to use dE/dx as a rough approximation in all systems.

ENERGY-LOSS DISTRIBUTIONS

It is convenient, therefore, to express the absorbed dose at a point in terms of an energy-loss distribution or spectrum. We define this function in the following way. First, we recall the expression for the differential dose element:

$$dD = (dJ/dE')\, \epsilon'\, dE' \quad , \tag{1}$$

where dJ/dE' is the differential energy spectrum; i.e., the number of particles per unit area with energies between E' and $(E' + dE')$ at the point of dose computation, and $\epsilon' = dE'/dx$ is the rate of energy loss of a particle with energy E'.

The integral of the above expression is the absorbed dose at the point:

$$\text{dose (in rads)} = 1.6 \times 10^{-8} \int_0^{\infty} \frac{dJ}{dE'}\, \epsilon'\, dE' \quad ,$$

where dJ/dE' is expressed in number of particles per cm^2-MeV, ϵ' is in MeV cm^2/g, and E' is in MeV. We assume here that the energy being lost is absorbed "locally" and so we restrict ourselves to incident charged particles heavier than electrons. In addition, we are neglecting in this first approximation the fact that, in some cases, high-energy secondary electrons or delta rays can deposit energy some distance from the track core.

We now define a function called the energy loss or dE/dx distribution function, $F(\epsilon')$, such that

$$F(\epsilon')\, d(\log \epsilon') = dD \quad . \tag{2}$$

We use the differential of the logarithm in the definition simply for convenience, since we shall see that it is convenient to plot the distribution as a linear function of the log of ϵ'.

Equating (1) and (2) and solving for $F(\epsilon')$, we obtain

$$F(\epsilon') = 2.303(dJ/dE')\, \epsilon'^2/(d\epsilon'/dE') \tag{3}$$

for the energy-loss distribution function. It is seen from Eq. (3) that $F(\epsilon')$ diverges whenever $d\epsilon'/dE'$ vanishes, i.e. at the maximum and minimum of the dE/dx vs E curve. These divergences show up in the distributions as "spikes."

From the definition of $F(\epsilon')$, in units of MeV/g, we have

$$\text{dose (in rads)} = 1.6 \times 10^{-8} \int_{-\infty}^{\infty} F(\epsilon')d(\log \epsilon') \quad . \tag{4}$$

If $F(\epsilon')$ is plotted graphically as a function of ϵ' on a logarithmic scale, equal distances along the abscissas have equal weights, and the importance of different dE/dx contributions can readily be evaluated. The spikes or points of divergence give no trouble in the dose integral because the areas under them contribute a small part of the total dose in a typical exposure situation.

ENERGY-LOSS DISTRIBUTION FROM A SOLAR-PARTICLE EVENT

The proton, helium- and heavier-ion fluxes comprising a solar-particle event provide a good example of a situation where energy-loss distributions are helpful in indicating the relative importance of various dE/dx contributions. Figure 1 shows energy-loss distributions resulting from two different energy spectra behind two different shielding thicknesses. Figure 1a gives the case for thick shielding, 5 g/cm^2 water equivalent and a rather steep particle spectrum. Figure 1b gives the case for thinner shielding, 1 g/cm^2 water

equivalent, and a flatter spectral shape. It was assumed that the integral particle spectra were exponential in rigidity with the form

$$J_j(\geq P) = J_{0j} \exp(-P/P_{0j}) \quad ,$$

where P is the rigidity or momentum per unit charge, $J_j(\geq P)$ is the number of particles per cm^2 of the jth particle type with rigidity equal to or greater than P, and J_{0j} and P_{0j} are constants for a given event. In Fig. 1a, we have $P_{0p} = P_{0\alpha} = P_{0M} = 80$ MV, which exemplifies a rather steep spectrum, and in Fig. 1b, $P_{0p} = P_{0\alpha} = P_{0M} = 180$ MV, which exemplifies a flatter spectrum. Here p, α, and M respectively stand for protons, helium ions, and ions of charge Z between 6 and 9, called M particles. In these calculations, the latter were assumed to have Z = 8. The J_0 values for protons and helium-ions were assumed to be sixty times that for the M particles. All these assumptions are reasonable from the limited data available on the spectra and composition of the larger solar-particle events that occurred in cycle 19.[6,7,8] In the figure, the areas under the curves have been normalized to unity by dividing by the dose.

The difference in relative contribution to the energy-loss distributions of the proton and helium-ion components arises about equally from the change in spectral shape and the change in shielding thickness. This example illustrates the relative importance of high- and low-dE/dx particles in contributing to the dose under differing environmental conditions for two typical large solar-particle events.

BIOLOGICAL CONSIDERATIONS

In the evaluation of a hazard from a specific radiation environment, the radiosensitivity of the biological organism involved must be considered. As indicated above, one parameter upon which this sensitivity depends is the dE/dx of the particles depositing the energy. The International Commission on Radiological Protection has quantified this concept; the value of this quantity is called the quality factor (QF).[9] In addition, the following dependence of QF on dE/dx has been suggested:

$$QF(\varepsilon) = 0.8 \times 10^{-2}\ \varepsilon$$

for QF < 20.0 and ε in MeV cm^2/g. The biologically important dose or dose equivalent in rem may be calculated as

$$\text{dose equivalent} = 1.6 \times 10^{-8} \int_{-\infty}^{\infty} F(\epsilon')QF(\epsilon')\, d(\log \epsilon') \quad .$$

Here the QF acts as a weighting factor that gives more weight to the higher dE/dx portion of the distribution.

The concept of QF, however, is artificial in the sense that its dependence on ϵ has simply been agreed upon as an upper-limit extrapolation to low dose rates and low doses of RBE's from radiobiological data. It would be of interest to use a more physically meaningful quantity whose dependence on dE/dx is perhaps similar but whose interpretation is that of the probability of a biologically significant interaction taking place. Such a quantity should be independent of dose rate, total dose, and all other physical characteristics of the radiation environment, and should have functional dependence only on the dE/dx of the radiation. This quantity could, of course, depend on biologically important parameters, e. g., on the availability of oxygen.

INACTIVATION CROSS SECTIONS

The inactivation cross section measured by Todd is an example of such a quantity.[10,11] In experiments with human kidney cells, Todd has measured the inactivation cross sections for inhibition of the cell's proliferative capacity in vitro as a function of dE/dx with various heavy ions at the HILAC of the Lawrence Radiation Laboratory. Evidence has been found for two types of radiation damage existing in the kidney cells studied. One type is irreparable; the other is reparable. Dependence of the inactivation cross section on dE/dx is different for the two types of damage. These cross sections are analogous to nuclear-scattering cross sections in that they are the probability per unit flux of the proliferative capacity of the cell being destroyed. The experimental cross sections are shown as a function of dE/dx in Fig. 2.[11] The cross section due to irreparable damage is labeled σ_1, and that due to reparable damage is labeled σ_2. Although both cross sections and therefore inactivation probabilities rise with increasing dE/dx, it should be noted that the reparable cross section dominates at low dE/dx, and the irreparable cross section at high dE/dx. This is consistent with the experimental fact that high dE/dx radiation in general produces more irreparable damage, while the damage caused by low dE/dx radiation is more readily reparable.

INACTIVATION HITS PER SITE

By using inactivation cross sections and the dE/dx distribution as described above, the number of lethal or inactivating hits per site can be calculated. The expression for the number of such hits, J(x), at a depth x is

$$J(x) = \int_0^\infty \frac{dJ}{dE'} \sigma(E')\, dE' \quad , \tag{5}$$

where dJ/dE' is the differential energy spectrum at a depth x, and $\sigma(E')$ is the inactivation cross section. This may be rewritten in terms of the energy-loss spectrum $F(\epsilon')$ of Eq. (3) as

$$J(x) = \int_{-\infty}^{\infty} F(\epsilon') \frac{\sigma(\epsilon')}{\epsilon'} d(\log \epsilon') \quad . \tag{6}$$

The integrand of Eq. (6) is just the dose integrand of Eq. (4) multiplied by a factor $\sigma(\epsilon')/\epsilon'$. This factor is analogous to a QF or RBE, but is independent of dose and dose rate, and depends only on the probability for inactivation, and on the dE/dx of the particle involved.

GALACTIC COSMIC-RAY HAZARD TO THE SKIN

As another example in the use of these concepts, the energy loss distribution for the galactic cosmic radiation under 0.2 gm/cm^2 of water-equivalent shielding is shown in Fig. 3. Only the most important contributions are shown here. When more than one nuclear species is present under a single designation, such as the M-particle group ($6 \leq Z \leq 9$), a representative Z and A have been chosen for that group. Recent experimental data have been used[12-16] and were extrapolated to lower energies where necessary. Above the distribution on the graph, the quantity $\sigma_1(\epsilon')/\epsilon'$ is plotted on an arbitrary scale. This indicates the weighting factor chosen in the computation of lethal or inactivation hits/site. The magnitudes of the lethal hits per site in this example are not meaningful, since the cross sections used were those measured for kidney cells in vitro. Unfortunately, inactivation cross sections for cells of the skin are not available at present. These would be more appropriate in a situation in space, for instance, where an astronaut in a space suit was engaging in extravehicular activity. Even in this situation, there would be shielding present--his own body and the nearby spacecraft--which would modify the result by decreasing the contributions from the high dE/dx particles, since they would not

be able to penetrate the nearby material without fragmenting and producing secondary particles. However, the ratios, of the values of lethal hits per site by a heavy component to that by protons gives us a feeling for the relative importance of the various components in causing skin damage. All that is required for the validity of this analysis is that the shape of the inactivation cross section for skin cells be similar to that for kidney cells. Inactivation cross sections have been found to have similar shapes in many different kinds of biological test objects even as far removed from human cells as haploid yeast cells and T1 bacteriophage.[11] Table I presents the ratios of irreparable lethal hits (i.e. from the σ_1 damage mechanism) of the various heavy components compared to those of protons. Only σ_1 damage was assumed, since σ_2 damage is reparable and presumably will be repaired at the low doses involved. It is seen that all components make roughly equal contributions, with the iron-nickel ion group making the largest contribution at these small depths. Deeper within the body, secondary production becomes important, and the calculation is not as straightforward. In principle, however, the calculation can be made for any position where the differential energy spectra of the various particles are known or can be calculated and the probability or cross section for a specific kind of biological damage or functional degradation is known as a function of dE/dx.

THE FRACTIONAL CELL LETHALITY CONCEPT

We now define the fractional cell lethality (FCL) as the fractional number of cells or sites killed or inactivated by the radiation.[17] If we define $\varphi(x)$ as the probability that at a depth, x, a site is still active, or in other words the fractional number of sites still active, then the change in φ in a time dt will be given by

$$-d\varphi = J(x,t)\,\varphi(x)\,dt \quad ,$$

where $J(x,t)$ is the number of inactivation hits per unit time at depth x. Integrating we obtain the familiar exponential dependence

$$\varphi(x) = \exp[-J(x)] \quad ,$$

where $J(x)$ is the time-integrated number of inactivation hits per site from Eq. (5) or (6).

For a single-hit damage mechanism, the FCL is simply

$$FCL(x) = 1 - \varphi(x) = 1-\exp[-J(x)] \quad .$$

For a combination of a single and a multi-hit mechanism as proposed by Todd, the expression becomes

$$FCL(x) = 1-\exp[-J_{\sigma_1}(x)] \left\{ 1 - [1-\exp(-J_{\sigma_2}[x])]^n \right\} \quad ,$$

where $J_{\sigma_1}(x) = \sum_j J_j(x)$ for the single-hit mechanism, and $J_{\sigma_2}(x)$ is similarly defined for the multi-hit mechanism. The summation is over the different types of particles present in the spectrum. The exponent, n, may be interpreted as the number of hits necessary to inactivate the site by the multi-hit mechanism. Its value is not important at low doses, since damage from the multi-hit mechanism is negligible.

FRACTIONAL CELL LETHALITY FROM SOLAR-PARTICLE EVENTS

As a final example, we calculate the FCL to an astronaut's kidney from several of the large events that occurred in solar cycle 19. The physical parameters for the various events are given in Table II and come from the work of Webber.[18] The contributions to the lethal hits per site from the heavier components relative to that from the protons are shown in Fig. 4 as a function of equivalent water shielding for the 12 November 1960 event. Also shown are the relative rad doses from each component for comparison. It is seen that the proton contribution dominates the He-ion contribution, although both are of the same order of magnitude and remain so, even at larger shielding thicknesses. The M particle contribution is down by an order of magnitude from the He-ion contribution and drops off more steeply with increasing thickness.

Calculations of FCL have been made at two points within the body of a seated astronaut for the three solar-particle events given in Table II. These calculations included the self-shielding provided by the body. In this case, we write the number of lethal hits per site at a body point and behind X g/cm^2 of vehicular shielding for the jth particle type as

$$J_j(X, \text{ body point}) = \sum_i f(x_i) J_j(X + x_i) \quad ,$$

where $f(x_i)$ is the fractional solid angle seen from the body point through a body thickness, x_i. These factors, which weight the J_j according to the distribution of body shielding around the point, have been calculated for various points within a seated 75-percentile man.[19] The two points chosen here were 4 and 6 cm into the body at the waist (right side, 25 cm up from the seat level, on the mid-sagittal line). The results are shown in Fig. 5. It is seen that

up to 7% of the kidney cells 4 cm inside the waist would have been inactivated in the 12 November 1960 event under 1 g/cm^2 vehicular shielding.

This calculation is just an example of how available cross-section data might be used to determine the biological damage and thus to evaluate the radiation hazard. Certainly, other cells are more critical to the body than kidney cells. It is hoped that inactivation cross sections or perhaps some other measurable quantity such as a malfunction cross section will be measured in the future for other more critical and perhaps less easily replaceable body cells. In addition, a way must be found to relate the calculated FCL to the functional degradation of the organ being considered. When such data become available, the problem of relating biological effects and functional degradation to particle energy can be more easily handled. It is felt that the FCL concept will be of some help in providing a quantitative measure of the hazard in situations where highly ionizing radiation and, therefore, irreparable processes play an important role.

Table I

Ratio of Lethal Hits by Heavy Particles to Lethal Hits by Protons in the Galactic Cosmic Radiation under 0.2 g/cm^2 Water Shielding

Particle	Z	Ratio
protons	1	1.00
He ions	2	0.72
M ions	6 to 9	0.78
LH ions	10 to 14	0.89
Fe-Ni ions	26 to 28	1.5

Table II

Particle Spectral Characteristics of Three Large Solar-Particle Events of Cycle 19

Date	J_{0p}	P_{0p} (MV)	$J_{0\alpha}$	$P_{0\alpha}$ (MV)	J_{0M}	P_{0M} (MV)
7/14/59	2.6×10^{10}	80	1.99×10^{10}	87	3.32×10^{8}	87
11/12/60	8.9×10^{9}	124	1.94×10^{9}	172	3.23×10^{7}	172
11/15/60	5.9×10^{9}	114	1.92×10^{9}	156	3.2×10^{7}	156

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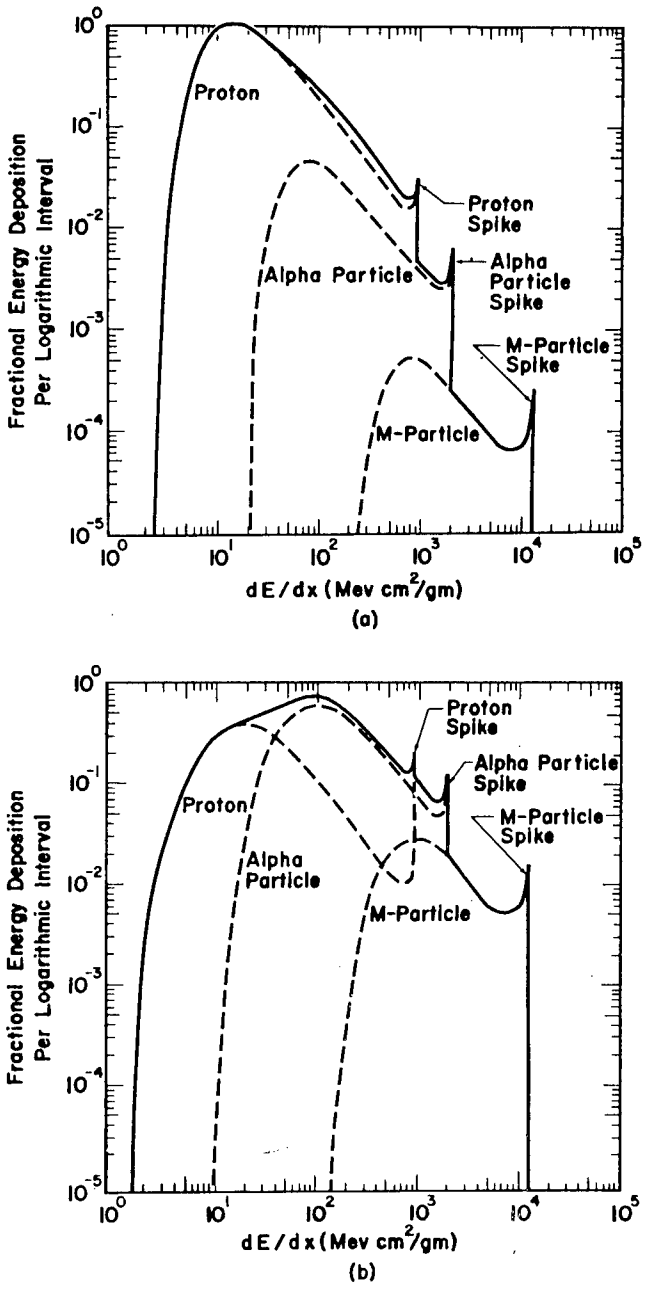

Fig. 1. Two examples of normalized energy-loss distributions under water shielding. (a) Shielding thickness of 5 g/cm^2 for a solar-particle event with $P_{0p} = P_{0\alpha} = P_{0M} = 80$ MV; (b) shielding thickness of 1 g/cm^2 for a solar-particle event with $P_{0p} = P_{0\alpha} = P_{0M} = 180$ MV. In both cases, J_{0p} and $J_{0\alpha}$ equal 60 J_{0M}.

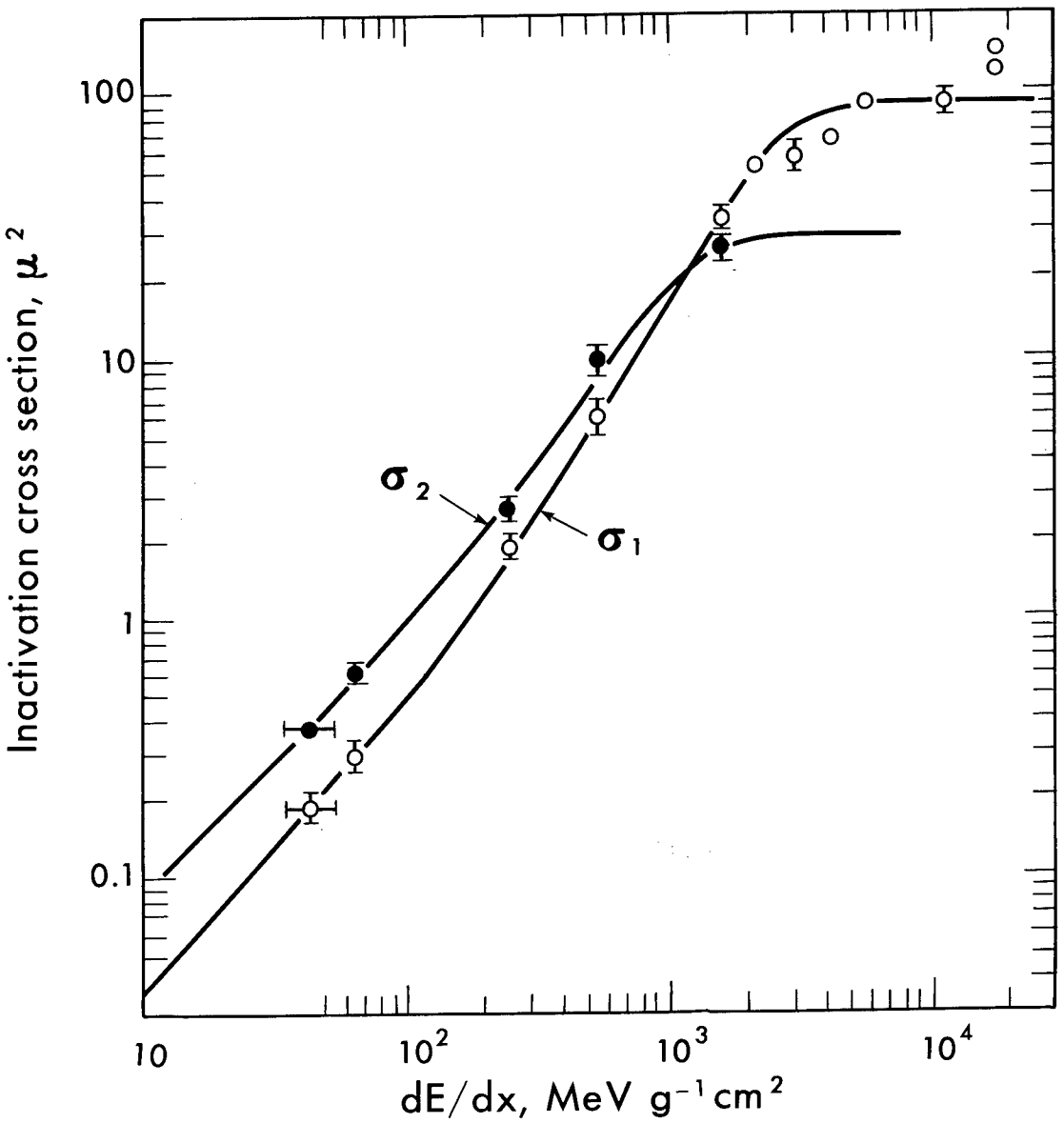

Fig. 2. Inactivation cross sections for irreversible (σ_1) and reversible (σ_2) damage to the proliferative capacity of human kidney cells in vitro as a function of dE/dx, as measured by Todd.[11]

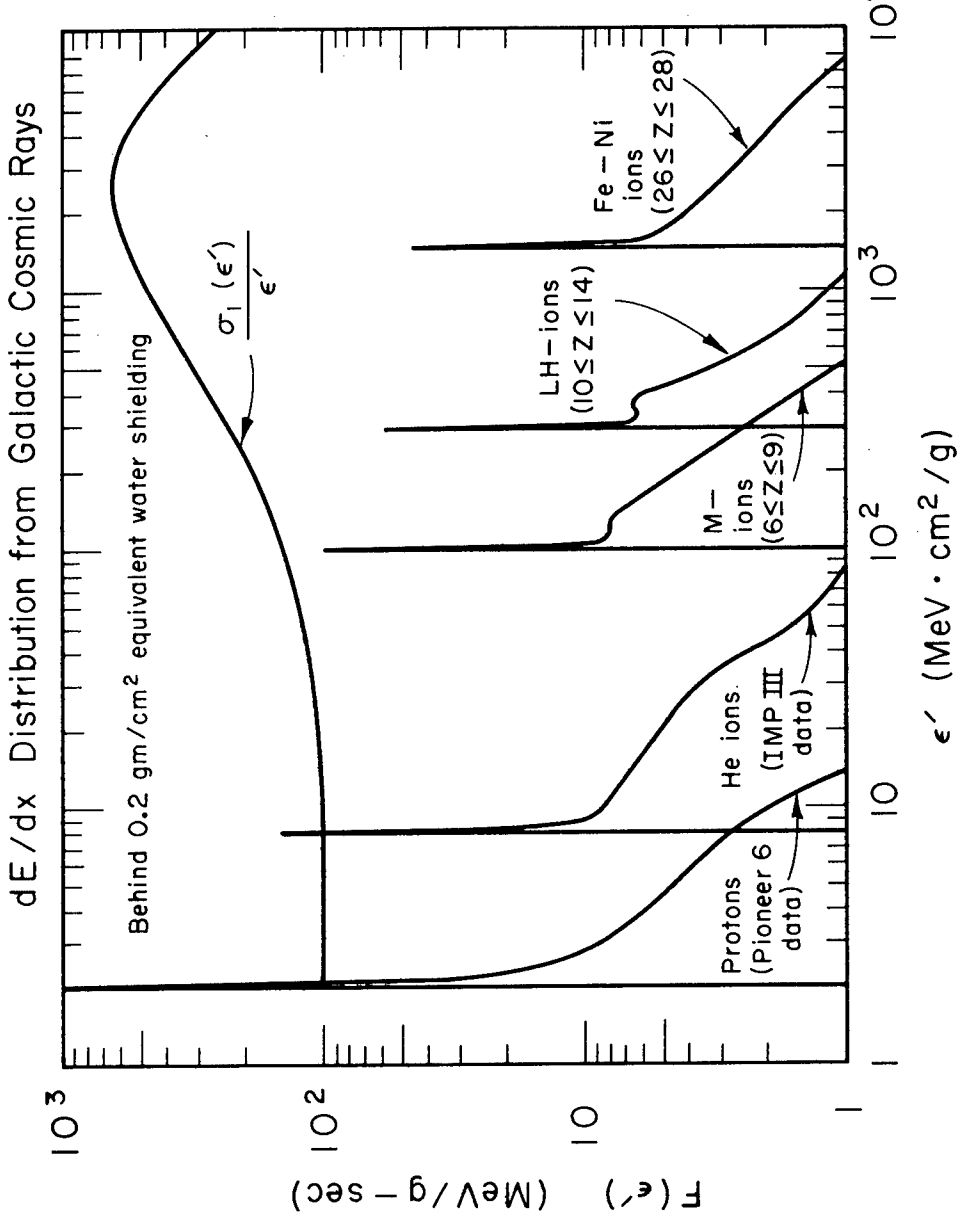

Fig. 3. The energy-loss distribution from galactic cosmic rays under 0.2 g/cm² water shielding. The upper curve gives the weighting factor $\sigma_1(\epsilon')/\epsilon'$ on an arbitrary scale for the lethal hits/site calculation.

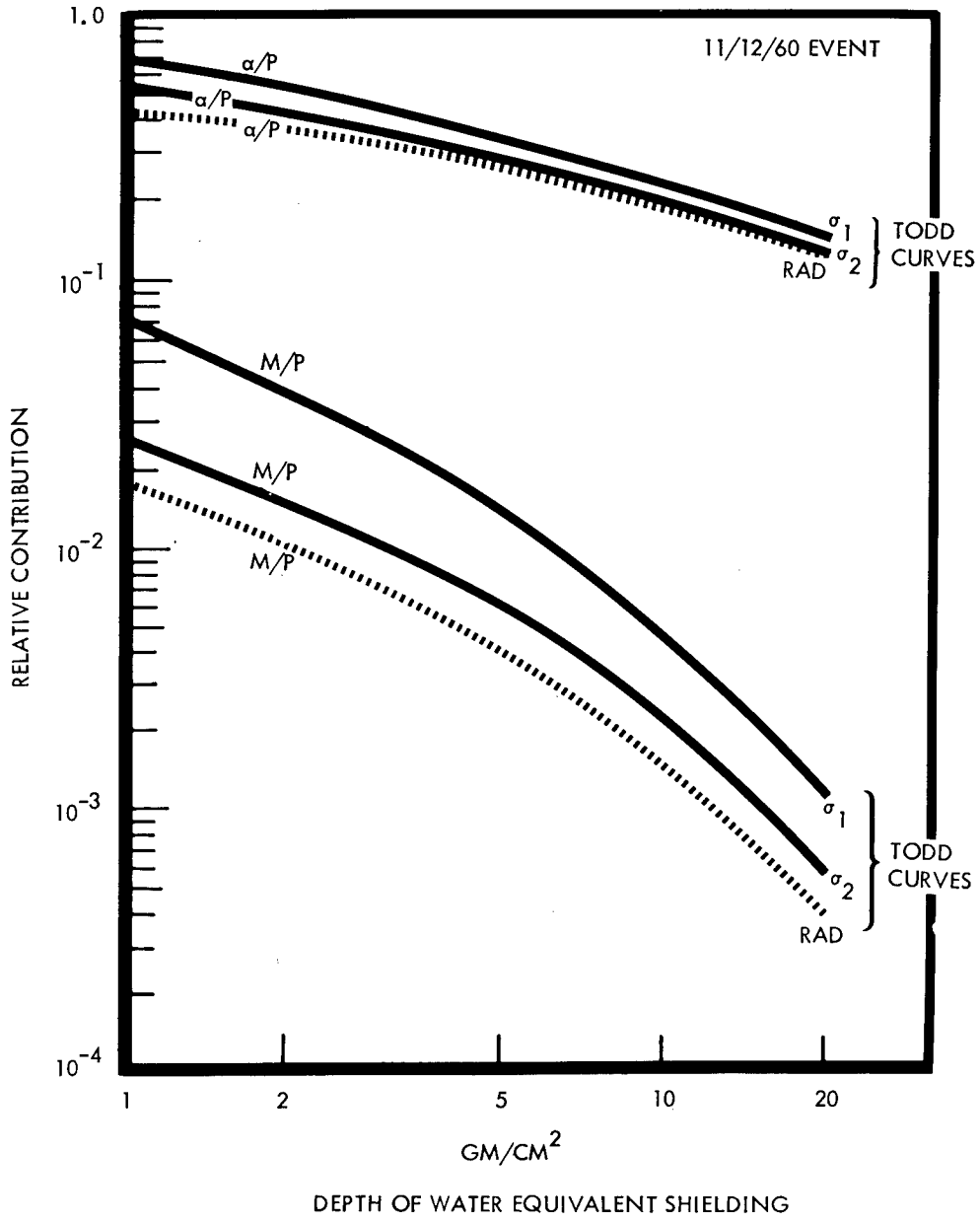

Fig. 4. Contribution of the He ions (α) and M particles (M) to the lethal hits/site and to the rad dose relative to protons (p) as a function of thickness for the solar-particle event of November 12, 1960.

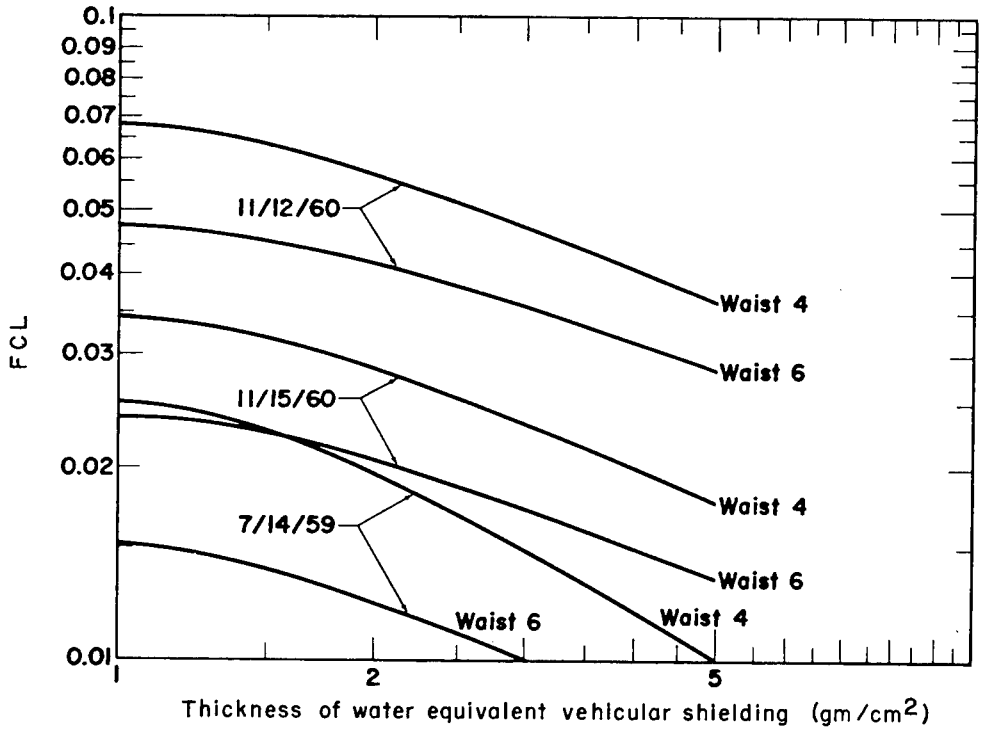

Fig. 5. Fractional cell lethalities (FCL) at two body points in a seated astronaut as a function of vehicular water-equivalent shielding from three solar-particle events.

SESSION SUMMARY

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SESSION SUMMARY

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The most important thematic impression that emerges from having heard these 11 papers is a feeling that the finer complexities inherent in the biology of shielding are being recognized as proper targets for research. During the period 1945 to 1958, when atmospheric testing of nuclear weapons was going on, it gradually became evident that one could not continue to ignore the more difficult physical and biological parameters in radiation environments. At the beginning of that era dose measurement devices were available which effectively integrated dose over 4 steradians. Similarly, the exposed person, in his own way, often integrated dose over 4 steradians. For reasons of convenience it became the practice to hope that the instrument and the man would integrate in the same manner, making it unnecessary for radiation hazard expert to investigate difficult problems such as the angular dependence of flux and energy spectrum. Similarly, during the period 1951 to 1958 it became apparent that biological considerations such as dose rate and LET were more complex than one might have hoped and existing knowledge was less adequate than had been thought for hazard evaluation purposes.

Throughout the session today there was a suggestion that the advent of space radiation hazards has revived many of the same problems and has, in effect, given those concerned a second chance and a new reason for reattacking the more difficult parameters discussed above. Thus we heard explained today techniques for computer representation of the human body and techniques for the conversion of energy loss to cell lethality probability. Also we heard a discussion of the need for a more refined evaluation of depth dose pattern when one is considering sublethal doses.

These are the very questions that were unsolved as of 1958 when the atmospheric testing of nuclear weapons came to a halt. It was most encouraging to note in today's speakers a fresh appreciation of these problems coupled with a renewed determination to work toward their solution. The emergence of this attitude will be, in the long run, of greater importance than the degree of success which happens to have been achieved on any one facet at a given time.