RADIOLOGICAL PROTECTION

A Summary Handbook of ICRP Publications and Recommendations

A. NAGARATNAM

Radiological Protection : A Summary Handbook of ICRP Publications and Recommendations

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FOREWORD

No technology evokes such strong emotions as nuclear energy. The immense possibility of harnessing the power of the atom to produce limitless power, and also to unleash its fury through nuclear weapons that would destroy humankind are really two faces of the same technology. While the world is slowly moving to a better realization of the destructive power of nuclear weapons and evolving agreements to control and finally eliminate them, it is also getting increasingly restive about the safety of nuclear technology. Recent accidents at Three Mile Island and most notably at Chernobyl have shaken our faith on the very applications using nuclear energy. The industrial and healing power of the atom is today seen through a veil of concern and fear. The new Luddites are busy damning any application using nuclear energy. More than ever it is now necessary that we understand the power of radiation as well as its safe limits.

The International Commission on Radiological Protection (ICRP) is actually playing this role, suggesting possibilities, and warning of the dangers from radiation exposure. Such is the expertise and maturity of ICRP that in spite of it being only a recommendatory body, its advice is sought and taken seriously by all concerned, including international organizations like WHO, FAO, ILO and IAEA. The mission of ICRP is to develop a coherent philosophy and a system of radiation protection that primarily takes into account the justification for radiation exposure and works to calculate an appropriate limit of radiation dosage for that exposure. More, it also suggests optimization of the system of protection so that the benefits far exceed possible hazards. The aim is to evolve a dosage as low as reasonably achievable for that objective. This has evolved into the famous ALARA principle.

The scope of ICRP publications covers a wide canvas: philosophy of perception, evaluation and management of risks; understanding of biological and genetic effects of radiation; technical measures to control radiation hazards; engineering aspects of design and operation of equipment; procedures for monitoring safety; accident evaluation and response; organization and management systems and, public awareness. ICRP has enlisted the services of experts from many countries to carry out this vast mandate.

The recommendations of ICRP are regularly published and updated. From 1959 up to now, it has produced 64 publications that run to 9000 pages! How does one wrestle with such an ocean of information? Mr Nagaratnam's Handbook is a response to this challenge. Using the language of ICRP, he communicates clearly and concisely the salient features of the information given in ICRP publications, exercising critical judgement in choosing relevant areas. By all standards, this is a daunting task, and Mr Nagaratnam has responded to it with his characteristic efficiency and insight.

Mr Nagaratnam is the ideal author for a publication of this kind. For over four decades he has worked in areas related to radiation, first as a scientist at the Institute of Nuclear Medicine and Allied Sciences in Delhi, and later as a Director of the Defence Laboratory at Jodhpur where he concerned himself with the industrial applications of radiation. In both these institutions, he has nurtured and sustained competent groups of engineers and scientists working on problems associated with the peaceful applications of nuclear energy. His experiences in working with scientists of the Bhabha Atomic Research Centre and the Atomic Energy Regulatory Board have also contributed to the usefulness of this Handbook.

Defence Scientific Information and Documentation Centre (DESIDOC) of the Defence Research and Development Organization (DRDO) has come forward to publish this volume as a symbol of their continuing commitment to help the scientists and engineers working in this area. I congratulate DESIDOC for this effort and am confident this volume will soon become a ready reference manual to all workers concerned with nuclear radiation not only in India but in other countries as well.

Pittsburgh
July 1994

VS ARUNACHALAM

PREFACE

Mankind is reaping enormous benefits from the peaceful applications of nuclear energy in various fields including power production, medicine, industry, agriculture and research. On the other hand, we know that excessive exposure to ionizing radiations can lead to adverse biological effects. In this respect, radiation is no different from other risks in modern life, since all facets of human advance almost invariably entail some concomitant risks. Wisdom lies in striking a happy and judicious balance in order to ensure that we continue to utilize radiation for human benefit while keeping the potential risk to an acceptable minimum. Radiation should be handled with care, but not fear.

Our knowledge of the biological effects of radiation and potential risks therefrom far exceeds our knowledge of any other hazardous agent, whether in the industrial field, or in the general environment affecting members of the public. The International Commission on Radiological Protection (ICRP) has been playing a pioneering role for decades in this direction. The extensive database that has been established over the decades by the ICRP, the methodologies, techniques and the organizational structures that have been developed to control radiation hazards, and, above all, the philosophy of risk evaluation and management that has been evolved by ICRP, would serve as valuable guides not only to those concerned with radiological protection but to scientists, technologists and administrators involved in all facets of occupational and industrial safety, as well as those concerned with environmental protection.

From 1959 to the end of 1993 ICRP has brought out 64 publications running to around 9000 pages. It is important that everyone connected with the uses of ionizing radiations should be familiar with at least the basic features of the thinking of ICRP as embodied in these publications. The present Handbook attempts to give in a concise, consolidated and codified form the salient features of all the relevant information contained in the voluminous ICRP publications. As far as possible, the language of the original publications themselves has been used.

The suggestion that I take up this assignment came from Dr VS Arunachalam, former Scientific Adviser to the Minister of Defence. I am grateful to him for his constant advice and support, as well as for his kindly agreeing to write the Foreword to this Handbook.

Grateful thanks are due to the ICRP for generously agreeing, in conformity with its policy of encouraging wide dissemination of the information contained in its reports, to using material contained in the relevant ICRP publications for preparing this Handbook. To quote from the communication received from Dr H Smith, Scientific Secretary of ICRP, "On behalf of the International Commission on Radiological Protection I agree to you using the information contained in ICRP Publications 1 to 64 to prepare the monograph. In so doing, however, I stress that this does not necessarily imply that the ICRP will agree to the contents of your monograph or your interpretation of ICRP philosophy".

I would like to express my gratitude to Dr APJ Abdul Kalam, Scientific Adviser to the Minister of Defence, Sri K Santhanam, Chief Adviser on Technologies, Defence Research and Development Organization, Sri KN Singh, Chief Controller, DRDO, Prof P Rama Rao, former Director and Distinguished Scientist, and Sri SLN Acharyulu, the present Director, Defence Metallurgical Research Laboratory, Hyderabad, for their administrative and technical support. Thanks are also due to Sri R Seshadri, Chief Construction Engineer (R&D), Secunderabad; Col S Vasudevan, Director of Estates (R&D), Secunderabad; and Lt Col (Retd) ER Sadasivan, Project Manager, Office of DOE (R&D), Secunderabad for their administrative help. Special mention must be made of Dr SS Murthy, Director, Sri A Lakshmana Moorthy, Scientist and their colleagues at the Defence Scientific Information and Documentation Centre, Delhi, for their untiring efforts in bringing out this publication in an excellent form, and of Sri DS Sastry for his meticulous and fine editing of the manuscript. The help extended by Dr AR Reddy, Director, and the Design Drawing Staff of the Defence Laboratory, Jodhpur in the preparation of several tables and figures is gratefully acknowledged. Dr SC Jain, Scientist from the Institute of Nuclear Medicine and Allied Sciences, Delhi was of great help in several ways during the preparation of the Handbook. It is a pleasure to record my appreciation of and gratitude to my wife Sarasvati for her support in innumerable ways.

Several publishers have given permission for reproducing many tables and figures. Detailed acknowledgments are given separately.

The order of presentation followed in this Handbook does not follow the chronological order in which the publications have come out. The

material has been presented in 7 parts, each dealing with one major aspect of the recommendations, and summarizing the various publications connected with it. A separate note following the Preface gives a brief summary of the way the contents of the Handbook have been arranged.

Subjective judgement had necessarily to be exercised in the choice and emphasis of the subject matter. The author hopes that no important aspect has been left out. The part dealing with radiological protection in the medical applications of radiation has deliberately been made rather exhaustive. Very few medical colleges or leading hospitals in our country seem to have access to ICRP publications. Since it is well known that medical exposure contributes the largest share to population exposure from man-made radiation sources, and since significant reductions in exposure levels, particularly in diagnostic radiology, can be effected by the utilization of simple and cost-effective protection measures without reduction in the diagnostic information of interest, it was considered worthwhile to deal with this subject somewhat elaborately.

ICRP periodically reviews and updates its database in the light of recent information, particularly in the field of biological effects of radiation. The Commission also regularly revises its reports and recommendations, incorporating newer thinking; this is a continuing exercise. Among the immediate tasks ICRP is currently engaged in are revision of earlier reports to bring them into conformity with the latest main (1990) recommendations, revision of several dosimetric models including those of the respiratory tract, Reference Man, embryo and fetus, and radon exposures, as well as extending the age-dependent dosimetric computations to more radionuclides for exposure of members of the public from internally incorporated radionuclides. In the present Handbook, the author had necessarily to content himself with summarizing reports that were brought out by ICRP till the end of 1993.

Hyderabad
20 June 1994

A. NAGARATNAM

SUMMARY OF THE CONTENTS OF THE HANDBOOK

Part I is an extended summary of the latest main recommendations, as brought out in the text of ICRP Publication 60 (1991), using the language of the original publication itself to the extent possible. The order of presentation is the same as that of ICRP 60. Starting from a brief history of the ICRP and the development of ICRP recommendations (Chapter 1), a summary is next given (Chapter 2) of the quantities and units in radiological protection. Chapter 3 summarizes current knowledge on the biological effects of radiation. Chapter 4 deals with the conceptual framework of radiological protection, an area in which ICRP has thoughtfully developed a unique, logical and comprehensive system. Chapter 5 deals with the application of the system of protection for proposed and continuing practices. The basis for the currently recommended numerical values for the dose limits is explained. Chapter 6 is concerned with the application of the system in the case of intervention (following emergencies and accidents). Chapter 7 deals with the implementation of the recommendations, with special reference to the organizational aspects. Chapter 8 is a summary (as given by the ICRP itself) of the recommendations.

Appendix 5.1 deals with the conceptual framework of protection from potential exposures (for which there is a possibility but no certainty of occurrence). Appendix 8.1 (written by the author) gives in somewhat greater detail than in chapter 1 the history and organization of ICRP. Appendix 8.2 (also written by the author) gives a fairly detailed account of the historical evolution of the recommendations. Appendix 8.3 is an updated version of the list of ICRP publications given in ICRP 60.

Part II deals with the biological effects of radiation. It starts (Chapter 9) with a discussion of basic radiobiology at the cellular level. Chapter 10 discusses deterministic effects in various tissues and organs. Chapters 11 and 12 are concerned respectively with the two main types of stochastic effects, viz. induction of cancer and hereditary effects. The sources from which our knowledge of biological effects of radiation is derived, including human experience and results of animal experiments, are discussed. Numerical values of risk estimates are given, and the margin of uncertainty in these figures is indicated. To give a sense of perspective, Appendix

12.1 tabulates data (taken from the 1988 Report of the United Nations Scientific Committee on the Effects of Atomic Radiation) on the current levels of doses from radiation sources, natural and man-made. It is natural that great interest is being evinced on the effects of radiation on the embryo and fetus, and Chapter 13 is devoted to this aspect. The main concern today in radiological protection is in relation to the effects of low dose exposure. The subject is beset with difficulties and uncertainties, and Chapter 14 summarizes the current situation. Chapter 15 discusses the biological effects of inhaled radionuclides, a subject of relevance in certain important stages in the operation of the nuclear fuel cycle.

Part III deals with external and internal dosimetry, a topic whose importance in radiological protection hardly needs emphasis. Chapter 16 deals with quantities used in radiological protection, and is an elaboration and extension of Chapter 2. Considerable effort has gone into the development of the 'Reference Man', with well-defined anatomical, physiological and metabolic characteristics, for standardization of dosimetric protocols; Chapter 17 gives a fairly exhaustive account of the characteristics of the Reference Man. Chapters 18 to 20 discuss in detail the mathematical methodologies for internal dosimetry. Chapters 21 to 24 give details of the dosimetric models (lung, GI tract, bone, and submersion in a radioactive cloud). Chapters 25 and 26 give numerical values of internal dosimetric data for important radionuclides, for occupational workers and members of the public respectively. The impact, particularly on the public, of radionuclide releases into the environment is an important area, but subject to many difficulties in assessment. Chapter 27 deals briefly with the models developed by ICRP appropriate for such assessment. Chapter 28 is devoted to external dosimetry.

ICRP has evolved a realistic system of operational radiation protection in various applications for effective implementation of its recommendations; this forms the subject matter of Part IV. The general principles of monitoring, and monitoring programmes for protection of the workers and members of the public, both under normal conditions of operation and in the event of a major accident, are discussed in Chapters 29, 30 and 32. Chapters 31 and 33 discuss the procedures for handling accidents and emergencies, with respect to protection of the workers and members of the population respectively. ICRP has recently been concerning itself with principles of limiting exposures of the public to natural sources of radiation, and Chapter 34 summarizes the relevant guidelines. With the increasing use of devices and techniques involving ionizing radiation for the teaching of science (such as X-rays and radiotracers), ICRP has given guidelines for protection of the students which are summarized in Chapter 35. Chapter 36 deals

with the principles for the disposal of solid radioactive waste; such practices involve probabilistic assessments of events not only in the present but even in the far distant future.

In view of its historic association with the International Congress of Radiology, as well as the great relevance of radiological protection in medical applications (diagnostic radiology contributes the overwhelming share to population exposure from man-made sources of radiation), ICRP has devoted special attention to this area and given detailed recommendations on specific applications including diagnostic radiology, radiotherapy, nuclear medicine, and biomedical research involving radiation exposure. Part V (Chapters 37 to 41) discusses these aspects. (Appendix 37.1 gives the ICRP guidelines regarding radiological protection in biomedical research.)

Over the last two decades or so we have come to realize that exposure from the daughter products of radon and thoron forms the major proportion of exposure of every member of the human population from natural sources. Doses from this source are highly variable, depending upon geological factors and life styles, and can be very high to people living in certain geographic areas. Part VI (Chapter 42) deals with this subject, including guidelines for limiting exposures of the public from excessive exposure to radon and its daughters inside buildings.

A noteworthy feature of ICRP recommendations is the deep thought that has been given to the problem of risk evaluation and acceptance, as well as to the problem of judicious allocation of resources to radiological protection in the overall context of peaceful applications of nuclear energy in various fields. Part VII deals with these subjects. ICRP has developed elegant methodologies for optimizing and decision-making in radiological protection. These are not merely confined to broad generalities but appropriate techniques have been developed for practical application in specific cases. This is dealt with in Chapter 43. One of the difficult problems that ICRP has tackled is the attempt to evolve a quantitative basis for comparing risks of different kinds, to arrive at a generally applicable parameter for risk expression, as well as to evolve a consensus on what is a level of 'acceptable risk', whether for an occupational worker or a member of the public; these tasks involve not merely scientific but societal value judgements on the perception and acceptance of risk. It is only by a careful consideration of all these factors that it would be possible to arrive at dose limits for radiological protection implying acceptable levels of risk. These questions are discussed in Chapters 44 and 45.

It may be mentioned that in the list of references at the end of each chapter, those pertaining to the different ICRP publications (which have

been referred to in the individual chapters in the text) have not been included, since Appendix 8.3 gives the list of all ICRP publications to-date.

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Springer-Verlag GmbH, Germany, for the table 'Summary of the Weibel model of the tracheo-bronchial region' from Weibel, Ewald R. (1963). *Morphometry of Human Lung*;

University of Minnesota Press, USA, for the figure 'Four general post-natal growth patterns according to age' from Harris, J.A., Jackson, C.M., Patterson, D.G. and Scammon, R.E. (Eds) (1930). *The Measurement of Man*. Copyright 1930, University of Minnesota Press, Minneapolis; and

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(a) Table 'Skin doses in primary beam in diagnostic radiology (median values in cGy per examination)' from UNSCEAR (1979). *Sources and Effects of Ionizing Radiation*; (b) table 'Estimation of approximate thresholds for clinically detrimental non-stochastic effects in various tissues based on response of patients in conventionally fractionated therapeutic X or gamma radiation' from UNSCEAR (1982). *Sources and Biological Effects*; (c) table 'Main characteristics of the A-bomb survivor, ankylosing spondylitis, and cervical cancer series' from UNSCEAR (1988). *Sources, Effects and Risks of Ionizing Radiation*; (d) table 'Estimates of genetic risk arrived at by UNSCEAR in its 1988 report using the direct method: low LET low dose-rate (chronic irradiation conditions)' from UNSCEAR (1988). *Sources, Effects and Risks of Ionizing Radiation*; and (e) author's appendix containing two tables on 'Average doses to the world population from natural and man-made sources of radiation' from UNSCEAR (1988). *Sources, Effects and Risks of Ionizing Radiation*.

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Part I

Main Recommendations of ICRP

(1990 Recommendations, ICRP Publication 60)

CHAPTER 1

INTRODUCTION

Ionizing radiation is the term used to describe the transfer of energy through space in the form of either electromagnetic fields or subatomic particles that are capable of causing ionization in matter. Ionization is the process by which atoms lose, or sometimes gain, electrons and thus become electrically charged, being then known as ions. When ionizing radiation passes through matter, energy is imparted to the matter as ions are formed.

Ionizing radiations and radioactive materials have always been features of our environment, but, owing to their lack of impact on our senses, we have been aware of them only since the end of the 19th century. Since that time, we have found many important uses for them and have developed new technological processes which create them, either deliberately or as unwanted side effects. The primary aim of radiological protection is to provide an appropriate standard of protection for man from the harmful biological effects of these radiations without unduly limiting the beneficial practices giving rise to radiation exposure. This aim cannot be achieved by the use of science alone. All those concerned with radiological protection have to make value judgements about the relative importance of different kinds of risk and about the balancing of risks and benefits. In this they are no different from those working in other fields concerned with the control of hazards. The International Commission on Radiological Protection (ICRP) concerns itself with the formulation of appropriate recommendations in matters of radiation protection.

1.1 THE HISTORY OF ICRP

The International X-ray and Radium Protection Commission was established in 1928, following a decision by the Second International Congress of Radiology. In 1950 it was restructured and acquired its present name of International Commission on Radiological Protection. Over the years, ICRP, while still retaining a relationship with the International Congress of Radiology, has greatly broadened its interests to take account of the increasing uses of ionizing radiation and of practices that involve the generation of radiation and radioactive materials.

ICRP works closely with its sister body, the International Commission on Radiation Units and Measurements (ICRU), and has links with the World Health Organization, the International Atomic Energy Agency, and other United Nations bodies like the United Nations Scientific Committee on the Effects of Atomic Radiation, the United Nations Environment Programme, the International Labour Organization, the International Standards Organization, the International Electrotechnical Commission, and the International Radiation Protection Association.

ICRP recommendations are confined to protection against only ionizing radiations. (ICRP considers that non-ionizing radiation is a subject outside its own field of competence.) ICRP emphasizes that ionizing radiation needs to be treated with care rather than fear and that its risks should be kept in perspective with other risks. The procedures available to control exposures to ionizing radiation are sufficient, if used properly, to ensure that radiation remains a minor component of the spectrum of risks to which all are exposed.

ICRP believes that the standard of environmental control to protect man will ensure that other species are not put to risk. Occasionally, individual members of non-human species might be harmed, but not to the extent of endangering whole species. ICRP concerns itself with mankind's environment only with regard to the transfer of radionuclides through the environment, since this directly affects the radiological protection of man.

ICRP issued its first report in 1928. The first report in the current series, Publication 1 (1959), contained the recommendations approved in 1958. Subsequent general recommendations have appeared as Publication 6 (1964), Publication 9 (1966), and Publication 26 (1977). Publication 26 was amended and extended by Statements in 1978, 1980, 1983, 1984, 1985 and 1987. Reports on more specialized topics have appeared as intermediate and subsequent publication numbers.

A somewhat more detailed account of the history and organization of ICRP is given in Appendix 8.1.

1.2 THE DEVELOPMENT OF ICRP RECOMMENDATIONS

The method of working of ICRP has not changed greatly over the last few decades. Since there is little direct evidence of harm at levels of annual dose at or below the limits recommended by it, a good deal of scientific judgement is required in predicting the probability of harm resulting from low doses of radiation from the observed data obtained at higher

doses and usually at high dose rates. ICRP's aim is to reach a consensus about the outcome of radiation exposures; it has not used either the most pessimistic or the most optimistic interpretation of the available data, but has aimed at using estimates that are not likely to underestimate the consequences of the exposures. The estimation of these consequences and their implications necessarily involves social and economic judgements as well as scientific judgements in a wide range of disciplines.

ICRP recommendations mainly provide guidance on the fundamental principles on which radiological protection can be based. Because of the differing conditions that prevail in various countries, the degree of detail has deliberately been restricted. However, because of its historical links with medical radiology, ICRP's advice in this area has often been more detailed. Local authorities will need to develop their own structures of legislation, regulation, licenses, and codes of practice designed to be broadly consistent with the recommendations. In fact, the recommendations have been widely used in the past and helped to provide a consistent basis for national and regulatory standards. ICRP has been concerned to maintain 'stability' in its recommendations. Frequent changes would only introduce confusion. However, ICRP reviews the newly published data annually. It is not likely that dramatic changes in the recommendations would be necessary, but, if required, ICRP would react rapidly.

Over the last three decades or so, there has been a significant change in the emphasis in the presentation and application of the system of protection recommended by ICRP. Initially, and into the 1950s, there was a tendency to regard compliance with the limits on individual doses as being a measure of satisfactory achievement. The advice that all exposures should be kept as low as possible was noted, but not often applied consciously. Since then, much more emphasis has been put on the over-riding requirement to keep all exposures "as low as reasonably achievable, economic and social factors being taken into account". This emphasis has resulted in substantial decreases in individual doses and has greatly reduced the number of situations in which the dose limit plays a major role in the overall system of protection. It has also changed the purposes of the dose limits recommended by ICRP. Initially, their main function was the avoidance of directly observable injuries. Later, they were also intended to limit the incidence of malignancies and hereditary effects caused by radiation. Although, over the years, the limits have been expressed in a variety of ways, in broad terms, the annual limit for occupational exposure of the whole body was reduced by a factor of about 3 between 1934 and 1950, and by a further factor of 3 by 1958. [Author's note: The present recommendations, in effect, reduce the annual limit by a further factor of 2.5, to the equivalent of 20 millisievert (mSv).]

1.3 FORM OF ICRP PUBLICATION 60

ICRP publication 60 has set out the recommendations in the form of a main text supported by more detailed annexes. The main text contains all the recommendations, together with sufficient explanatory material to make clear the underlying reasoning. It is intended to be used by those concerned with policy, who can turn to the supporting annexes for more detailed information.

Chapters 2 and 3 deal with the physical quantities and units used in radiological protection and with the biological effects of radiation. Chapter 4 describes the conceptual framework of radiological protection and leads on to Chapters 5 and 6 which deal with the main recommendations. Chapter 7 discusses the practical implementation of the recommendations. Finally, there is a summary of the recommendations.

There are four annexes to ICRP 60, viz. (A) Quantities used in radiological protection; (B) Biological effects of ionizing radiation; (C) Bases for judging the significance of the effects of radiation; and (D) A list of ICRP publications.

In the present Handbook, the Chapters 1-8 in Part I follow the pattern given in the main text of ICRP 60, and are basically only a slight condensation of the material in the original text, generally using the phraseology of the text itself. There are three appendices to Part I (appendices 8.1, 8.2 and 8.3), dealing respectively with (i) History and organization of ICRP, (ii) Evolution of ICRP recommendations, and (iii) List of ICRP publications. Appendices 8.1 and 8.2 are not from the original publications, but are notes prepared by the author.

CHAPTER 2

QUANTITIES USED IN RADIOLOGICAL PROTECTION (SUMMARY)

2.1 INTRODUCTION

This chapter explains in simple terms the principal quantities used in radiological protection. The formal definitions and more detailed information are given in Chapter 16. The International System of Units (SI) is used.

Historically, the quantities used to measure the 'amount' of ionizing radiation have been based on the gross number of ionizing events in a defined situation or on the amount of energy deposited in a defined mass of material. These approaches ignore the discontinuous nature of the process of ionization, but are justified empirically by the observation that the gross quantities (with adjustments for different types of radiation) correlate fairly well with the resulting biological effects.

Future developments may show that it would be better to use other quantities based on the statistical distribution of events in a small volume of material corresponding to the dimensions of biological entities such as the nucleus of the cell or its DNA. Meanwhile, however, ICRP continues to recommend the use of macroscopic quantities (known as 'dosimetric quantities').

Before discussing dosimetric quantities, it is necessary to have some information on the biological effects of radiation (described in Chapter 3). The process of ionization changes atoms and molecules, at least transiently, and may thus sometimes damage cells. If cellular damage does occur, and is not adequately repaired, it may prevent the cell from surviving or reproducing; or it may result in a viable but modified cell. The two outcomes have profoundly different implications for the organism as a whole.

Most organs and tissues of the body are unaffected by the loss of even substantial numbers of cells, but if the number lost is large enough, there will be observable harm reflecting a loss of tissue function. The probability of causing harm will be zero at small doses, but above some level of dose

(the 'threshold') it will increase steeply to unity (100%). Above the threshold, the severity of the harm will also increase with the dose. This type of effect is called 'deterministic' (and was previously called 'non-stochastic').

The outcome is very different if the irradiated cell is modified rather than killed. Despite the existence of highly effective defence mechanisms, the clone of cells resulting from the reproduction of a modified but viable somatic cell may result, after a prolonged and variable delay called the 'latency period', in the manifestation of a malignant condition - a cancer. The probability of a cancer resulting from radiation increases with increments of dose, probably with no threshold, and in a way that is roughly proportional to dose, at least for doses well below the thresholds for deterministic effects. The severity of the cancer is not affected by the dose. This kind of effect is called 'stochastic', meaning 'of a random or statistical nature'. If the damage occurs in a cell whose function is to transmit genetic information to later generations, any resulting effects, which may be of many kinds and severity, are expressed in the progeny of the exposed person. This type of stochastic effect is called 'hereditary'.

2.2 BASIC DOSIMETRIC QUANTITIES

The fundamental dosimetric quantity in radiological protection is the 'absorbed dose', D. This is the energy absorbed per unit mass and its unit is joule per kilogram, which is given the special name 'gray' (Gy).

(Author's note: The earlier name for the unit of absorbed dose was the 'rad'. One rad corresponds to an energy absorption of 100 ergs per gram. 1Gy = 100 rad.)

Absorbed dose is defined in terms that allow it to be specified at a point, but it is used in this report to mean the average dose over a tissue or organ. The use of the average dose as an indicator of the probability of subsequent stochastic effects depends on the linearity of the relationship between the probability of inducing an effect and the dose (the dose-response relation) - a reasonable approximation over a limited range of dose. The dose-response relation is not linear for deterministic effects.

2.2.1 Radiation Weighting Factors

The probability of stochastic effects depends not only on the absorbed dose but also on the type and energy of the radiation. This is taken into account by weighting the absorbed dose by a factor related to the quality

of the radiation. In radiological protection, it is the absorbed dose averaged over a tissue or organ and weighted for the radiation quality that is of interest. This weighting factor is called the 'radiation weighting factor', W_R, and is selected for the type and energy of the radiation incident on the body, or, in the case of sources within the body, the radiation emitted by the source (Table 2.1).

2.2.2 Equivalent Dose

This weighted absorbed dose is called the 'equivalent dose' in a tissue or organ, H_T. The equivalent dose in tissue T is given by the expression

$$H_T = \sum_R W_R \cdot D_{T,R}$$

where $D_{T,R}$ is the absorbed dose over the tissue or organ T, due to radiation R. The unit of equivalent dose is joule per kilogram with the special name 'sievert' (Sv).

(Author's note: The equivalent dose was earlier called the 'dose equivalent', and was obtained by weighting the absorbed dose at a point - rather than averaged over an organ or tissue - by the 'quality factor', Q. The earlier unit for the dose equivalent was the 'rem': 1 Sv = 100 rem.)

The value of the radiation weighting factor for a specified type and energy of radiation has been selected to be representative of values of the 'relative biological effectiveness' (RBE) of that radiation in inducing stochastic effects at low doses. The RBE of one radiation compared with another is the inverse ratio of the absorbed doses producing the same degree of a defined biological end-point. The values of W_R are broadly compatible with the values of Q which are related to the quantity 'linear energy transfer' (LET), a measure of the density of ionization along the track of an ionizing particle. ICRP has chosen a value of unity for all radiations of low LET, including X- and gamma radiations of all energies. The choice for other radiations is based on observed values of RBE, regardless of whether the reference radiation is X- or gamma radiation.

When the radiation field is composed of types and energies with different values of W_R, the absorbed dose must be subdivided into blocks, each with its own value of W_R, and summed up to give the total equivalent dose. (Alternatively, it may be expressed as a continuous distribution of energy where each element of absorbed dose from the energy element between E and $E + dE$ is multiplied by the value of W_R from the relevant block in Table 2.1, or as described in the relevant section of Chapter 16).

2.2.3 Tissue Weighting Factors and Effective Dose

The relationship between the probability of stochastic effects and equivalent dose also depends on the organ or tissue irradiated. A further quantity, derived from the equivalent dose, is therefore defined to indicate the combination of different doses to different tissues in a way which is likely to correlate well with the total of stochastic effects. The factor by which the equivalent dose in a tissue or organ T is weighted is called the 'tissue weighting factor', W_T which represents the relative contribution of that organ or tissue to the total detriment due to these effects resulting from uniform irradiation of the whole body (as explained in Chapter 3). The weighted equivalent dose (a doubly weighted absorbed dose) is called the 'effective dose', E (previously called the 'effective dose equivalent'). Its unit is joule per kilogram, with the special name sievert. Recommended values for the tissue weighting factors are given in Table 2.2.

The effective dose is the sum of the weighted equivalent doses in all the tissues and organs of the body. It is given by the expression

$$E = \sum_T W_T . H_T$$

where H_T is the equivalent dose in tissue or organ T and W_T is the weighting factor for tissue T. (It is also the sum of the doubly weighted absorbed doses in all the tissues and organs of the body.)

It is desirable that a uniform equivalent dose over the whole body should give an effective dose numerically equal to that uniform equivalent dose. This is achieved by normalizing the sum of the tissue weighting factors to unity. As a simplistic approximation, it is assumed that W_R is independent of the tissue or organ, and W_T independent of the type and energy of radiation.

The biological effects depend also on the distribution of the dose in time (dose rate and protraction of exposure). Previously, provision was made for possible weighting factors, other than the tissue and radiation weighting factors, by the inclusion of an unspecified weighting factor N; any effect of the time distribution of the dose could have been accommodated by assigning a set of values to N. In practice this has not been attempted, and ICRP has now decided to drop the use of N. (The effect of all exposure conditions other than those dealt with by W_R and W_T can be covered by using different values of the coefficients relating equivalent dose and effective dose to the probability of stochastic effects, rather than by using additional weighting factors.) Further, in this system there is

scope for changing the values for these two weighting factors in the light of newer radiobiological knowledge.

Both equivalent dose and effective dose are quantities intended for use in radiological protection, including the assessment of risks in general terms. They provide a basis for estimating the probability of stochastic effects only for absorbed doses well below the threshold for deterministic effects.

2.3 SUBSIDIARY DOSIMETRIC QUANTITIES

Several subsidiary dosimetric quantities have proved useful. Following an intake into the body of a radioactive material, there is a period during which the material gives rise to doses in the tissues at varying rates. The time integral of the equivalent dose rate is the 'committed equivalent dose', $H_T(\tau)$, where τ is the integration time (in years) following the intake. If τ is not specified, it is implied that the value is 50 years for adults and from intake to age 70 years for children. By extension the 'committed effective dose', $E(\tau)$, is similarly defined.

The dosimetric quantities referred to above all relate to exposure of an individual. Quantities related to exposed groups or populations are also defined. These quantities take account of the number of people exposed to a source by multiplying the average dose to the exposed group by the number of individuals in the group. The relevant quantities are the 'collective equivalent dose', S_T, which relates to a specified tissue or organ, and the 'collective effective dose', S; the unit for these is the man-sievert. The collective quantities can be broadly thought of, with certain limitations, as representing the total consequences of the exposure of a population or group.

The collective effective dose resulting from the presence of radioactive materials in the environment may be accumulated over long periods of time, covering several generations of individuals. The total collective effective dose to be expected from a given situation is the integral over all time of the collective effective dose rate resulting from, i.e. committed by, a single release (or a unit period of practice in the case of a continuing operation). If the integration is not over infinite time, it can be truncated at a definite time. When considering the consequences of a unit period of practice, it is sometimes convenient to distinguish between the collective effective dose already delivered and that committed over all time.

The 'dose commitment' ($H_{c,T}$ or E_c) is a calculational tool and is defined as the infinite time integral of the 'per caput dose rate' ($\dot{H}_T$ or $\dot{E}$) due to a specified event, such as a unit of practice (e.g. a year of practice):

$$H_{c,T} = \int_0^\infty \dot{H}_T(t)dt$$

$$E_c = \int_0^\infty \dot{E}(t)dt$$

In the case of an indefinite practice at a constant rate, the maximum annual per caput dose rate ($\dot{H}_T$ or $\dot{E}$) in the future for the specified population will be equal to the dose commitment of one year of practice, irrespective of changes in population size. If the practice is continued only over a time period τ, the maximum future annual per caput dose will be equal to the corresponding truncated dose commitment, defined as

$$H_{c,T}(\tau) = \int_0^\tau \dot{H}_T(t)dt$$

$$E_c(\tau) = \int_0^\tau \dot{E}(t)dt$$

2.4 OTHER QUANTITIES

Several other quantities are of use in radiological protection. One of these is the 'activity', A, of a quantity of radionuclide. Activity is the average number of spontaneous nuclear transformations taking place per unit time. Its unit is the reciprocal second, s^{-1}, given the special name 'becquerel' (Bq).

(Author's note: The earlier unit of activity was the 'curie' (Ci); 1 Ci = 3.7×10^{10} Bq).

Some operational quantities of interest in the measurement of radiation fields for protection purposes and defined by the ICRU are discussed in Chapter 16.

In relating the probability of stochastic effects to dosimetric quantities, it is convenient to use a probability coefficient. For example, the 'fatality probability coefficient' is the quotient of probability that an increment of dose will cause death and the magnitude of that dose. Such coefficients necessarily relate to a specified population.

It is often useful to use generic terms that can apply to any of the dosimetric quantities. For example, the term 'dose' can be applied to equivalent or effective dose, depending on the context; similarly, the term 'exposure' is used in a generic sense to mean the process of being exposed to radiation.

Table 2.1: Radiation weighting factors[1]
(from ICRP 60)

Type and energy range[2]	Radiation weighting factor, W_R
Photons, all energies	1
Electrons and muons, all energies[3]	1
Neutrons, energy < 10 keV	5
10 to 100 keV	10
> 100 keV to 2 MeV	20
> 2 to 20 MeV	10
> 20 MeV	5
Protons, other than recoil protons, energy > 2 MeV	5
Alpha particles, fission fragments, heavy nuclei	20

1. All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

2. The choice of values for other radiations is discussed in Chapter 16.

3. Excluding Auger electrons emitted from nuclei bound to DNA.

Table 2.2: Tissue weighting factors[1]
(from ICRP 60)

Tissue or organ	Tissue weighting factor, W_T
Gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder[2,3]	0.05

1. The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose they apply to workers, to the whole population, and to either sex.

2. For purposes of calculation, the remainder is composed of the following additional tissues and organs: adrenals, brain, upper large intestine, small intestine, kidney, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other tissues and organs subsequently become identified as having a significant risk of induced cancer, they will then be included either with a specific W_T or in this additional list constituting the remainder. The latter may also include other tissues or organs selectively irradiated.

3. In those exceptional cases in which a single one of the remainder tissues or organs receives an equivalent dose in excess of the highest in any of the 12 organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder as defined above.

CHAPTER 3

BIOLOGICAL ASPECTS OF RADIOLOGICAL PROTECTION

3.1 BIOLOGICAL EFFECTS OF RADIATION

We have already considered briefly in Chapter 2 some aspects of the biological effects of radiation. The process of ionization changes atoms, at least transiently, and may thus alter the structure of the molecules containing them. Molecular changes may also be caused by the excitation of atoms and molecules if the excitation energy exceeds the binding energy between atoms. About half the energy deposited in tissue by ionizing radiation is due to excitation, but this is of less consequence than ionization. If the affected molecules are in a living cell, the cell itself may be damaged, either directly if the molecule is critical to the cell's function, or indirectly by causing chemical changes in adjacent molecules, e.g. the production of free radicals. The most important form of damage is to the DNA; this damage may prevent survival or reproduction of the cell, but frequently the damage is repaired by the cell. If that repair is not perfect, it may result in a viable but modified cell. The occurrence and proliferation of a modified cell may be influenced by other changes in the cell caused either before or after exposure to radiation (e.g. from other carcinogens or mutagens).

If enough cells in an organ or tissue are killed or prevented from reproducing and functioning normally, there will be a loss of organ function - a 'deterministic' effect. The loss of function will become more serious as the number of affected cells is increased. A modified somatic cell may still retain its reproductive capacity and may give rise to a clone of modified cells that may eventually result in a cancer. A modified cell in the gonads, with the function of transmitting genetic information to descendants, may transmit incorrect hereditary information and may cause severe harm to some of those descendants. These somatic and hereditary effects, which may start from a single modified cell, are called stochastic effects. Because of the complex processes involved in the development of the conceptus to an embryo and a fetus, deterministic and stochastic effects on the unborn child are discussed separately.

There is some experimental evidence that radiation can act to stimulate a variety of cellular functions, including proliferation and repair. Such stimulation is not necessarily beneficial. In some circumstances, radiation also appears to enhance immunological responses and to modify the balance of hormones in the body. In particular, radiation may be able to stimulate the repair of prior radiation damage, thus decreasing its consequences, or may be able to improve immunological surveillance, thus strengthening the body's natural defence mechanisms. Most of the experimental data on such effects, currently termed 'hormesis', have been inconclusive, mainly because of statistical difficulties at low doses. Further, many relate to biological endpoints other than cancer or hereditary effects. The available data on hormesis are not sufficient to take them into account in radiological protection.

3.2 THE CONCEPT OF DETRIMENT

In the context of radiation protection, ICRP uses four terms: *change, damage, harm* and *detriment*. Changes may or may not be harmful. Damage represents some degree of deleterious change, for example to cells, but is not necessarily deleterious to the exposed individual. Harm denotes clinically observable deleterious effects that are expressed in individuals (somatic effects) or their descendants (hereditary effects). Detriment is a complex concept, combining the probability, severity and time of expression of harm. It cannot easily be represented as a single variable. ICRP now uses the term 'risk' only descriptively.

Detriment, a concept introduced by ICRP in its 1977 recommendations (ICRP 26, 1977), is a measure of the total harm that would eventually be experienced by an exposed group and its descendants as a result of radiation exposure. It has many components, including health detriment, which is the only type of detriment considered here explicitly. The concept of detriment is useful in many applications, e.g. (a) to assess the consequences of continued or cumulative exposures in order to recommend dose limits; (b) to compare consequences of different distributions of equivalent dose within the body; (c) to provide a basis for assessing the valuation of a unit of collective dose for justification and optimization purposes.

In ICRP 26 (1977) detriment was defined as the mathematical expectation of the number of cases of a radiation-induced health effect weighted by a factor representing the severity of the effect. The weighting factor was taken as unity for death of the individual and for severe hereditary effects in his descendants. Smaller weighting factors were implied (but

not specified) for less severe effects. For the individual, the detriment is the probability of the deleterious effect weighted by its severity.

If the value of the detriment from each of several detriments is small, then these can be added together to give the total detriment; in this particular case it is implicit that the relevant doses are small, well below the thresholds for deterministic effects.

Ideally, detriment should be represented by an extensive quantity, i.e. one that allows the detriment to a group to be added as additional exposures occur to individuals and as more individuals are added to the group. This requirement cannot be met fully at present.

ICRP has concluded that it is undesirable to select a single approach to represent detriment but that a multi-dimensional concept is required. The earlier method of aggregating the different facets of detriment used in ICRP 45 (1985) to derive a single quantity, a 'unified index of harm', is used only to a limited extent in the present approach. (The aggregative method has however been retained for choosing tissue weighting factors, because these are used only to make adjustments for the different sensitivities of tissues; since it is rare for single tissues to be irradiated alone, except for lung, and perhaps for thyroid and skin, the choice of W_T is not very sensitive to the procedure for aggregating the different aspects of detriment.)

3.3 QUANTITATIVE ESTIMATES OF THE CONSEQUENCES OF EXPOSURE

For developing a system of protection, we must know how the probability of stochastic effects and the severity of deterministic effects vary with dose. The most relevant sources of information are those obtained directly from studies of radiation effects on humans. For deterministic effects we have information from side effects of radiotherapy, effects on early radiologists, effects of atomic bombs at Hiroshima and Nagasaki, and from consequences of nuclear accidents. For stochastic effects, we have epidemiological studies on Hiroshima-Nagasaki survivors, patients exposed to radiation for diagnosis and treatment, and workers exposed to radiation or radioactive sources at work. In addition, a great deal of information on mechanisms of radiation injury and dose-response relations has been obtained from studies on micro-organisms, *in vitro* cell cultures and animals; extrapolation of this information to humans is not easy.

3.3.1 Deterministic Effects

In many organs and tissues there is a continuous process of loss and replacement of cells. An increase in the rate of loss (e.g. following radiation exposure) may be compensated for by an increase in the replacement rate, but there will be a transient or permanent reduction in number of cells. If the decrease is large enough, there will be clinically observable pathological conditions such as a loss of tissue function. If the damage to a vital structure is large, the end result will be death. The probability of harm will be zero at small doses up to hundreds or thousands of millisieverts depending on the tissue and increase rapidly to 100% above the 'threshold' dose. The dose-response relation is sigmoid. Above the threshold, the severity of harm will increase with dose (and usually with dose rate, because of less time for repair). This type of effect was earlier called 'non-stochastic' but is now called by ICRP as 'deterministic'. Although the initial cellular changes are essentially random, the large number of cells involved in the initiation of a clinically observable effect gives it a deterministic character.

In addition to loss of cells, there may occur damage to supporting blood vessels leading to secondary tissue damage and replacement of functional cells with fibrous tissue leading to a reduction in organ function. Examples are visual impairment (cataract), temporary or permanent sterility.

For low and moderate damage, some functional deterministic effects are reversible; examples are decrease of glandular secretions (salivary glands, thyroid), neurological effects (changes in EEG or retinograms), and vascular reactions (erythema, subcutaneous oedema).

The radiation weighting factors are normally intended for stochastic effects. For $W_R > 1$, the weighting factors for deterministic effects would be smaller than for stochastic effects and the use of the equivalent dose would over-estimate the predicted deterministic effects for high LET radiations like neutrons.

Few tissues show clinically significant effects following acute doses of a few gray; for chronic exposures serious effects are unlikely at dose rates less than 0.5 Gy/y. However, the gonads, lens of the eye and bone marrow show higher sensitivities. Table 3.1 gives the thresholds for some deterministic effects.

3.4 STOCHASTIC EFFECTS IN EXPOSED INDIVIDUALS

The response of the body to the development of a clone of modified cells is complex. The clone is very likely to be eliminated by the body's defences, but, if not, it may result, after a prolonged and variable delay (latency period), in the development of a malignancy. Radiation-induced cancers are not distinguishable from those occurring from other causes. The defence mechanisms are not likely to be totally effective, so that there may not be a threshold in the dose-response relation. The probability of a cancer will depend on the number of clones of modified cells initially created (since this number will influence the probability of at least one clone surviving) which in turn is related to the dose. The severity is influenced by the type and location of the cancer. The process of radiation carcinogenesis appears to be random, although there may be some individual variation in susceptibility to induction of radiation-induced cancer, reflecting genetic and physiological variations. Some individuals with genetic diseases may differ substantially from the mean.

It seems that no stochastic effects other than cancer (and benign tumours in some organs), and possibly mental retardation following exposure *in utero*, are induced in the exposed individual by low to moderate doses. In particular, any life-shortening found in exposed human populations and in experimental animals after low doses has been due to excess radiation-induced cancer mortality.

Several million ion pairs are created every year in the DNA molecules of a human being by exposure to natural background radiation. Clearly, the process of passing from the creation of an ion pair in DNA to the manifestation of a cancer is very rarely completed. Only one death in four is attributable to cancer even in developed countries and radiation is responsible for only a small fraction of the cancer deaths.

3.4.1 Uncertainties in The Various Studies on Exposure

Epidemiological studies do not provide us the exact information needed. Japanese data are extensive (about 80,000 persons followed up) but the follow-up period is not adequate for all cancers to have appeared; about 60% of the initial survivors are still alive. The population was heterogeneous comprising both sexes and persons of all ages, and the dose spread fairly uniformly over the whole body. Number of excess cancer cases is significant at the 95% level of confidence for doses above 0.2 Sv. Excesses of lower significance are found at doses around 0.05 Sv. The doses were

incurred at very high dose rates, whereas information is needed in radiological protection at very much lower dose rates.

The studies on patients also pose problems. Irradiations are not uniform and the subjects may not be representative of the general population. The studies on workers that have yielded information relate to ^{226}Ra workers in the early years of the present century and to miners (mainly uranium miners) who inhaled radon and its daughters in the middle years of the century. Exposures were protracted, doses localized to bone and lung tissues, and from alpha particles; further, accurate estimates of radioactive intake are difficult. Studies on early radiologists show some stochastic effects, but dose estimation is not easy and quantitative risk estimates have not been possible. Studies on atomic energy workers have yielded some risk estimates, but with very wide confidence limits.

Numerous estimates involving exposure of populations to low doses have been reported. These include exposure to fallout, military personnel exposed to weapon tests, persons in the environment of nuclear plants, fetuses exposed to diagnostic X-rays, other medically irradiated populations, and populations living in high natural background areas (in India, Brazil, Colorado in USA, and China). But they suffer from the following methodological shortcomings: small sample size, lack of adequate controls, and extraneous factors; further, 'positive' findings are reported but negative ones are not. Overall, they have contributed little to quantitative estimates of risk.

In almost all cases (apart from accidents and treatment of patients) doses are incurred over long periods of time and at rates that do not add greatly to the doses from natural background. The annual addition from artificial sources ranges from a small fraction of the annual dose from natural sources to about ten times that annual dose. (The lung is a special case because the equivalent dose from radon daughters is very variable and is sometimes several thousand times the equivalent dose to other parts of the body from natural sources.) The fact that doses from natural sources affect all parts of the body decreases the importance of the dose-response relation at doses close to zero. Small doses are always additions to the natural background dose, and a linear relation between incremental dose and incremental probability of a deleterious effect will be an adequate approximation, whatever the shape of the dose-response relation.

3.4.2 Dose-response Relationship

The simplest dose-response relation is that of a straight line through the origin. The human epidemiological data are not sufficiently precise to confirm or exclude that relationship. However, almost all data relating to stochastic changes in cells *in vitro* and in simple biological organisms as well as induction of animal tumours show curvilinear relationships for low LET radiations, with the slope at low doses being less than at high doses. For low doses and dose rates, it is unlikely that more than one ionizing event will occur in the critical parts of a cell during which repair mechanisms can operate, and the dose-response will be linear. At higher doses and dose rates, two or more events may combine, producing an enhanced effect reflected by a quadratic term in the dose-response relation. At still higher doses, where cell killing becomes important, the slope again decreases. (The results for high LET radiations are more nearly rectilinear over the range of doses below those causing appreciable cell killing.) The above discussion makes it clear that at all levels of dose below the recommended dose limits, ICRP's use of a simple proportional dose-response relation is valid.

There is sufficient evidence to justify making an allowance for non-linearity when interpreting data for low LET radiation at high doses and dose rates to give estimates of the probability of effects at low doses and low dose rates. ICRP has decided to reduce by a factor of 2 the probability coefficients obtained directly from observations at high doses and dose rates. (This value may be arbitrary but conservative.) This reduction factor is called the 'Dose and Dose Rate Effectiveness Factor', DDREF, and has been used for all absorbed doses below 0.2 Gy and from higher absorbed doses when the dose rate is less than 0.1 Gy/h. (A more detailed discussion is given in Chapter 11.)

Another major difficulty in interpreting human data is the number of cancer deaths yet to appear. For some cases like leukaemia in Japanese survivors and bone cancers in patients injected with ^{224}Ra, the rate of appearance has fallen back close to the expected rate in a matched control population. For other cancers, the rate is still enhanced, and in the Japanese study, still rising.

3.4.3 Multiplicative and Additive Risk Projection Models

For most types of cancers, the excess mortality seems, after an initial period of very low risk (the minimum latency period), to have the same

pattern in time as the natural mortality due to the same type of cancer. If this continued throughout life, there will be a simple proportion between the natural cancer mortality and the excess mortality due to radiation for the whole time after the minimum latency period. The lifetime excess can then be obtained by the 'multiplicative risk projection model' by multiplying the observed natural mortality by a factor drawn from observation over only part of life.

For other cancers, after the initial latency period, the rate rises over a period of years after exposure and then remains fairly constant (and independent of the natural mortality), or, as with leukaemia and bone cancer, falls. The 'additive risk projection model' which can be applied to such a pattern predicts an eventual total probability of death of about half the value predicted by the multiplicative model. The additive model is no longer seen to be consistent with most of the epidemiological observations.

The multiplicative model predicts a later mean age of attributable (*i.e.* radiation-induced) death, and thus less time lost per death, than the additive model. The currently predicted loss of life expectancy per unit dose is not sensitive to the choice between the two models.

Because of uncertainties of recording cancer incidence rather than mortality, most of the human data are expressed in terms of excess radiation-induced mortality. However, ICRP wants to take into account cancer incidence also in its evaluation of the harm or detriment.

3.4.4 Nominal Probability Coefficient

The estimated probability of a fatal cancer per unit effective dose is called the 'nominal fatality probability coefficient'. Earlier, only fatal cancer risk coefficients were used without allowing for reduction in that probability resulting from competing causes of death. This correction is essential if the multiplicative model is used, and has been included in ICRP 60. For simplicity, the same coefficients are used for both men and women and for a representative population of wide ranges of ages (although the values would actually be age- and sex-specific). A small difference is, however, introduced between the coefficients for workers (adults above 18) and the general population which includes the more sensitive younger age groups.

ICRP's nominal probability coefficients have been based mainly on Japanese data. The data for irradiated ankylosing spondylitics lead to a lower risk estimate for fatal cancer by a factor of two. For leukaemia the additive model is used and for all other cancers the multiplicative model; this may overestimate the cancer probability at older ages because the multiplying factor may not persist over the whole span of life. The effect of competing causes of death reduces the importance of any such error.

Another question is how to transfer conclusions from the Japanese data to other populations. Either the absolute mortality rate per unit dose can be applied to the other populations or the transfer can be made by using the proportional increase in the mortality rate of each type of cancer in turn. In either case, the mortality pattern of the new population has to be used to allow for competing causes of death. ICRP has averaged over five populations to give a reasonable representation of a typical population.

For high doses and high dose rates of low LET radiations, the lifetime fatality probability coefficient for a representative population of both sexes and of working age comes out to about 8×10^{-2} per Sv for the sum of all malignancies. This value, combined with a DDREF of 2, leads to a nominal probability coefficient for workers of 4×10^{-2} per Sv. The corresponding values for the whole population, including children, are about 10×10^{-2} per Sv for high doses and dose rates and 5×10^{-2} per Sv for low doses and low dose rates (Table 3.2). Typically, the multiplicative model shows a mean loss of life per attributable cancer death of 13-15 years, while the additive model gives a corresponding figure of about 20 years.

Extensive data exist on the relation between bone cancer and radium content of workers in the early luminizing industry, between bone cancer and activity of ^{224}Ra injected in patients, and between lung cancer and exposure to radon and its progeny in mining environments. In all these cases, dosimetry is difficult and it is not easy to arrive at dose-response relations. However, it is known from experiments on cells and animals that, per unit absorbed dose, high LET radiation causes more stochastic effects than low LET radiation.

Values of RBE do not lead directly to values of the radiation weighting factors. The standard radiation type used experimentally is either X-rays of a few hundred keV energy or gamma rays of about 1 MeV. While these radiations are equally effective at high doses and high dose rates, there is a factor of about two in the biological effectiveness between these two energy bands at low doses. For simplicity, ICRP has not distinguished

between the two types and has selected values of radiation weighting factors for other radiations broadly representative of the observed RBE values relative to either X or gamma rays (Table 2.1). The nominal fatality probability coefficients per unit equivalent dose and unit effective dose for high and low radiations are then the same. In the case of lung cancer from inhaled radon, the epidemiological data yield a direct relation between cumulative exposure to radon progeny and the excess probability of lung cancer. In this case it is reasonable to express the attributable risk coefficient per unit of radon exposure and not per unit dose to the lung or the bronchial epithelium. (Radon exposures are discussed in Chapter 42.)

3.4.5 Stochastic Effects in Progeny

If the damage occurs in the germ cells of the gonads, this damage, called 'hereditary' (mutations and chromosomal aberrations), may be transmitted to the descendants of the exposed individual. Radiation has not been identified as a cause of such effects in man but experimental studies on plants and animals suggest that such effects will occur, and it may be presumed that such damage will occur in man also.

Hereditary effects vary widely in their severity. Dominant mutations lead to genetic disease predominantly in the first and second generation offspring; some of them are seriously damaging and life-threatening. Chromosomal aberrations may result in congenital abnormalities in children. Recessive mutations produce little effect in the first few generations but make a contribution to the general pool of genetic harm in subsequent generations. There are also many deleterious conditions (multifactorial disorders) that have a substantial incidence in mankind and for which the normal incidence may be maintained partly by new genetic mutations and partly by environmental factors. In ICRP 26 separate consideration was given to the hereditary effects in the first two generations (of importance to the exposed individual) and all subsequent generations (of interest to society). ICRP 60 attributes the whole detriment to the exposed individual.

For low doses and dose rates, the nominal hereditary effect probability coefficient for serious effects (excluding multifactorial effects) over all generations related to the gonad doses distributed over the whole population is 0.5×10^{-2} per Sv. About 80% of the effects are due to dominant and X-linked mutations. Of these 15% occur in each of the first two generations. The probability coefficient for multifactorial effects, weighted for severity, is probably 0.5×10^{-2} per Sv. Because of the different age distribution of

a working population, the coefficients are slightly smaller than for the general population (by about 40%). ICRP considers that the nominal hereditary effect probability coefficients of 1 x 10^{-2} per Sv for the whole population and 0.6 x 10^{-2} per Sv for workers adequately represent the weighted number of hereditary effects to be expected in all generations. This only includes weighting for severity. With further weighting for years of life lost if the harm occurs (as is explained later), the corresponding numbers will be 1.3 x 10^{-2} and 0.8 x 10^{-2} per Sv (see Tables 3.2 and 3.3).

3.4.6 Effects of Antenatal Exposure

The effects on the conceptus depend on the time of exposure relative to conception. During the first week when the number of cells is small and their nature is not yet specialized, the loss of even a few cells may result in a failure to implant or to an undetectable embryonic death. Any transformation at this stage is also likely to cause the death of the conceptus rather than result in stochastic effects expressed in the liveborn. Exposure of the embryo in the first three weeks following conception is not likely to result in deterministic or stochastic effects in the liveborn child, despite the fact that the central nervous system and the heart are beginning to develop in the third week. During the rest of the period of major organogenesis (from the third week after conception), malformations may be caused in the organ under development at the time of exposure. These effects are deterministic with a threshold in man estimated to be about 0.1 Gy.

Throughout the period from 3 weeks from conception until the end of the pregnancy, there may be stochastic effects resulting in an increased probability (probably a few times that of the population as a whole) of cancer in the liveborn.

There is a general downward shift in the distribution of IQ with increasing dose *in utero*. ICRP assumes that the shift is proportional to dose. A value of about 30 IQ points (30%) per Sv for fetal exposure during the 8-15 week period post-conception is indicated by Japanese experience. A smaller downward shift occurs for exposure during the 16-25 week period. This appears to be a deterministic effect, with a threshold determined only by the minimum shift in IQ that can be clinically recognized.

The second finding is of a dose-related increase in the frequency of 'severely retarded' children. There appears to be an excess probability of 0.4 at 1 Sv. Because of the Gaussian shape of the IQ distribution, this

excess number will be very small at small IQ shifts, rising steeply as the shift approaches 30 IQ points. At doses of the order of 0.1 Sv, no effect would be detectable in the general distribution of IQ. The effects at all levels of dose are less marked following exposure in the 16-25 week period after conception and have not been observed for other periods. These observations relate to high doses and high dose rates and their use might overestimate the risks.

3.5 TISSUE WEIGHTING FACTORS

The tissue weighting factor introduced to define the effective dose is intended to ensure that a weighted tissue equivalent dose would produce broadly the same degree of detriment irrespective of the tissue involved. An aggregated representation of detriment has been adopted for this purpose. It includes four components: the probability of attributable fatal cancer, the weighted probability of attributable non-fatal cancer, the weighted probability of severe hereditary effects, and the relative length of life lost. Since effective dose will be used only over ranges where the probability of attributable death is small, even the contribution of the fatal component to detriment can be treated as additive when several organs are irradiated. Each consequence can then be weighted by a factor chosen to represent its severity. As in ICRP 26, death and severe hereditary effects are both given a weighting factor of 1.

A type of cancer that is difficult to cure, and thus has a high lethality fraction and usually a reduced quality of life for the survivors, would have a high weighting factor for the non-fatal events, while an easily cured cancer would have a low weighting factor for the non-fatal events. The weights would then range from 0.01 for non-fatal skin cancer to 0.99 for non-fatal leukaemia. The weighting factor for the severity of hereditary effects is already included in the probability coefficients.

A second type of weighting factor is applied to take account of the different mean latency time for different types of cancer. This weighting is the relative time lost due to an attributable cancer death, or in the case of non-fatal cancers and hereditary effects, the relative time of impaired life, taken for cancers as the same as the time lost for the same type of cancer. The products of the mortality coefficient and the weighting factors for morbidity and time lost are normalized to give a total of unity and thus provide a basis for the tissue weighting factors. Rounded values of these factors have been given in Table 2.2.

The data in Table 3.3 are representative for a nominal population of equal numbers of men and women. Except for the breast, the difference between the sexes is small.

If the equivalent dose is fairly uniform over the whole body, it is possible to obtain the probability of fatal cancer associated with that effective dose from the nominal fatality probability coefficients. If the distribution is non-uniform, this use of the nominal coefficient will be less accurate because the tissue weighting factors include allowance for non-fatal and hereditary conditions. As an approximation for a wide variety of distribution of equivalent dose, the non-fatal somatic detriment adds 20-30% to the fatal detriment.

Table 3.1: Thresholds for deterministic effects
(based on ICRP 60)

Effect	Acute dose (Gy)	Dose rate for prolonged exposure (Gy/y)
Sterility in male:		
Temporary	0.15	0.4
Permanent	3.5-6	2
Sterility in female:		
Permanent	2.5-6 (older women more sensitive)	> 0.2
Impairment of vision	2-10	> 0.15
(thresholds for high LET radiations 2-3 times lower than above values.)		
Clinically significant depression of blood-forming process	0.5	> 0.4
$LD_{50/60}$ in heterogeneous population	3-5	

Table 3.2: Nominal probability coefficients for stochastic effects
(from ICRP 60)

Exposed population	Detriment (10^{-2} per Sv)[1]			
	Fatal cancer[2]	Non-fatal cancer	Severe hereditary effects	Total
Adult workers	4.0	0.8	0.8	5.6
Whole population	5.0	1.0	1.3	7.3

1. Rounded values

2. For fatal cancer, the detriment coefficient is equal to the probability coefficient

Table 3.3: Nominal probability coeffients for tissues and organs
(for a population of equal numbers of both sexes and a wide range of ages)
(from ICRP 60)

Tissue or organ	Probability of fatal cancer (10^{-2} Sv^{-1})		Aggregated detriment (10^{-2} Sv^{-1})	
	Whole Population	Workers	Whole Population	Workers
Bladder	0.30	0.24	0.29	0.24
Bone marrow	0.50	0.40	1.04	0.83
Bone surface	0.05	0.04	0.07	0.06
Breast	0.20	0.16	0.36	0.29
Colon	0.85	0.68	1.03	0.82
Liver	0.15	0.12	0.16	0.13
Lung	0.85	0.68	0.80	0.64
Oesophagus	0.30	0.24	0.24	0.19
Ovary	0.10	0.08	0.15	0.12
Skin	0.02	0.02	0.04	0.03
Stomach	1.10	0.88	1.00	0.80
Thyroid	0.08	0.06	0.15	0.12
Remainder	0.50	0.40	0.59	0.47
Total	5.00	4.00	5.92	4.74
	Probability of severe hereditary disorders			
Gonads	1.00	0.6	1.33	0.80
Grand total (rounded)			7.3	5.6

CHAPTER 4

THE CONCEPTUAL FRAMEWORK OF RADIOLOGICAL PROTECTION

4.1 INTRODUCTION

This chapter deals with the general policy of radiological protection. It introduces the idea of source-related and individual-related assessments. It outlines the basic system of protection for occupational, medical and public exposures and distinguishes between a 'practice' which causes exposures and 'intervention' which decreases exposures.

4.2 THE BASIC FRAMEWORK

Everyone is exposed to radiation from natural and artificial sources. A realistic system of radiological protection must therefore have a clearly defined scope and cover, in a consistent way, a wide range of circumstances.

The basic framework of radiological protection necessarily has to include social as well as scientific judgements, because the primary aim is to provide an appropriate standard of protection without unduly limiting the beneficial practices giving rise to exposure. Since there are thresholds for deterministic effects, it is possible to avoid them by restricting the doses to individuals. But stochastic effects cannot be completely avoided because no threshold can be invoked for them. The basic framework is intended to prevent the occurrence of deterministic effects and to ensure that all reasonable steps are taken to reduce the induction of stochastic effects.

Most decisions about human activities are based on an implicit form of balancing benefits against costs and disadvantages, leading to the conclusion that a particular course of action or practice is, or is not, worthwhile. Further, the conduct of a practice should be adjusted to maximize the net benefit to the individual or to society. In radiological protection, as in other areas, it is becoming possible to formulate and quantify proce-

dures that help in reaching decisions. When the benefits and detriments do not have the same distribution throughout the population, there is bound to be some inequity; this can be minimized by attention being given to the protection of individuals. Further, many current practices give rise to doses that will be received in the future, and these should also be taken into account in the system of protection. Current practices may also give rise to a probability, but not a certainty, that exposures will occur. The probability of incurring the exposures is then important, in addition to the magnitude of the exposures.

It is convenient to think of the processes causing exposures as a network of 'events' and 'situations'. Each part of the network starts from a 'source' (not necessarily a physical source of radiation). For example, when radioactive materials are released into the environment as waste, the installation as a whole may be regarded as the source. Radioactive materials then pass through simple or complex environmental pathways, eventually leading to exposure of individuals. Individuals may be exposed to radiation from more than one source.

It is rarely necessary to treat this network as a single entity. Provided that the individual doses are well below the threshold for deterministic effects, the contribution to an individual dose from a single source has an effect that is independent of the doses from other sources, and each source can then be treated on its own. Assessments of the effectiveness of protection can be related to the source giving rise to the individual doses ('source-related') or related to the individual dose received by a person from all the relevant sources ('individual-related').

Source-related assessments make it possible to judge whether a source is likely to bring benefits sufficient to outweigh any disadvantages that it may have, and whether steps have been taken to reduce the exposure that it will cause. The source-related assessment will take account of the magnitude and the probability of occurrence of individual doses attributable to that source, and of the number of individuals so exposed, but will not consider the additional contributions from other sources. We have therefore also to consider an individual-related assessment of the total doses from all the relevant sources, in order to determine whether any individual has too high a probability of stochastic effects and whether any individual dose approaches one of the thresholds for deterministic effects.

Activities that increase the overall exposure to radiation are called by ICRP as 'practices'. 'Intervention' is the term applied to those activities that decrease the overall exposure.

Steps to control individual exposure can be taken at any point in the network linking the sources to the individuals. The action may be applied to the source, the environment, or the individual. Actions applied at the source will be preferable since they will be most effective and least disruptive (unless they fail as the result of an accident). Actions applied to the environment or to individuals are more obtrusive and may have social disadvantages, not all of which are foreseeable.

ICRP's system of protection is intended to be as general as possible, partly for consistency and partly to avoid changes of policy resulting from the demarcation of different situations.

ICRP uses a division into three types of exposure: *occupational exposure*, which is the exposure incurred at work; *medical exposure*, which is the exposure of persons as part of their diagnosis or treatment; and *public exposure*, which comprises all other exposures.

In the control of occupational exposure, it is usually possible to apply controls at all three points - at the source, by fixing its characteristics, shielding and containment; in the environment, by ventilation or additional shielding; and at the individual, by requiring working practices and the use of protective clothing and equipment. In medical exposures, controls are also applied at all three points, mainly as part of the primary function of diagnosis or treatment. In public exposure, the controls should normally be applied at the source.

In the case of a new practice, there is the option of accepting the practice, if necessary with modifications, or rejecting it. Existing practices can be reviewed and can be modified; but even if withdrawn, the sources and pathways that they involve may persist. For accidents, once they have occurred, the only available action is intervention. In most cases of practices and intervention, the magnitude of the exposures will be predictable, but with some uncertainty. Sometimes, there will be situations of 'potential exposure', but with no certainty that it will occur. It is often possible to apply some degree of control to both the probability and the magnitude of potential exposures.

4.3 THE SYSTEM OF RADIOLOGICAL PROTECTION

The system is based on the following general principles:

(a) No practice involving exposures should be adopted unless it produces sufficient benefit to the exposed individuals or to society to offset the detriment that it causes (THE JUSTIFICATION OF A PRACTICE).

(b) The magnitude of individual doses, the number of people exposed, and the likelihood of incurring exposures where these are not certain to be received, should all be kept *as low as reasonably achievable, economic and social factors being taken into account.* This procedure should be constrained by the restrictions to doses to individuals ('dose constraints') or the risks to individuals in the case of potential exposures ('risk constraints'), so as to limit the inequity likely to result from the inherent economic and social judgements (THE OPTIMIZATION OF PROTECTION).

(c) The exposure of individuals resulting from the combination of all the relevant practices should be subject to dose limits (or to some control of risks in the case of potential exposures). These are aimed at ensuring that no individual is exposed to risks that are judged to be unacceptable from these practices in any normal circumstances. Not all sources are susceptible of control by action at the source and it is necessary to specify the sources to be included as relevant before setting a dose limit (INDIVIDUAL DOSE AND RISK LIMITS).

ICRP's system of protection recommended for intervention is based on the following general principles:

(i) The proposed intervention should do more good than harm, i.e. the reduction of detriment resulting from the reduction of dose should be sufficient to justify the harm and the costs, including social costs, of the intervention.

(ii) The form, scale and duration of the intervention should be optimized so that the net benefit of the reduction of dose, i.e. the benefit of the reduction in radiation detriment, less the detriment associated with the intervention, should be maximized.

Dose limits do not apply in the case of intervention. Principles (i) and (ii) can lead to 'intervention levels' which give guidance to the situations in which intervention is appropriate. There will be some level of pro-

jected dose above which, because of serious deterministic effects, intervention will be justified.

4.4 RADIOLOGICAL PROTECTION IN PROPOSED AND CONTINUING PRACTICES

4.4.1 Justification of a Practice

Decisions on human activities are often carried out in two stages. In the first stage each option is examined separately and a 'short list' of preferred actions is selected. The second stage is the final selection. This may involve either the introduction of a new practice or the replacement of one existing practice by another. In the latter case the net benefit of the change will be the relevant feature rather than the net benefit of each option separately. When practices involving exposure or potential exposure are being considered, the radiation detriment (in addition to other detriments and costs) should be explicitly included in the process of choice. Often the radiation detriment will be a small part of the total. The justification of a practice thus goes far beyond the scope of radiological protection, and ICRP limits the use of the term 'justification' to establishing that the net benefit be positive; the choice of the best among available options is usually a task beyond the responsibility of radiological protection agencies.

Justification is required also when existing practices are being reviewed. If the review shows that the practice cannot be justified, its withdrawal should be considered, keeping in mind that the disadvantages of withdrawing a well-established practice may be more obvious than the advantages of introducing a new practice; further, withdrawal of the practice may not always result in withdrawal of all the associated sources of exposure. Preventing the further extension of an existing practice that is no longer justified may be a reasonable compromise.

4.4.2 Optimization of Protection

Once a practice has been justified and adopted, it is necessary to consider how best to use the resources in reducing the radiation risks to the individuals and the population. The broad aim should be to ensure that the magnitude of the individual doses and the number of people exposed (and the likelihood of potential exposures) are kept as low as

reasonably achievable, economic and social factors being taken into account. If the next step of reducing the detriment can be achieved only with a deployment that is seriously out of line with the consequent reduction, it is not in society's interest to take that step, provided that individuals have been protected, and protection can then be taken to be optimized.

Judgements on optimizing protection can range from simple common sense to complex techniques of cost-benefit analysis or multi-attribute analysis. Such judgements are also not purely quantitative - they involve preferences between detriments of different kinds and between the deployment of resources and health effects. Guidance on this subject is given in ICRP publications 37 (1983) and 55 (1989). It is appropriate to use the effective dose as a surrogate for detriment to an individual and the collective effective dose for the collective detriment.

The optimization procedure, which is essentially source-related, should be carefully structured. It should be first applied at the design stage where it will have some generic aspects and where cost-effective dose reduction can be achieved. Account has also to be taken of the substantial advantages offered by engineering standardization. Further optimization should be carried out at the operational level, which is usually informal, involving common sense changes in procedures, but often very effective.

As mentioned earlier, optimization may introduce some inequity between one individual and another. This can be minimized by source-related 'dose constraints' (previously called 'upper bounds') in the optimization procedure. For potential exposures, the corresponding concept is the 'risk constraint'.

4.4.3 Individual Dose Limits

If justification and optimization have been conducted effectively, there will be few cases where limits on individual dose will have to be applied. Such limits provide an upper bound to prevent excessive individual detriment from a combination of practices. Dose limits should be applied only in the control of practices.

It is ICRP's intention to choose the values of dose limits so that any continued exposure just above the limits would result in additional risks that could be reasonably described as 'unacceptable' in normal circumstances. This involves difficult social judgements.

There have been several misconceptions about dose limits. The dose limit is widely, but erroneously, regarded as a line of demarcation between 'safe' and 'dangerous'. It is also widely, and erroneously, seen as a simple way of keeping exposures low and forcing improvements. It is again seen as the sole measure of the stringency of the protection system. These misconceptions are, to a certain extent, strengthened by incorporation of dose limits into regulatory instruments; causing a dose limit to be exceeded becomes an infraction of rules and sometimes a statutory offence.

It will not be appropriate to apply dose limits to all types of exposures in all circumstances. In situations for which they were not intended, e.g., emergencies or special operations, they can be replaced by specially defined prescriptive limits or doses that call for the initiation of a specific course of action; these levels are often called 'investigation levels' and 'action levels', or in more general cases, 'reference levels'. The specification of dose limits and choice of values are discussed in Chapter 5.

4.4.4 Potential Exposures

Not all exposures occur as forecast. There may be accidental departures from planned procedures, equipment failure, or environmental changes (*e.g.* after disposal of radioactive waste). Such events can be broadly foreseen and their probability estimated. Such potential exposures have to be considered as part of the assessment of practices, but they may also lead to calls for intervention. If the probability of occurrence of potential exposures is high, so that several such events may occur in a year, it should be assumed that the doses resulting from the event will certainly occur.

Dose limits do not apply directly to potential exposures. They should be supplemented by risk limits, which take account of both the probability of incurring a dose and the detriment associated with it if the dose were received. Further, a potential exposure may become a real exposure and call for intervention. The problems are discussed in Chapter 5.

4.5 RADIOLOGICAL PROTECTION BY INTERVENTION

In some situations, the sources, pathways, and the exposed individuals are already in place when the decision about intervention is being considered. A typical example of such situations is exposure to natural background

radiation. Accidents and emergencies will have to be considered as sources of potential exposure when dealing with practices, but if they occur, may call for intervention. Chapter 6 deals with intervention.

In most situations, intervention cannot be applied at the source and has to be applied in the environment and to individuals' freedom of action. The countermeasures will always have some disadvantages, and should be justified. Their form, scale and duration should then be optimized to maximize the net benefit. ICRP recommends against the application of any dose limit for deciding on the need for, and scope of, intervention. Nevertheless, at some level of dose, approaching that for serious deterministic effects, intervention will become almost mandatory.

4.6 ASSESSMENT OF THE EFFECTIVENESS OF THE SYSTEM OF PROTECTION

There should be an overall assessment of the effectiveness in practice of the total system of protection, which should be treated as a coherent system. The assessment should be based on the distribution of doses achieved and on an appraisal of the steps taken to limit the probability of potential exposures. Mere compliance with the dose limits is not a sufficient demonstration of satisfactory performance. Comparison with comparable operations and trends with time will often indicate the possibility of improvements. The assessment is more difficult for potential exposures, because it is not always easy to directly determine their probability. For intervention, the assessment should concentrate on the effectiveness of the forward planning and, retrospectively, on the effectiveness of the action taken in particular cases.

CHAPTER 5

THE SYSTEM OF PROTECTION FOR PROPOSED AND CONTINUING PRACTICES

5.1 INTRODUCTION

This chapter indicates how ICRP develops the concepts described in Chapter 4 in the contexts of occupational, medical and public exposures. It relates to practices but excludes intervention. The main structure of the recommended control procedures is set out. Although, by and large, these three types of exposures can be treated separately, there are several interactive situations which are discussed later in the chapter. The practical implementation of the system is discussed in Chapter 7.

5.2 TYPES OF EXPOSURE

5.2.1 Occupational Exposure

The conventional definition of occupational exposure to any hazardous agent includes all exposures incurred at work, regardless of their source. Here, the meaning of 'occupational exposure' is limited to exposures incurred at work as the result of situations that can reasonably be regarded as being the responsibility of the management.

ICRP considers that handling of materials containing traces of natural radionuclides and exposures to natural background radiation should be excluded from occupational exposure and treated separately, unless the regulatory authority has ruled otherwise. There should be a requirement to include exposures to natural sources as part of occupational exposure only in the following cases:

(a) Operations in work places where the regulatory authority has decided that radon needs special attention (*e.g.*, places like uranium mines, many hard rock and underground mines, spas);

(b) Operations with and storage of materials not usually regarded as radioactive, but which contain significant traces of natural radionuclides and which have been identified by the regulatory agency;

(c) Operation of commercial jet aircraft (mainly the aircraft crew; perhaps also couriers who fly more often than other passengers); and

(d) Space flight.

Exposures to natural sources in workplaces need to be taken into account only if they would be controlled in their own right as indicated in the previous paragraph [Cases (a)-(d)]. Elsewhere, they need not be included in radiation monitoring results or in statistical reports of occupational exposures.

Exposure of workers from artificial sources in the workplace should be included in occupational exposure unless they have been formally excluded from regulatory control or exempted by the regulatory authority. Guidance on exclusion and exemption is discussed in Chapter 7.

5.2.2 Medical Exposure

Medical exposure is confined to exposures incurred by individuals as part of their own medical diagnosis or treatment, to exposures (other than occupational) incurred knowingly and willingly by individuals helping patients undergoing diagnosis or treatment, and to exposures of volunteers in a biomedical research programme. Exposure of an individual to stray radiation from the diagnosis or treatment of other persons, as well as occupational exposure of staff, are not included.

5.2.3 Public Exposure

This encompasses all exposures other than occupational and medical exposures. (Exposure to natural sources is by far the largest component of public exposures.)

5.3 THE APPLICATION OF THE SYSTEM OF PROTECTION

The system of protection described in Chapter 4 can usually be applied in the same way in all types of exposures.

Many of the methods of controlling exposures are applied without reference to the age or sex of those exposed, and so it is desirable to set limits and to optimize protection in ways that are independent of age and sex. Moreover, differences in the age and sex distribution in determining risk coefficients would have only a small effect on the definition of effective dose and on the nominal probability coefficients.

The dose limits recommended here apply only to the sum of dose contributions from a relevant set of exposures (external exposure in the specified period plus the committed dose from intakes during the same period), and not to those from all sources of radiation.

5.4 SYSTEM OF PROTECTION IN OCCUPATIONAL EXPOSURE

5.4.1 The Optimization of Protection in Occupational Exposure

An important feature of optimization is the choice of dose constraints, the source-related values of individual dose used to limit the range of options to be considered for optimization. It is possible to predict the level of individual doses likely to be incurred in well-managed broad classes of occupation (such as X-ray diagnostic departments, operation/inspection/maintenance of nuclear power plants). Limits prescribed by regulatory agencies and restrictions applied by managements to specific operations as part of the day-to-day control of exposures are not constraints in the sense used here; these limits should be established on the basis of optimization. It will be appropriate for dose constraints to be fixed at the national or local level. Although optimization should take account of both actual and potential exposures, decisions about potential exposures often have no implications for actual exposures and can be dealt with separately (see later in this chapter).

5.4.2 Dose Limits in Occupational Exposure

Dose limits apply to all occupational exposure (as defined earlier), including that resulting from minor mishaps in operation and from maintenance in operations not necessarily envisaged by the designers. This is an extension of ICRP's previous concept of dose limits and represents a substantial increase in the stringency of the ICRP recommendations, regardless of any change in the magnitude of the dose limits.

The basis of choosing a limit corresponding to an acceptable individual risk is elusive. ICRP 26 (1977) used a comparison with the rates of accidental death in conventional industries. These comparisons are not altogether satisfactory for a number of reasons. For example, standards of industrial safety are neither constant nor uniform world-wide; mortality data relate to averages over whole industries while dose limits apply to individuals; comparisons were limited in ICRP 26 to mortality data with no inclusion of non-fatal conditions; society may not expect to have the same standards of safety across a wide range of industries.

The present more comprehensive approach of ICRP is to establish a dose level above which the consequences for the individual would be widely regarded as unacceptable. For this purpose, the dose limit can be expressed as a lifetime dose received uniformly over the working life, or as the annual dose received every year of work. In the past, ICRP had used the attributable probability of death or serious hereditary conditions as the basis for judging the consequences of an exposure. This quantity, while still a major factor, is not adequate, and other factors (like length of life lost due to an attributable death and incidence of non-fatal conditions) have also to be considered in arriving at the detriment.

A single aggregated index representing the detriment, while attractive in theory, leads to practical difficulties in judging the implications of this aggregated index. ICRP uses three words to indicate the tolerability of an exposure (or risk). 'Unacceptable' indicates that the exposure would not be reasonably acceptable in any normal situation. 'Tolerable' exposures are not welcome but can be reasonably tolerated. 'Acceptable' means that they can be accepted without further improvement, i.e. when the protection has been optimized. A range of quantifiable factors taken together has been considered by ICRP to provide a basis for judgement.

Several possible values of dose are selected. These test values are expressed as the total accumulated dose at a uniform rate during a working life of 47 years or as annual doses received every year over a working lifetime. The relationship between annual and accumulated dose is valid for external exposure and for short-lived internal emitters. For long-lived internal emitters retained for a long time in the body, the dose is spread out in time and may not all be delivered during the lifetime of the individual. The assessment then overestimates the consequences of internal exposure expressed in terms of the 50-year committed equivalent dose.

The consequences of the continued uniform exposure to each of the test values in turn are evaluated, and a view reached as to which value

gives rise to a combination of consequences which is just short of acceptable. This approach makes it possible to consider a wide range of interrelated attributes such as :

- Lifetime attributable probability of death;
- Time lost if the attributable death occurs;
- Reduction of life expectancy (combination of the first two attributes);
- Annual distribution of the attributable probability of death; and
- Increase in age-specific mortality rate (*i.e.* probability of dying in a year at any age, conditional on reaching that age).

These attributes relate to mortality. Morbidity due to non-fatal cancers and hereditary disorders is allowed for by using the number of non-fatal conditions, weighted for severity, as discussed in chapter 3, and for the period of life lost or impaired. For non-fatal cancers, this weighted number amounts to about 20% of the detriment due to fatalities and for hereditary conditions to approximately 20% of the number of fatalities for workers (and about 27% for the whole population).

The test values selected as a possible basis for the annual dose limit were 10, 20, 30 and 50 mSv, corresponding to lifetime dose of 0.5, 1.0, 1.4 and 2.4 Sv. Combinations of attributes were considered to enable judgements made on the basis of the whole structure. The attributes for the test values of annual effective dose are shown in Table 5.1. (Further details are given in Annexes B and C of ICRP 60.)

The first combination reviewed was that of the probability of an attributable fatal cancer and the average period of life lost if the attributable fatality occurs. For an annual dose, received every working year, this combination can be expressed as a lifetime probability of losing a stated period of time. This period is almost independent of the annual dose, since, at low doses, it depends only on the time of the attributable death and not on its probability. For the combination of an additive risk model for leukaemia and a multiplicative model for other cancers, the loss is 13 years. For the purely additive model, it is around 20 years. Another attribute, itself a combination of these data, is the mean loss of life expectancy at age 18 years as a result of a subsequent occupational exposure.

In Table 5.1, results derived from the data available in 1977 for an annual dose of 50 mSv over 40 years are included for comparison. (These

numbers were not used by ICRP 26 as the basis for selection of the dose limit at that time; the selection was made on a different basis - comparing the average fatal cancer risk in radiation work with the fatality risk in 'safe' non-radiation occupations and assuming a ratio of 10:1 between the maximum and the average risk.)

Regarding the way in which the probability of attributable death varies with time (Fig. 5.1), the combined effect of latency and the extended period of exposure is to produce a distribution sharply peaked in time at older ages for both the additive and multiplicative models. The age of maximum (unconditional) annual probability of attributable death following the exposure of a population of equal numbers of men and women over a whole working lifetime (age 18 to 64) occurs at 68 years for the additive model and 78 years for the multiplicative model, and is almost independent of the annual dose. The term 'unconditional' is used to indicate that the probability is not conditional on reaching the age for which the probability is quoted. The conditional probability continues to rise indefinitely.

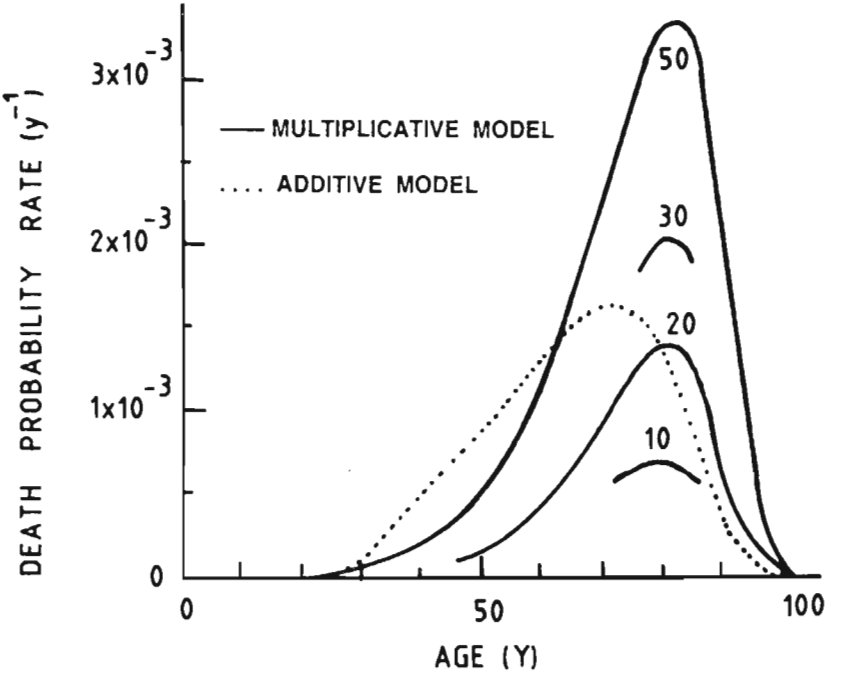

Figure 5.1. The unconditional death probability rate (the attributable death age probability density normalized for lifetime risk) for exposure from age 18 to 65 years (curves are for females) (from ICRP 60).

The results indicate that a lifetime effective dose of 2.4 Sv (corresponding to 50 mSv/year) would be regarded by many as being clearly too high. In particular, the reduction of life expectancy by 1.1 years and the fatality probability of 8.6% that radiation hazards would be the cause of the worker's death, albeit at a late age, would be widely seen as excessive. ICRP has concluded that the working lifetime dose should not exceed 1 Sv; further, the protection system should be such that this figure would only rarely be approached. Deterministic effects would also have to be protected against.

If the limits are based on doses accumulated over a period of many years, such long control periods can be misused by allowing a rapid accumulation of doses and intakes near the start of a control period. Flexibility of this kind also weakens the emphasis on achieving control by design, transferring the emphasis to operational controls. On the other hand, the earlier ICRP recommendations of a rigid control period of one year is inflexible. For similar reasons, ICRP dose not now recommend the use of lifetime limits.

Flexibility might be provided by setting the limit in the form of a total dose accumulated over a period of a few years, while retaining an annual limit higher than the annual average over a longer period. ICRP believes that 5 years would provide sufficient flexibility. It recommends an effective dose limit of 20 mSv per year averaged over 5 years (100 mSv in 5 years) with the further provision that the effective dose should not exceed 50 mSv in any single year. The 5-year period should not be introduced and then applied retrospectively. It is implicit that the dose constraint for optimization should not exceed 20 mSv in a year.

Regulatory authorities may provide temporary relaxations in the dose limits because of the difficulties of responding rapidly to an increase in the stringency of the operations on plant and equipment already in existence. Such arrangements should be regarded as transient.

Dose limits are not to be seen as a target but as the point at which regular, extended, lifetime occupational exposure at those levels can reasonably be regarded as only just tolerable. The use of source-related dose constraints selected by the regulatory agencies and their application in the process of optimization are additionally recommended.

These limits are sufficient to ensure avoidance of deterministic effects in almost all body tissues, except for the lens of the eye (which makes a negligible contribution to the effective dose) and the skin which may be

subject to localized exposures. Separate dose limits are needed for these tissues.

For the lens of the eye the annual dose limit is 150 mSv (as earlier). In the case of skin, for stochastic effects the equivalent dose can be averaged over the whole area of the skin. The stochastic effects are expected to arise in the basal layer at a nominal depth of 7 mg cm^{-2}, i.e. 70 μm (range 2-10 mg cm^{-2} or 20-100 μm). Some deterministic effects occur at the same depth, others arise in the deeper layers of the dermis (30-50 mg cm^{-2}, or 300-500 μm).The limitation on the effective dose provides protection against stochastic effects. To prevent deterministic effects for localized exposures, an annual limit of 500 mSv averaged over any 1 cm^2, regardless of the area exposed, is recommended. This limit, applied to the skin of the face, will also protect the lens of the eye against localized exposure from radiations of low penetrating power like beta particles. The same limit applies to hands and feet.

For internal exposure, annual limits on intake (ALI) are provided in ICRP publication 61 (1991) and will be based on a committed effective dose of 20 mSv. The estimated intakes may be averaged over 5 years to provide some flexibility. This will in practice ensure that the lifetime equivalent dose in any organ will not lead to deterministic effects. Till revised occupational limits for radon are recommended, the existing recommendations in ICRP publication 47 (1986) remain valid.

5.4.3 The Occupational Exposure of Women

If a woman is, or may be, pregnant, additional controls have to be considered to protect the unborn child, as the conceptus is at times more prone than the post-natal individual to deterministic injuries and may be more sensitive to induction of later malignancies. Deterministic effects, including significant mental retardation, will not occur if the exposure of the mother does not exceed the dose limits now recommended for occupational exposure, regardless of the time distribution of the exposure. Accidental higher exposures may be more damaging to the conceptus than to the mother. ICRP recommends no special occupational dose limits for women in general, since the existing system will provide, in the case of a woman who may be pregnant, a standard of protection to the conceptus broadly comparable with that for members of the public.

Once pregnancy has been declared, the conceptus should be protected by applying a supplementary dose limit to the surface of the mother's

abdomen (lower trunk) of 2 mSv for the remainder of the pregnancy and by limiting intakes of radionuclides to about 1/20 ALI. The normal system of protection recommended by ICRP (particularly the use of source-related dose constraints) will usually provide a guarantee of compliance with this limit without the need for specific restrictions for women. The employment should be of a type that does not carry a significant probability of high accidental doses and intakes. (Regulatory agencies should identify such situations.)

5.5 THE SYSTEM OF PROTECTION IN MEDICAL EXPOSURE

5.5.1 Justification

The justification of a practice leading to medical exposures should be dealt with in the same way as for any other practice. Most of the benefits and detriments accrue to the individuals undergoing diagnosis or treatment, but account should be taken of all the resulting exposures, including occupational and public exposures. The practice should first be defined in broad terms. However, each procedure is subject to a separate decision on a case-by-case basis for justification. This will not be necessary for routine diagnostic procedures based on common indications, but may be important for complex investigations and therapy. Guidance for these procedures is given in ICRP publications 34, 44 and 52. Chapters 37 to 41 of this Handbook discuss in detail the question of radiation protection in medical applications.

5.5.2 Optimization

Because most procedures causing medical exposures are strongly justified and for the direct benefit of the exposed individual, not much attention has been given to optimization, and there is considerable scope for dose reduction in diagnostic radiology by simple, low cost measures without loss of diagnostic information. Doses for the gamut of medical exposure investigations cover a range of two orders of magnitude. Dose constraints or investigation levels may be prescribed by the regulatory authority for some common diagnostic procedures. They should be applied with flexibility to allow higher doses wherever warranted by sound clinical judgement.

Constraints would be more stringent in clinical research involving exposure of volunteers who may not get any direct benefit from the exposure.

5.5.3 Dose Limits

If the practice is justified and the protection optimized, the dose to the patient will be as low as compatible with the medical purposes. ICRP therefore recommends that dose limits should not be applied to medical exposures. (Dose constraints may however be considered.)

For similar reasons, it is not appropriate to include doses from medical exposures when considering compliance with dose limits from occupational or public exposures.

5.5.4 Medical Exposure of Pregnant Women

Information on likely pregnancy should be obtained from the patient herself. If the last expected period has been missed, the woman should be assumed to be pregnant. Diagnostic and therapeutic exposures of the abdomen of women likely to be pregnant should be avoided unless there are strong clinical indications.

5.6 THE SYSTEM OF PROTECTION IN PUBLIC EXPOSURE

The control of public exposure in normal situations is exercised by controls at the source. For long-lived radionuclides discharged into the environment, the concept of dose commitment to the 'critical group' (see next para) is useful (as an alternative to long-term environmental models linking releases to individual and collective doses). The collective effective dose commitment per unit of practice can be used for justification and optimization. (Part of the effective dose may be received only in the distant future.)

5.6.1 Optimization

In practice, almost all public exposure is controlled by optimization procedures, including the widespread use of dose constraints. It is convenient to class together individuals who form a homogeneous group with respect to exposures to a single source. A 'critical group' is such a group typical of those most highly exposed to that source. Dose constraints should be applied to the mean dose in the critical group. In formulating dose constraints, possibility of exposure from other sources has to be kept in mind. Constrained optimization helps to develop practical restrictions on the source, e.g. restrictions on releases of wastes into the environment.

5.6.2 Dose Limits

With the widespread use of dose constraints at the source, generally applicable dose limits are rarely limiting in practice. But since the public may be theoretically exposed to several sources, ICRP has recommended dose limits for public exposure. These limits are limited to doses incurred in practices. Intended emissions of radionuclides from installations (including mines and waste disposal sites) should be treated as practices and resulting doses subject to the dose limits. Intervention situations are excluded from the scope of dose limits. Separate attention has also to be paid to potential exposures. (Doses from radon in buildings and other natural exposures are outside this scope.)

How do we arrive at dose limits for public exposure? One approach is the same as for occupational exposures, *i.e.* assess the consequences of various possible dose limits and judge the point at which the consequences can reasonably be described as unacceptable. The latter part of the exercise is more difficult than for occupational exposure. Alternatively, the judgement can be based on the variations in natural background levels. Variations from place to place (excluding large variations in dose from radon in dwellings) can hardly be called unacceptable.

Study of the consequences of continued additional exposures, ranging from 1 to 5 mSv per year, suggests a value of the annual dose limit not much above 1 mSv (although the change in age-specific mortality rate even at 5 mSv per year is very small). Excluding the variable exposure to radon, the annual effective dose from natural sources is about 1 mSv, with values at high altitudes and in some geological areas of at least twice this. On the basis of all these considerations, ICRP recommends an annual dose limit of 1 mSv.

In deriving dose limits for public exposure, allowance must be made for variations in environmental pathways to man, variations in effectiveness of source control procedures and the consequent transient dose increases. (Doses due to major accidents - which will involve intervention - are not subject to dose limits). ICRP has currently recommended that the limit for public exposure should be an effective dose of 1 mSv per year. In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year. Because this represents only a slight change from the previous recommendation, ICRP recommends that the 5-year period should be applied retrospectively when the new recommendation is being

implemented. It is implicit in this limit that the constraints for optimization in the design of new installations should be smaller than 1 mSv per year.

Limits are also needed for the lens of the eye and localized areas of the skin. Because the total period of exposure may be nearly twice as long as for occupational exposure, and because the exposed individuals may show a wide range of sensitivity, ICRP has adopted an arbitrary reduction factor of 10 in the dose limits for public exposure compared to occupational exposure. The annual limits are 15 mSv for the lens and 50 mSv averaged over any 1 cm^2 area of the skin. The recommended dose limits for occupational and public exposure are summarized in Table 5.2.

5.7 POTENTIAL EXPOSURES

The initial treatment of potential exposures should form part of the system of protection applied to practices (but the exposures, if they occur, may lead to intervention). The objectives are prevention and mitigation. The probability of the sequences of events that may increase exposures should be reduced. It involves maintaining the reliability of operating and safety systems, and of the working practices. Mitigation is the limitation of exposures if any of these sequences do occur. It involves the use of engineered safety features and operational procedures to control each sequence of events, and if it occurs, to limit consequences. Arrangements for mitigation should not be restricted to plans for intervention. Steps should be taken at the design and operation stages to reduce the consequences of accident sequences so that intervention may not become necessary.

For coherence in the treatment of actual and potential exposures, the concept of detriment should be extended to include the probability of occurrence of the situation giving rise to the detriment. Techniques for achieving this are still being developed. Uncertainties in estimating the probability of occurrence will usually be much greater than uncertainties in estimating the probability of consequences should they occur.

The simplest approach is to consider the overall (*a priori*) individual probability of attributable death from cancer, rather than the effective dose, as the quantity to be used for protection. This probability is the product of the probability of incurring the dose and the lifetime conditional probability of attributable death from the dose were it to have been incurred. A restriction corresponding to a dose limit can then be expressed

in the form of a risk limit, i.e. a limit on the fatality probability. This use of the overall individual risk is a reasonable starting point but not sufficient. Low probability, high dose events may call for lower risk constraints than would be needed for high probability, low dose situations. Further, actual doses and dose rates may be higher than those for which risk factors have been obtained, and deterministic effects may also become important.

The specification of collective detriment from potential exposures is difficult, and probably not appropriate. For example, is it correct to assume that a frequent event with small consequences and a rare event with large consequences are equally detrimental if the expectation values of the consequences are the same? Multi-attribute analysis for collective detriment is complex.

A simpler approach is possible for both individual and collective exposures if the doses will be small (say, less than the dose limits) even if the event were to occur. We can then use justification and optimization for the product of the expected dose and its probability of occurrence (as if this were a dose that is certain to occur).

5.7.1 Justification

If information is available, detriment associated with a proposed practice should include that from the potential exposures in the assessment of justification.

5.7.2 Optimization

If the options for applying the protection system to potential exposures do not alter the other exposures resulting from the practice, the potential detriment can be used for optimization. Sometimes, the two sets of exposure are interdependent, and optimization should be carried out for both taken together. In either case, the procedure must be constrained by an individual risk limit, or, more probably, by source-related and sequence-related individual risk constraints.

5.7.3 Individual Risk Limits and Constraints

The risk limit can be defined by analogy with the dose limit, but it will have a different character. The probability of events leading to

potential exposures cannot be determined by observation but only by probability safety assessment techniques. We can usefully define a series of risk constraints applicable to the attributable probability of death. ICRP dose not yet recommend an annual risk limit for individuals.

There is also the possibility of potential doses in medical exposures. Errors in dosimetry and equipment failure have given rise to injurious, and sometimes fatal, doses to patients. ICRP does not recommend any specific value for risk constraints in this context.

ICRP publication 64 (1993), 'Protection from potential exposures : A conceptual approach' elaborates on the principles and objectives of ICRP in this area. Its contents are summarized in Appendix 5.1.

5.8 INTERACTIVE SITUATIONS

There are some cases where the system of protection applied to one situation changes the condition in another. For example, a reduction in public exposure from radioactive waste releases may result in an increased occupational exposure due to additional waste processing and storage. A combined collective effective dose from these two forms of exposure (if necessary, suitably weighted to take account of the difference in perception of the effect of unit collective dose for the two cases, although ICRP would prefer only the unweighted collective dose to be used) can be used for optimization. Another example is the interaction between potential exposure and occupational or public exposure. The inspection of a plant may reduce the probability of failures but only at the expense of additional occupational exposure. This form of interaction can be dealt with only by multi-attribute analysis.

Table 5.1: Attributes of detriment due to occupational exposure of the working population

(from ICRP 60)

Annual effective dose (mSv)	10	20	30	50	50 (1977 data)
Approximate lifetime dose (Sv)	0.5	1.0	1.4	2.4	2.4
Probability of attributable death (%)	1.8	3.6	5.3	8.6	2.9
Weighted contribution from non-fatal cancer (%)[1]	0.4	0.7	1.1	1.7	
Weighted contribution from hereditary effects (%)[1]	0.4	0.7	1.1	1.7	1.2
Aggregated detriment (%)[2]	2.5	5	7.5	12	
Time lost due to an attributable death, given that it occurs (years)	13	13	13	13	10-15
Mean loss of life expectancy at 18 years (years)	0.2	0.5	0.7	1.1	0.3-0.5

1. Weighted for severity and loss of lifetime.
2. The sum of the probability of attributable fatal cancer or equivalent detriment (rounded).

Table 5.2: Recommended dose limits for occupational and public exposure[1]
(from ICRP 60)

Application	Dose limit	
	Occupational exposure	Public exposure
Effective dose	20 mSv per year averaged over defined period of 5 years[2]	1 mSv per year[3]
Annual equivalent dose in		
Lens of the eye	150 mSv	15 mSv
Skin[4]	500 mSv	50 mSv
Hands and feet	500 mSv	-

1. The limits apply to the sum of the relevant doses from external exposure in the specified period and the 50-year committed dose (to age 70 years for children) from intakes during the same period (See text).

2. With the further provision that the effective dose should not exceed 50 mSv in any single year. Additional restrictions apply to the occupational exposure of pregnant women (See text).

3. In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

4. The limitation on the effective dose provides sufficient protection for the skin against stochastic effects. An additional limit is needed against localized exposures in order to prevent deterministic effects (See text).

APPENDIX 5.1

PROTECTION FROM POTENTIAL EXPOSURES: A CONCEPTUAL FRAMEWORK

INTRODUCTION

ICRP 60 distinguishes between normal and potential exposures. Potential exposure is exposure that, while not certain to occur, can be anticipated as a result of introducing or modifying a practice and to which a probability of occurrence can be assigned. A combination of the probability of a potential exposure with the dose (individual or collective) resulting from it can be presented as an *a priori* probability of harm. The initial consideration of potential exposures, therefore, should form part of the system of protection applied to practices, with the recognition that the exposures, if they occur, may lead to intervention. ICRP 64 , 'Protection from potential exposure: A conceptual approach' (1992) elaborates on the principles and objectives of ICRP 60 as they relate to potential exposure and shows how the fundamental safety concepts can practically be applied to all potential exposure situations.

Consequences of potential exposure may be highly variable. For example, in the case of industrial radiography the consequences will be fairly predictable and limited (such as overexposure of a few individuals). For a nuclear installation the consequences may be of a wide range, including economic and social disruption, in addition to radiation health effects. For a waste disposal repository, the consequences may occur in the distant future.

BASIC CONCEPTS

In addition to the concepts dealt with in ICRP 60, there is a somewhat more detailed discussion on probability in ICRP 64.

If the probability of an event related to one observed unit, such as a radiation source, is p, then the probability of at least one event in a group of N units will be

$P_N = 1 - (1-p)^N$

which for small values of the product pN becomes $P = pN$, but for large values of pN approaches but never exceeds unity.

If the probability per unit time is dp/dt, the probability of at least one event over the time period T will be $P_T = 1 - e^{-(dp/dt)T}$. This probability is dimensionless and cannot exceed unity.

The frequency in time, f, is the average number of events per unit time. For events to be observable, the inverse value ($1/f$) of the frequency (having the dimension time) should be shorter than the time period. If the observation relates to a large number of units (N), each with an event probability rate of dp/dt, the mathematical expectation of the number of events over a time period T will be $\tilde{n} = (dp/dt)NT$.

The mathematical expectation of frequency will be $\tilde{n}/T = (dp/dt)N$. If $dp/dt << 1/T$ and N is large, the outcome will show a Poisson distribution.

A distinction is to be made between the probability rate (or expectation of frequency) and the actually observed frequency. If $T < 1/f$, the most likely observations are zero or one event. With $T < 0.7(1/f)$, the probability of no event is always larger than 50%.

Safety analysis starts with estimates of frequencies of initiating events and combines them with the estimated probabilities of failure of various safety functions, leading to an assessment of the *a priori*, i.e., unconditional, probability rate, dp/dt for each specified end effect. The probability of the final event in a given time span can then be assessed using the expression for P_T given earlier. Safety analysis will identify the most likely sequence of event sequences where safety improvements may be appropriate. The result of the safety analysis, sometimes expressed by the inverse value of the estimated event frequency, is a useful figure of merit in comparison of safety results. A sensitivity analysis of the safety analysis results will indicate the robustness of the probability assessment with regard to variation between experts of the subjective judgements involved.

THE BASIS OF RADIATION SAFETY

Processes that may cause potential exposure may be thought of as a network of potential events and situations. A 'scenario' is a specific combination of events, features and processes starting from an initiating

event and leading to radiological consequences. A 'scenario-related safety assessment' is the estimation of risk associated with a particular scenario. It allows a judgement to be made of whether all reasonable steps have been taken to reduce the corresponding risks. The 'source-related assessment' is a combination of assessment of all possible scenarios, taking into account both the magnitude of individual risks and the number of individuals exposed. In most potential exposure situations, it will not be feasible to perform 'individual-related assessments'.

In addition to the basic principles of justification, optimization and individual risk limitation, implementation of a safety programme should be based also on sound technical and managerial principles. The most important technical principle is that there should be layers (structures, components, procedures) of overlapping safety provisions ('defence-in-depth'). Low overall probabilities of failure are most easily achieved by a combination of independent protective layers such that the probabilities of failure are multiplicative; further, human and mechanical failures as well as unforeseen failure modes have less effect on the overall protection.

The first emphasis is on accident prevention measures; this must be supplemented by accident mitigation measures. Both should be addressed primarily during the design phase. Emergency planning (e.g., sheltering, short-term evacuation) is a last-resort mitigation measure. The possibility of human errors as one of the primary contributors to many events should be taken into account.

Adoption of sound engineering principles and practices, a comprehensive quality assurance programme, qualified and trained personnel, and periodic safety assessments are other technical principles for achieving safety.

The primary responsibility for safety rests squarely with the management. Regulatory bodies should provide regulatory as well as advisory frameworks to the management bodies. At the managerial level, a consistent and pervading 'safety culture' must be inculcated in all workers.

PRACTICAL APPLICATIONS

The safety levels associated with sources of potential exposures are usually established by the designers or operators and judged by the competent authority. Judgements on optimization are not purely quantitative; they reflect preference between detriment of different kinds and between

the deployment of resources and the radiation detriment. The optimization process should be carefully structured, aiming at coherency and consistency. Its application to the design and implementation of the safety measures will result in reduction of potential doses and their probabilities in cost-effective ways.

Relevant factors like probability of the exposure, total risk, distribution of risks, possibility of deterministic effects, public *vs.* occupational exposures, applicability of safety measures, should all be taken into account. Progressively higher weighting should be applied to higher doses and to some types of radiological consequences (such as large-scale or long-lived ground contamination). Final decisions about optimized safety should result from considerations (qualitative and quantitative) of both normal and potential exposures.

For potential exposures, individual risk constraints (in the process of optimization) should be of the same order of magnitude as the health risk imposed by dose limits for normal exposures (although dose limits themselves are not applicable). The probabilities of doses being incurred and/or the doses themselves can be constrained by improvements in reliability of systems, procedures, and human actions.

One procedure for applying source-related constraints to probabilistic events is to express the probability of an event sequence as a function of the dose that will be delivered should the sequence occur. Such probabilities can be worked out for various scenarios. These probabilities can then be compared with those established for various potential scenarios and judgements made on the need for increasing safety reliability or augmenting redundancy of safety systems.

As the complexity of the system increases, the sequences linking an initiating event and the final consequences will form increasingly complex event or fault trees. Sequences should be grouped based on key attributes which characterize similar conditions for the analysis of consequence. The probability of each group would be the sum of the probabilities for each sequence in the group. The sequence leading to the worst consequences in the group as a whole can be used to represent the group in a conservative risk analysis.

For choice of individual risk constraints, sequences that have been grouped together based on radiation safety considerations might be chosen for the values listed in Table APP 5.1.1. (The criteria are consistent with those specified in ICRP 46 for waste disposal.) For constraints involving

consequences other than potential individual exposure (such as socially disruptive effects) multiattribute analysis will have to be resorted to.

Assessment Techniques

These comprise deterministic and probabilistic assessment methods. The former is used first to provide safety, with appropriate safety margins. The latter is then used to identify weaknesses in the safety systems which otherwise might have been overlooked in the former method.

Deterministic methods are characterized by rigidly specified scenarios (design basis scenarios) in terms of initiating events and component failures.

In probabilistic safety assessment (PSA) (techniques of which have been well developed in the 1970s), the probability of a set of predetermined consequences for various sources or practices is evaluated. The detrimental end effects are first defined and estimates made of their probabilities. All relevant initiating events and associated events that may lead to the specific set of detrimental effects are then mapped (e.g., power failure in a nuclear reactor for which the frequency is known; rare events like earthquakes in low seismicity regions for which empirical data are lacking). For each initiating event, potential event sequences are mapped, fault tree-event tree sequences modelled, and branching points in the trees assigned probabilities (describing the failure probability of certain systems, which may be known from operating experience, or which may have to best guess estimates by experts). For complex systems, computer support may be needed for such analysis. The end result is an estimate of the frequency of the chosen set of detriments occurring, together with a set of initiating events and sequences determining this frequency of occurrence. This may be used for assessing the risk of potential exposure.

There are several sources of uncertainty in such estimates. Established methods of quality assurance (e.g., peer review) will improve the reliability of the analysis.

Application in Practice

The techniques of potential exposure assessment have been employed for many years in the case of nuclear power plants. They can also be applied to other complex facilities. In some of these cases, there may not be operations centralized in a control room (as for power reactors), pro-

cesses may be subject to frequent changes, and chances of human error may be more; emphasis should therefore be placed on assessment of human reliability, proper operating procedures, training and managerial surveillance.

Examples of less complex sources (which have however led to serious injuries and death due to combination of human and equipment failures) are food and medical irradiation facilities and industrial radiography. Greater use of PSA techniques would be rewarding in such cases.

Loss of source security or improper management of industrial and medical sealed sources has led to loss and theft of sources, resulting in injury and death, as well as widespread contamination in some cases. Sealed sources should be clearly labelled and securely guarded, and provision for reducing spread of contamination in the event of damage after loss or theft incorporated in the design.

Potential exposure should be considered in medical applications. Human failure has been a major component of many therapy accidents leading to serious injury or death. PSA assessments, including consideration of the human-machine interface and human reliability would be effective. Software errors have led to substantial errors in patient treatment before the error was discovered. Quality assurance of dosimetry software, as well as independent checks and calculations by two persons at critical stages are recommended.

The problem of probabilistic risk assessment in the context of waste disposal has been dealt with in ICRP 46 (1985).

Exclusion of Scenarios and Event Sequences

Provisions are often made for excluding some situations from the scope of regulatory instruments due to lack of ability to affect either the probability or consequences of the dose (e.g., meteor strike). Such scenarios may be taken into account in decisions about justification.

Exemption of Scenarios and Event Sequences

In normal exposures the grounds for exemption are that the source gives rise to small individual doses (of the order of 10 μSv) and that protection is optimized. There is no analogous basis for exempting potential exposure from the regulatory system. However exemption may be

considered if the practice has been justified, safety options have been selected by optimization, and the resulting individual risk is trivial.

REGULATION IN THE CONTEXT OF POTENTIAL EXPOSURES

The operating management should first assess the expected frequency and possible consequences of events that might give rise to doses substantially higher than normal, taking into account a wide range of initiating causes (including those outside the operator's control, such as floods and storms). The operating procedures necessary to deal with such events, should they occur, should be specified. The next stage is regulatory review. Procedures for reviewing the operator's assessment should be worked out. The use of risk constraints related to individual sequences should be considered. These may make it unnecessary to establish overall risk limits, which are difficult to select and enforce.

Compliance with risk limits and constraints has to be judged from the results of the quality of the design, operation and maintenance, as well as the quality of the management arrangements.

Table APP 5.1.1: Range of probabilities in a year from which constraint may be selected

(from ICRP 64)

Sequence of events leading to doses in the following categories	Range of probability
Those treated as part of normal exposures	10^{-1} to 10^{-2}
Those leading to stochastic effects only but above dose limits	10^{-2} to 10^{-5}
Where some effects may be deterministic	10^{-5} to 10^{-6}
Where death is likely to result	$< 10^{-6}$

CHAPTER 6

THE SYSTEM OF PROTECTION IN INTERVENTION

6.1 INTRODUCTION

This chapter deals with situations where the sources of exposure and the exposure pathways are already present and the only type of action available is intervention. It deals mainly with intervention applying to public exposure, including not only intervention following accidents, but also material on occupational exposure in emergencies.

The proposed intervention should be justified, i.e. do more good than harm, and the form, scale and duration of the intervention should be chosen to optimize protection. ICRP recommends against the use of dose limits for deciding on the need for, or scope of, intervention.

6.2 THE BASIS OF INTERVENTION IN PUBLIC EXPOSURE

In judging the benefits and detriments of intervention, the comparison should first be made for those at risk (although there will also be an impact on the rest of society and judgements should be wide enough to cover these impacts).

In justification, we have to decide that the disadvantages of each component of intervention are more than offset by the reduction in dose likely to be achieved. In optimization we have to decide on the method, scale and duration of the action to obtain the maximum net benefit. The difference between the disadvantages and benefits, expressed in the same terms, e.g. costs, including social costs with an allowance for anxiety, should be positive for each protective action and should be maximized.

The cost of intervention is not just the monetary cost. Some actions (e.g. short-term or long-term removal of people from their homes) may involve non-radiological risks or serious social impacts.

6.3 SITUATIONS IN WHICH REMEDIAL ACTION MAY BE NEEDED

Many situations in which intervention is being considered are of long standing and do not call for urgent action, while accidents require immediate action.

6.3.1 Radon in Dwellings

Both the individual and collective doses from radon are higher than those from any other source. In many countries some individual doses are substantially higher than even occupational dose limits. Improvements will have to be achieved by intervention involving modifications to the dwellings or to the behaviour of its occupants. ICRP 39 has recommended the use of action levels to help in deciding when to advise remedial action in existing dwellings. The choice of an action level is complex, since there may be economic implications for the community and the individuals depending upon the scale of recommended action. For owner-occupied dwellings, general guidance may be adequate, leaving the final decision to the occupier. In countries with substantial numbers of rented dwellings, it may be desirable to establish national action levels. For the general case, action level may well be at that level which defines a significant, but not unmanageable, number of houses in need of remedial work.

The problem of new dwellings has some similarity to that of existing dwellings because radon concentrations cannot be determined with confidence until the dwelling has been completed and occupied for a year or so; it is then an existing dwelling.

Guides or codes can be established for the construction of new dwellings in selected areas to have a reasonable assurance that the exposures will be below some chosen level. ICRP is reviewing the situation but has not made any specific recommendation on the level. Meanwhile, the guidance in ICRP publication 39 (1984) remains valid. Chapter 42 of this Handbook deals in detail with the problem of radon exposures.

6.3.2 Radioactive Residues from Previous Events

Examples are burial of long-lived wastes from mining, or luminizing with radium compounds. Construction of dwellings on mining tailings used as land-filled material has caused problems. Buildings used for

radium work have subsequently been put to other uses, with the radium being discovered only years later. There have been several accidents in which long-lived radionuclides have been dispersed in residential and agricultural areas. The remedial actions vary greatly in scale and may themselves give rise to problems of occupational exposure and waste disposal. Such situations should be dealt with in accordance with the recommendations for practices. No general solutions are available, but optimization procedures can give some guidance.

6.4 ACCIDENTS AND EMERGENCIES

6.4.1 Intervention Affecting the Public

The types of protective action, costs and expected reduction in individual and collective doses have to be evaluated. A substantial amount of preliminary work on economic and environmental models and on accident forecasting is needed.

Small-scale, short duration, intervention may be costly without being effective. As the scale and duration are increased, the effectiveness initially increases without a marked increase in costs. Eventually, further increases will lead to a reduction in net benefit. There is then a range of values of the possible intervention level, within which there is an optimum level, for which, if the net benefit is positive, intervention will be justified. The initial emergency planning should include the choice of intervention levels that will be justified and reasonably optimized.

Each action has to be considered on its own merits. For example, a decision about control of an individual foodstuff is independent of decisions about other foodstuffs and of decisions about sheltering or evacuation. The doses that would be incurred on all relevant pathways (some subject to protective action and some not) should be assessed. Doses causing serious deterministic effects or a high probability of stochastic effects would call for a review of additional protective action. An intervention level of dose received by all the pathways should be chosen at the planning stage. ICRP publications 40 (1984) and 63 (1993) have set out the general principles for planning intervention after an accident that involve short- and medium-term (but not long-term) actions, as well as quantitative guidance on intervention levels.

6.4.2 Limitation of Occupational Exposure in Emergencies

Occupational exposures directly due to an accident can be limited only by the design of the safety features of the plant and by the provision of emergency procedures. In the case of a serious accident, it may not be possible to keep the doses within occupational dose limits.

In addition to direct exposures as a result of the accident, there will also be exposures of emergency teams during emergency and remedial action. The doses are likely to be higher than in normal situations but can be limited by operational controls. Exposures in the control of the accident and in the urgent remedial work should not give effective doses of more than about 0.5 Sv except for life-saving actions. The effective dose to skin should not exceed 5 Sv. Once the emergency is under control, remedial work should be treated as part of the occupational exposure incurred in a practice.

CHAPTER 7

IMPLEMENTATION OF THE COMMISSION'S RECOMMENDATIONS

7.1 INTRODUCTION

This chapter shows how the operational level of protection should be developed from the requirements of the regulatory agencies and the ICRP recommendations. It gives advice on the measurement of doses (monitoring) and on possible bases for exemption from regulatory requirements. It deals with both practices and intervention.

The main practical responsibilities fall on the designers and operators of equipment and installations. Governments should establish a framework of regulatory and advisory functions aimed at helping the operating managements to meet their responsibilities and ensure protection.

The organizational structures used in the control of practices should, as far as possible, also be used for intervention in order to maintain consistency. Planning for intervention in the event of an emergency should be an integral part of normal operating procedures. Where there is no operating management (e.g. for radon in dwellings) intervention should normally be the responsibility of the regulatory agency.

The recommendations have been set out as a logical sequence of concepts:

- Allocation of responsibility
- Basic recommendations
- Requirements of regulatory bodies
- Management requirements
- Validation of performance

To a large extent these stages are the same for all types of exposure situations (except for intervention where there may not always be a relevant management).

7.2 RESPONSIBILITY AND AUTHORITY

'Responsibility' (a prospective concept) is the duty to establish objectives, to provide measures needed to achieve these objectives, and to ensure that these measures are properly carried out. Those bearing responsibility should have the 'authority' to commit the resources needed to meet these responsibilities. 'Accountability' (a retrospective component of responsibility) requires a programme of continuing review of performance to determine how effectively the objectives are achieved and to correct failures.

The primary responsibility for achieving and maintaining control of radiation exposures rests squarely on the management bodies of the concerned institutions. Governments have the responsibility to set up regulatory agencies which must provide both the regulatory and advisory frameworks.

The responsibilities and authority are delegated to an extent depending on the complexity of the duties. The working of this delegation should be examined regularly. There should be a clear line of accountability running right to the top. Although the operating organization has the primary responsibility, often there is some *de facto* transfer of responsibility from the operator to the regulator. Such requirements (mandatory rules imposed on the operators), while effective, should be justified.

Administrative devices are not, of themselves, enough to achieve protection. Everyone in an undertaking should regard protection and accident prevention as integral parts of his every-day functions.

7.3 THE RECOMMENDATIONS OF THE COMMISSION

The recommendations provide a basis from which to derive regulatory requirements and guidance to the operating managements. The widespread adoption of the recommendations will give consistency of aims and standards across many countries and provide a degree of uniformity of procedures. ICRP therefore tries to make clear the reasons for its recommendations and has deliberately included some flexibility in order to maintain consistency without rigidity.

Acceptance of the quantities discussed in Chapter 2 and of the proposed values of the nominal probability coefficients as well as the radiation and tissue weighting factors will simplify world-wide comparisons of

doses and practices and will help in the development of engineering standards for instrument design and performance.

7.4 REGULATORY REQUIREMENTS

Regulatory provisions are not an alternative to management requirements but a bridge between ICRP recommendations and the management requirements. Regulatory agencies have a large part in assessing the justification of a practice (as well as in prohibiting practices not regarded as being justified). One important national and international need is the provision of adequate resources for the education and training of professional and technical staff in radiological protection (which cannot be provided by the regulatory agencies alone).

7.4.1 The Regulation of Practices

One feature of regulation is the source-related constraints to be applied to optimization. (Regulatory constraints are not the same as prescriptive regulatory limits.) Limits prescribed by regulatory agencies and restrictions applied by managements in day-to-day control are not constraints in the sense used here. These constraints should be established on the basis of the results of optimization. Prescriptive limits may apply not only to doses but to other features under management control such as releases to the environment. It is not satisfactory to consider prescriptive limits as an alternative to optimization nor to set design or operational limits or targets as an arbitrary fraction of the dose limit.

Many operations can be conducted so that the standard of protection is set by the process of constrained optimization and not by the dose limits. Mandatory dose constraints or investigation levels can be set by the regulatory agency for classes of operations. Exceeding an investigation level would require an investigation of the optimization programme.

Special attention should be given to justification and optimization when an individual is seen to be consistently exposed at a high level close to the individual dose limit. The regulatory agencies should be concerned with public exposures because of the probability of individuals being exposed to more than one source.

The regulatory provisions may be general, or directed to a class of installations. The regulatory agency should consider both the source-related and the individual-related approaches. In certain cases control of

the source may have to be achieved by inter-state or inter-agency collaboration.

The objectives and methods of regulatory agencies may sometimes have to be subject to formal international requirements. There is also a range of international engineering standards, some of which have a bearing on protection. Advisory documents by responsible international bodies provide valuable inputs.

7.4.2 Regulation in the Context of Potential Exposures

As a first step, the management should assess the expected frequency and possible consequences of events (such as accidents, major errors in design and operation) that might give rise to doses substantially higher than normal. Account should be taken of a wide range of initiating causes, even those outside the operator's control, such as floods and storms. The operator should prepare procedures necessary to deal with such events.

The second stage is that of regulatory review of the operator's assessments. Particularly in the few installations where the consequences of an accident might be severe, a more detailed review should take place, possibly linked to a prior approval or licensing. The use of risk constraints related to individual sequences should be considered. (These may make unnecessary the establishment of overall risk estimates which are difficult to select and enforce.)

The quality of the management arrangements, including design, performance and reliability of plant, equipment, quality of test procedures, operating instructions, and training should be assessed.

7.5 MANAGEMENT REQUIREMENTS

The most important step is the establishment of a safety-based attitude in everyone concerned, from design to decommissioning. This can only be achieved by a commitment to training, recognition that safety is not only a personal responsibility but is also of major concern to the top management, and close links between management and the work-force.

A formal structure should be created for dealing with protection, including optimization and issuing of clear operating instructions. Design and operation should be parts of a unified system called 'management requirements'.

The aim of the management requirements should be to set out the practical basis for protection. Attention should be given to the layout of plant and equipment. Techniques for control of exposures include choice of radiation sources, use of shielding and distance to reduce radiation fields, restrictions on time spent in the proximity of sources, and use of several stages of containment. Plans should be prepared for dealing with potential exposures and accidents (including safety analysis, assessment of reliability of all principal systems, operating and maintenance procedures, and performance of human operators). These plans should be periodically reviewed and written management requirements prepared, which should be clear, unambiguous and eminently practical. Management requirements should take due account of requirements of regulatory agencies, and also draw on ICRP recommendations, manuals of good practice and engineering standards.

7.5.1 The Classification of Workplaces and Working Conditions

Management must maintain control over the sources of exposure and the workers. Working places containing radiation sources should be formally designated. Two types of working places are considered, viz. 'controlled areas' and 'supervised areas'.

A controlled area is one in which normal working conditions, including the possible occurrence of minor mishaps, require the workers to follow established procedures and practices aimed specifically at controlling exposures. A supervised area is one in which the working conditions are kept under review but specialized procedures are not normally needed. Where only external radiation hazards exist, designated areas may be defined in terms of dose rates at the boundary. Anyone outside the designated areas will not need to be regarded as occupationally exposed; actual doses received outside the designated areas should be below the dose limits for public exposure. The earlier definition of a supervised area as one where doses are likely to be less than 3/10 of the occupational dose limit is now regarded by ICRP as too arbitrary. ICRP now recommends that the designation should be decided either at the design stage or locally by the management on the basis of operational experience.

In previous recommendations, ICRP defined two types of working conditions based on the expected level of individual doses. This was originally intended to help in the choice of workers to be subject to individual monitoring and special medical surveillance. ICRP no longer recommends such a classification.

7.5.2 Operational Guides

General principles to keep exposures low should be translated into specific statements that the designers and operators can use as guides. These should indicate the maximum levels of exposure expected in operations. The management is responsible for making these guides and establishing operational instructions. But the guides are not targets and are not sufficient, but only provide an envelope within which to work. In addition, there should be an obligation to consider available options and to achieve operational objectives conforming to optimization to suit specific situations.

While in principle the operating guides should include material on 'safety goals' (standard of reliability needed to limit potential exposure situations), there are difficulties in practice. It becomes necessary to depend on past experience, often codified in the form of engineering standards.

7.5.3 Reference Levels

'Reference levels' are values of measured quantities above which some specific action should be taken. They include 'recording levels', above which a result should be recorded, lower values being ignored; 'investigation levels', above which the cause or the implications of the result should be examined; and 'intervention levels', above which some remedial action should be considered. The use of these levels avoids unproductive work.

7.5.4 Occupational Services for Protection and Health

The management must provide access to occupational services (either in-house or consultancy) dealing with protection and health, which should give specialist advice and arrange for necessary monitoring provisions, both inside and outside the installation. The head of the protection service should have direct access to the senior management.

The principal role of the occupational health service is the same as in any other occupation. Physicians supervising the health of a force of radiation workers need to be familiar with their tasks and working conditions, and then decide on the fitness of each worker. The radiation component of the working environment by itself very rarely has a significant influence now-a-days on the decision.

The supervising physician may be required to counsel workers in three special categories. The first is women who are, or may become, pregnant. They should be advised to inform the physician as soon as they think they may be pregnant, so that the management can be advised to arrange for any necessary change of duties. The second group comprises individuals who have been exposed substantially in excess of the dose limits. Only in exceptional conditions will clinical tests or treatment be indicated. The physician should be prepared to provide such specialist facility at short notice if needed. (For example, chromosomal aberration analysis of peripheral lymphocytes can give useful results and reassurance after suspected accidents. In-house provision for this test is rarely needed because there would be many laboratories in the country to which blood samples can be sent for such analysis.) The third group contains volunteers for biomedical research programmes involving radiation exposure. Doses in these cases will be normally small compared to occupational exposures, and will be limited by dose constraints. Reference to an ethics committee is needed to ensure the soundness of the research aims and methods for selecting volunteers.

Individual medical and dose records are considered as confidential; this desirable element should not compromise the availability of the data to the management, supervising physician, and others involved in protection.

7.6 ASSESSMENT OF DOSES

The measurement or assessment of doses is fundamental to the practice of radiological protection. Neither the dose equivalent nor the effective dose can be measured directly. Hence 'best estimates' must be inferred with the aid of realistic but simple environmental, metabolic and dosimetric models. The models should not be likely to under-estimate the doses, although they should also not excessively over-estimate them. For justification and optimization purposes, both over- and under-estimation are liable to cause misuse of resources and models have to be realistic. Where practicable, estimates should be made of the uncertainties inherent in these results.

7.6.1 Dosimetry in Occupational Exposure

It is feasible to monitor individual doses in occupational exposures. To avoid a wasteful use of resources in monitoring and record-keeping, it is necessary to identify groups of workers for whom individual monitoring

is necessary. The decision depends on many factors, including maintaining good industrial relations. The decision taken by the management should be subject to review by the regulatory agency. Three major factors influence the decision, viz. the expected levels of dose or intake in relation to the limits, their likely variations, and complexity of measurement and interpretation of monitoring procedures. Individual monitoring for external exposure is simple and cheap, and should normally be done for all occupational workers. It gives information on control of exposures, confirms the classification of workplaces, gives reassurance to workers, and provides inputs for review of optimization. Individual monitoring for intakes is more difficult and should be routinely used only for workers in controlled areas involved in handling of unsealed sources. Guidance on types of work calling for individual monitoring is given in ICRP publication 35 (1982) and on interpretation of individual monitoring for workers in ICRP publication 54 (1988).

For calculating annual limits on intake (ALIs) it would be more appropriate to use the 35-year committed effective dose for workers (mean working life of 40 years, age 18-65 years, and an expectation of life of 75 years) than the earlier value of the 50-year committed effective dose. But since the difference is small, even for long-lived, long-retained nuclides, ICRP recommends the retention of the earlier value for occupational exposure. In assessing possible health implications of an intake, account has to be taken of the actual age at intake. In discussing the health implications of a given intake with the workers, the intake can be related to the ALI more convincingly than the committed dose can be related to the annual dose limit.

The assessment of collective doses is to be based on recorded doses from individual monitoring programmes (supplemented by use of data on low individual doses derived from models based on measurements at the workplace).

It is possible to achieve an accuracy of 10% (at the 95% confidence level) for measurement of radiation fields under laboratory conditions. In the workplace where the energy and orientation of the radiation field are rarely known, uncertainties by a factor of 1.5 are acceptable for external individual doses. But an uncertainty by a factor of at least 3 in estimation of intakes and committed equivalent and effective doses may have to be accepted.

7.6.2 Dosimetry in Medical Exposure

In medical exposure, assessment of doses to patients is of critical importance in radiotherapy (and is dealt with by the International Commission on Radiation Units and Measurements, ICRU). Quality control programmes including frequent measurements on equipment are important in this case. In diagnostic radiology, there is rarely a need for routine dose assessment, but periodic quality control of equipment should be made and optimization encouraged. In nuclear medicine, administered activity should always be recorded, in which case the doses, based on standard models, would be readily available.

7.6.3 Dosimetry in Public Exposure

Routine individual monitoring is normally not necessary and is not recommended. Dose assessment is then dependent on models for pathways between the source and the individual, sometimes supplemented by environmental monitoring. As mentioned earlier, the models should relate to reasonably homogeneous 'critical groups' which are chosen to represent the individuals most likely to be highly exposed. Individual dose limits can be applied to the mean values for the critical group. ICRP publication 43 (1985) deals with the selection of critical groups.

The integrating period for committed effective dose is from the age of intake to 70 years for children and 50 years for adults. ICRP publication 56 (1989) provides age-specific relationships between intake and committed effective dose.

It is rare for the collective dose to be predominantly composed of doses to members of the critical group. Dose assessments for justification and optimization have to be based on more general models. These models and the dose assessments therefrom can be validated by selective measurements on environmental materials for short-term predictions. Techniques of sensitivity and uncertainty analysis may have to be used for longer-term predictive models (extending over many centuries and over large areas).

7.7 COMPLIANCE WITH THE INTENDED STANDARD OF PROTECTION

All concerned organizations should have a duty to verify their compliance with their own objectives and procedures by a system analogous to financial auditing. Regulatory agencies should conduct similar audits,

and should also have the duty of, and authority for, assessing the level of protection and compliance with the regulatory provisions achieved by managements, which should also cover potential exposures. The verification procedures should include a review of quality assurance programmes and some form of inspection. However, inspection is only a form of sampling and cannot cover all eventualities; it is at best a mechanism for persuading those inspected to put their houses in order.

7.7.1 Record Keeping

Any system of validation includes record keeping (as required by the regulatory agencies and the management). The type of record, degree of detail and retention period should be defined formally. A balance has to be struck between the complexity of the initial entry of data and the possible future use of the records. The value of most records decreases with time as does the likelihood of their being needed. Individual dose records should be retained for periods comparable to the expected lifetime of the individual. Monitoring results of workplaces should be retained for a few years (in case of likely reassessment). Details of releases of waste to the environment should be retained for at least 10 years, with summaries being kept for several decades.

7.8 EMERGENCY PLANNING

In the event of an emergency that may affect the public, there should usually be a shift in responsibility. The operating management at the scene of the initiating event will take initial control of the event. But if the event is beyond the operator's premises this may not be appropriate, and the regulatory agency might have to take the responsibility for decisions and also decide who shall be responsible for implementing the actions following its decisions.

Accidents or operational misjudgements may call for urgent action. Responsibility for planning local emergency action should fall primarily on the operating management. More general, especially national, planning should be the responsibility of the regulatory agency. Local and national plans need to be coordinated and linked to other plans dealing with accidents not involving radiation. There may be necessity for bilateral agreements with neighbouring states if major installations are located at national boundaries.

The first key area of difficulty is the recognition that an accident has occurred and that emergency action is needed. This is easy if the accident is to a major plant, but dangerous situations due to lost or misused radiographic sources have been difficult to recognize. The second problem area is the rapid acquisition and interpretation of data. There will be a widespread demand for data to provide reassurance even in unaffected areas. Thirdly, the interpreted data have to lead to decisions and actions or to a convincing conclusion that no action is needed. It should be recognized that the initial decisions will often have to be made by someone on the spot, regardless of the formal chain of responsibility. However, provision should also be made for the more formal making of decisions on a longer time-scale. The fourth problem area is the communication system which is difficult to maintain, especially with the public. Local advice is fairly straightforward, but it is difficult to disseminate reassurance to the much larger areas where no action is called for.

Many parts of emergency plans will not be in routine use, but they must be maintained in a state of readiness by regular exercises.

The state of emergency should be formally declared (at the local or more widespread levels) and provision should also be made for withdrawal of the emergency and of any countermeasures.

In an emergency the emphasis shifts from dose limits to intervention levels for immediate decision-making. Plans should include a set of intervention levels promulgated by the regulatory agency for various possible scenarios to provide a basis for urgent decisions. The choice of these levels should be based on the dose averted by the proposed action. This latter cannot be estimated immediately, and hence derived intervention levels should be established for quantities that can be measured or estimated at the time of use. The intervention levels should not be treated as limits but are only guides to action.

To avoid unnecessary restrictions in international trade, especially in foodstuffs, derived intervention levels could be derived as a special case to indicate a line of demarcation between freely permitted exports or imports ('intervention exemption levels') and those that should be the subject of special decisions. Trade in materials above these levels should not automatically be prohibited, but might be subject to temporary controls.

7.9 EXCLUSION AND EXEMPTION FROM REGULATORY CONTROL

To avoid excessive regulatory procedures, most regulatory systems include provisions for granting exemptions in cases where a practice is justified but where regulatory provisions are unnecessary, as well as for complete exclusion of some situations from the scope of regulatory requirements. Exemption is an important component of the regulatory function.

Exemptions can be given in two cases: (i) if the source gives rise to small individual as well as small collective doses in both normal and accident conditions; and (ii) when no reasonable control procedures can achieve significant reduction in individual or collective doses.

The basis for exemption on the grounds of trivial doses is much sought after, but difficult to establish. For example, both individual and collective doses from a single smoke detector will be trivial, but for smoke detectors in general, the individual doses can still be small but the collective dose may be substantial. Exemption is necessarily source-related while the triviality of the dose is individual-related.

When the exempt source comprises a class of devices, it may not be appropriate to exempt their manufacture and large-scale storage. The devices themselves can be made subject to approved engineering standards and their sale and use can then be exempted; their eventual disposal will also have to be exempted.

Sources that are essentially uncontrollable, such as cosmic radiation and potassium-40 in the body, can best be excluded rather than exempted.

Some sources give rise to widespread exposures involving only very small individual doses, and the reduction of such doses may be impossible with any reasonable deployment of resources. The method of exemption of such cases, ignoring the collective dose if the individual doses are all very small, is in use, not always explicitly, and often leads to conclusions that are broadly consistent with the ICRP system of protection. Nevertheless, ICRP does not recommend this approach. The extent to which small individual doses should be included in the estimation of collective doses for purposes of optimization depends on the extent to which the contributions from these doses influence the choice between options. Further guidance is given in ICRP publication 55 (1989).

CHAPTER 8

SUMMARY OF 1990 RECOMMENDATIONS OF ICRP (ICRP PUBLICATION 60)

8.1 INTRODUCTION

The recommendations are intended to be of help to regulatory and advisory agencies and to management bodies and their professional staff. They deal only with ionizing radiation and with the protection of man. Ionizing radiation needs to be treated with care rather than fear and its risks should be kept in perspective with other risks. Radiological protection cannot be conducted on the basis of scientific considerations alone. All those concerned have to make value judgements about the relative importance of different kinds of risk and about the balancing of risks and benefits.

8.2 QUANTITIES USED IN RADIOLOGICAL PROTECTION

ICRP uses macroscopic dosimetric quantities while recognizing that microdosimetric quantities based on the statistical distribution of events in a small volume of material may eventually be more appropriate. The principal dosimetric quantities are the mean absorbed dose in a tissue or organ, D_T, which is the energy absorbed per unit mass; the equivalent dose in a tissue or organ, H_T, formed by weighting the absorbed dose by the radiation weighting factor, W_R; and the effective dose, E, formed by weighting the equivalent dose by the tissue weighting factor, W_T, and summing over the tissues. The time integral of the effective dose rate following an intake of a radionuclide is called the committed effective dose, $E(\tau)$, where τ is the integrating time (in years) following the intake. The unit for absorbed dose is the gray (Gy), and the unit of both equivalent and effective dose is the sievert (Sv). The values of the radiation and tissue weighting factors are given in Tables 8.1 and 8.2 respectively.

The collective effective dose is the product of the mean effective dose in a group and the number of individuals in that group. With some reservations, it can be thought of as representing the total consequences of

the exposure of a population group.

'Dose' is used by ICRP as a generic term that can apply to any of the dosimetric quantities. The term 'exposure' is also used in a generic sense to mean the process of being exposed to radiation or radioactive material. The significance of an exposure in this sense is determined by the resulting doses.

8.3 BIOLOGICAL ASPECTS OF RADIATION PROTECTION

Ionizing radiation causes both deterministic and stochastic effects in irradiated tissue. Radiological protection aims at avoiding deterministic effects by setting dose limits below their thresholds. Stochastic effects are believed to occur, albeit with low frequency, even at the lowest doses and therefore have to be taken into account at all doses.

Deterministic effects result from the killing of cells which, if the dose is large enough, causes sufficient cell loss to impair the functioning of the tissue. The probability of causing such harm will be zero at small doses, but above some level of dose (the threshold for clinical effect) the probability will increase sharply to unity (100%). Above the threshold, the severity of the harm will increase with dose. Thresholds for these effects are often at doses of a few Gy or dose rates of a fraction of a Gy per year.

At Hiroshima and Nagasaki it was found that for children exposed *in utero* during the critical 8-15 week period following conception, there was a downward shift in the distribution of IQ with increasing dose which can result, after higher doses, in an increase in the probability of severe mental retardation. The effect is presumed to be deterministic with a threshold related to the minimum shift in IQ that can be recognized.

Stochastic effects may result when an irradiated cell is modified rather than killed. Modified somatic cells may subsequently, after a prolonged delay, develop into a cancer. There are repair and defence mechanisms that make this a very improbable outcome. Nevertheless, the probability of a radiation-induced cancer increases with increments of dose, probably with no threshold. The severity of the cancer is not affected by the dose. If the damage occurs in a cell whose function is to transmit genetic information to later generations, any resulting effects, which may be of many different kinds and severity, are expressed in the progeny of the exposed person. This type of stochastic effect is called 'hereditary'.

ICRP has estimated the probability of a fatal cancer based mainly on studies of Japanese survivors and their assessment by bodies like the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) and the Biological Effects of Ionizing Radiation (BEIR) Committee of the United States National Academy of Sciences, which have estimated the lifetime cancer risk from the accumulated data to 1985, the new dosimetry (Dosimetry System 1986, DS86), and projection to lifetime by a multiplicative or modified multiplicative model, for high dose, high dose rate exposures. ICRP has concluded that the most probable dose-response is linear-quadratic in form for low LET radiation. The linear coefficient at low doses or low dose rates is obtained from the high dose, high dose rate estimates of risk by dividing by a DDREF (Dose and Dose Rate Effectiveness Factor) of 2. The nominal fatal cancer probabilities for a working population and for a general population (which differ somewhat because of the greater sensitivity of young people) are given in Table 8.3. ICRP has also made estimates of how this fatal cancer risk is distributed among organs and the length of life lost for cancer in each of these organs by further analysis of the Japanese data.

The estimates of severe hereditary effects are also based on the assessments of UNSCEAR and BEIR of experimental data on animals. Evidence suggests that these estimates are not less than the corresponding effects in man. For low doses and dose rates, the probability coefficients for severe hereditary effects in all generations (resulting about equally from dominant and X-linked mutations on the one hand and multifactorial diseases weighted for severity on the other) are given for both a working population and a general population in Table 8.3.

The term 'detriment' is used by ICRP to represent the combination of the probability of occurrence of a harmful health effect and a judgement of the severity of that effect. Detriment is a multi-dimensional concept whose principal components are the following stochastic quantities: the probability of attributable fatal cancer, the weighted probability of attributable non-fatal cancer, the weighted probability of severe hereditary effects, and the length of life lost if the harm occurs. The values of this aggregated detriment at low doses for both a working population and a general population are also given in Table 8.3.

The distribution of the detriment in organs and tissues has also been assessed by considering first the fatal cancer probability in each of them, multiplying by an appropriate factor for non-fatal cancer (which is determined by the severity (lethality factor) for that cancer), adding in the probability of severe hereditary effects, and adjusting for the relative length

of life lost. This distribution of aggregate detriment among organs is represented, after appropriate rounding, by the tissue weighting factors, W_T given in Table 8.2.

The effective dose, which is the sum of the weighted equivalent doses in all the tissues and organs of the body is given by

$$E = \sum_T W_T . H_T$$

where H_T is the equivalent dose in tissue or organ T and W_T is the weighting factor for tissue T. It can also be expressed as the sum of the doubly weighted absorbed dose in all the tissues and organs of the body.

8.4 THE CONCEPTUAL FRAMEWORK OF RADIOLOGICAL PROTECTION

A system of radiological protection should aim to do more good than harm, should maximize the net benefit, and aim to limit the inequity that may arise from a conflict of interest between individuals and society as a whole.

Some human activities, called by ICRP as 'practices', increase the overall exposure to radiation, while 'intervention' refers to activities that can decrease the overall exposure by influencing the existing causes of exposure.

ICRP considers three types of exposure: occupational exposure, which is the exposure incurred at work, and principally as a result of work; medical exposure, which is the exposure of persons as part of their diagnosis or treatment; and public exposure, which comprises all other exposures.

In practices and in intervention, it will often be virtually certain that exposures will occur and their magnitude will be predictable, albeit with some degree of error. However, in 'potential exposures', there will be a potential for exposure but no certainty that it will occur.

8.4.1 The System of Protection in Practices

The system is based on the following general principles:

(a) No practice involving radiation exposure should be adopted unless it produces sufficient benefit to the exposed individuals or to society to

offset the radiation detriment it causes. (THE JUSTIFICATION OF A PRACTICE.)

(b) In relation to any particular source within a practice, the magnitude of individual doses, the number of people exposed, and the likelihood of incurring potential exposures should all be kept 'as low as reasonably achievable, economic and social factors being taken into account'. This procedure should be constrained by restrictions on the doses to individuals (dose constraints), or the risks to individuals in the case of potential exposures (risk constraints), so as to limit the inequity likely to result from the inherent economic and social judgements. (THE OPTIMIZATION OF PROTECTION.)

(c) The exposure of individuals resulting from the combination of all the relevant practices should be subject to dose limits, or to some control of risk in the case of potential exposures. These are aimed at ensuring that no individual is exposed to radiation risks that are judged to be unacceptable in any normal circumstances. Not all sources are susceptible of control by action at the source and it is necessary to specify the sources to be included as relevant before selecting a dose limit. (INDIVIDUAL DOSE AND RISK LIMITS.)

8.4.2 The System of Protection in Intervention

This is based on the following principles :

(a) The proposed intervention should do more good than harm, i.e. the reduction in detriment resulting from the reduction in dose should be sufficient to justify the harm and the costs, including social costs, of the intervention.

(b) The form, scale, and duration of the intervention should be optimized so that the net benefit of the reduction of dose, i.e.the benefit of the reduction in radiation detriment, less the detriment associated with the intervention, should be maximized.

Dose limits do not apply in the case of intervention. Principles (a) and (b) can give guidance to the situations where intervention is appropriate. There will be some level of dose above which, because of serious deterministic effects, intervention will almost always be justified.

Any system of protection should be treated as a coherent system which should include an overall assessment of its effectiveness in practice. This

should be based on the distribution of doses achieved and on an appraisal of the steps taken to limit the probability of potential exposures.

8.5 THE CONTROL OF OCCUPATIONAL EXPOSURE

8.5.1 Dose Constraints

It will usually be appropriate for dose constraints to be fixed at the national or local level, mainly based on the results of optimization. These are concerned with the source-related values of individual dose used to limit the range of options considered for optimization. For many broad classes of operations (like work in X-ray diagnostic departments, routine operation or inspection and maintenance of nuclear plants) it is possible to estimate the level of individual doses likely to be incurred. This information can then be used to establish corresponding dose constraints. Limits prescribed by regulatory agencies and restrictions applied by the management to specific day-to-day operations are not constraints in the sense used here.

8.5.2 Dose Limits

Dose limits are needed as part of the control of occupational exposure, both to impose a limit on the choice of dose constraints and to provide protection against errors of judgement in the application of optimization. The aim is to establish, for a defined set of practices, and for regular and continued exposure, a level of dose above which the consequences for the individual would be widely regarded as unacceptable. The most important parameter for judging the consequences of an exposure is the attributable probability of death or severe hereditary disorders, but other factors have also been taken into account in deciding on the detriment.

The dose limits for occupational exposure are summarized in Table 8.4. ICRP recommends a limit on effective dose of 20 mSv per year, averaged over 5 years (100 mSv in 5 years), with the further provision that the effective dose should not exceed 50 mSv in any single year. The 5-year period would have to be defined by the regulatory agency, e.g., as discrete 5-year calendar periods. ICRP would not expect the period to be introduced and then applied retrospectively. It is implicit that the dose constraint for optimization should not exceed 20 mSv in a year.

Subject to medical advice in individual cases, there need be no special restrictions applied to the exposure of an individual following a control period in which his exposure has exceeded a dose limit. Such events should call for an examination, usually by the regulatory agency, of the design and operational aspects of protection in the installation concerned, rather than for restrictions or penalties applied to the exposed individual. If the dose is unknown, or thought to be high, a physician should be consulted.

Because of the difficulties in responding rapidly to an increase in stringency of operations on plant and equipment already in existence, ICRP recognizes that regulatory agencies may wish to make temporary use of higher dose limits. Such arrangements should be regarded as transient.

The dose limit is not to be seen as a target; it represents the point at which regular, extended, deliberate, occupational exposure can reasonably be regarded as only just tolerable.

The restrictions on effective dose are sufficient to avoid deterministic effects in all body tissues except the lens of the eye, which makes a negligible contribution to the effective dose, and the skin, which may well be subjected to localized exposures. The annual limits are 150 mSv for the lens and 500 mSv for the skin, averaged over any 1 cm^2, regardless of the area exposed.

For internal exposures, annual limits on intake will be based on a committed effective dose of 20 mSv. The estimated intakes may be averaged over a period of 5 years to provide some flexibility. The occupational limits for radon are under review. Meanwhile the values given in ICRP publication 47 (1986) remain valid.

8.5.3 The Occupational Exposure of Women

The basis for the control of the occupational exposure of women who are not pregnant is the same as that for men and ICRP recommends no special occupational dose limit for women in general.

Once pregnancy has been declared, the conceptus should be protected by applying a supplementary equivalent dose limit to the surface of the woman's abdomen (lower trunk) of 2 mSv for the remainder of the pregnancy and by limiting intake of radionuclides to about 1/20 of the ALI. The use of the normal system of protection, particularly the use of source-

related dose constraints, will usually provide an adequate guarantee of compliance with this limit without the need for specific restrictions on the employment of pregnant women. The employment should be of a type that does not carry a high probability of high accidental doses and intakes. High dose and high risk occupations from which pregnant women should be excluded should be defined by regulatory agencies.

8.6 THE CONTROL OF MEDICAL EXPOSURE

The practices leading to medical exposures should be defined and justified in broad terms. However, each procedure, either diagnostic or therapeutic, is subject to a separate decision, so that there is an opportunity to apply a further, case-by-case, justification for each procedure. This will not be necessary for simple diagnostic procedures based on common indications, but may be important for complex investigations and therapy.

There is considerable scope for dose reduction in diagnostic radiology using optimization techniques. Consideration should be given to the use of dose constraints, or investigation levels, selected by the appropriate professional or regulatory agency, for application in some common diagnostic procedures. They should be applied with flexibility to allow higher doses where indicated by sound clinical judgement.

Constraints and optimization should also be considered when the procedures are not intended to be of direct value to the exposed individual, as in research studies involving volunteers.

Medical exposures are intended to provide a direct benefit to the exposed individual. If the practice is justified and the protection optimized, the dose to the patient will be as low as is compatible with the medical purposes. ICRP therefore recommends that dose limits should not be applied to medical exposures. Further, it is not appropriate to include the doses incurred by patients in the course of medical exposures when considering compliance with dose limits applied to occupational or public exposures.

Diagnostic and therapeutic procedures causing exposures of the abdomen of women likely to be pregnant should be avoided unless there are strong clinical indications. Information on possible pregnancy should be obtained from the patient herself. If the most recent expected menstrual period has been missed, the women should normally be assumed to be pregnant.

8.7 THE CONTROL OF PUBLIC EXPOSURE

The control of public exposure is normally exercised by the application of controls at the source (by constrained optimization and prescriptive limits) rather than in the environment. It is convenient to class together individuals who form a homogeneous group with respect to exposures to a single source. A 'critical group' which is typical of those most highly exposed should be identified and dose constraints applied to the mean dose in the critical group for optimization purposes.

8.7.1 Dose Limits

The scope of dose limits for public exposures is confined to the doses incurred as a result of practices. Where intervention is the only possible action to reduce exposures (e.g. some cases of natural exposures like radon in dwellings and in the open), no dose limits can be prescribed.

ICRP now recommends that the limit for public exposure should be expressed as an effective dose of 1 mSv in a year. However, in special circumstances, a higher value can be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

ICRP has selected the dose limit as corresponding to a value that is just short of unacceptable for continued exposure as the result of deliberate practices.

ICRP recommends annual limits of 15 mSv for the lens of the eye and 50 mSv for skin averaged over any 1 cm^2 to protect against deterministic effects.

The recommended dose limits for occupational and public exposures are summarized in Table 8.4.

8.8 POTENTIAL EXPOSURES

The initial treatment of potential exposures should form part of the system of protection applied to practices, but the exposures, if they occur, may lead to intervention. There should be two objectives, prevention and mitigation. Prevention is the reduction of the probability of the sequences of events that may cause increased exposure. Mitigation is the limitation of the exposures if any of these sequences do occur. A great deal can be accomplished at the stage of design and operation to reduce

the consequences of accidental sequences so that intervention may not become necessary.

A coherent system that includes both actual and potential exposures in the same framework would call for application of multi-attribute analysis and is being developed.

A simpler approach is possible if the doses, both for individual and collective exposures, are likely to be small (not in excess of dose limits) even if they occur; it is then adequate to use the conventional procedures of justification and optimization for the product of the expected dose and its probability of occurrence as if this were a dose that were certain to occur.

8.9 THE SYSTEM OF PROTECTION IN INTERVENTION

Before a programme of intervention is initiated, it should be demonstrated that it would be justified, i.e do more good than harm, and that the form, scale and duration of the intervention have been chosen so as to optimize protection. The difference between the disadvantages and the benefits, expressed in the same terms, e.g. costs, including social costs with an allowance for anxiety, should be positive for each action adopted and should be maximized by settling the details of that action.

8.10 RADON IN DWELLINGS

This aspect needs special attention because both the individual and collective doses from radon are higher than those from almost any other source. If improvements are needed in existing dwellings, they have to be achieved by intervention involving modifications to the dwellings (such as sealing of radon entry points in the flooring).

ICRP recommends the use of action levels to help in deciding when to require remedial action in existing dwellings. The choice of an action level is complex, depending on the level of exposure, the likely scale of action and its economic implications for the individual and the community. For new dwellings, guides for construction in selected areas can be established so that it is highly probable that exposures will be below some chosen reference level. Till ICRP issues further recommendations, ICRP publication 39 (1984) should be used.

8.11 INTERVENTION AFTER ACCIDENTS

Each protective action has to be considered on its own merits, based on the dose expected to be averted by it. In addition, the doses that would be incurred via all the relevant pathways of exposures, some subject to protective actions, and some not, should be assessed. Doses likely to cause serious deterministic effects or a high probability of stochastic effects would call for a review of the feasibility of additional protective action.

Occupational exposures of emergency teams during emergency and remedial action can be limited by operational controls. Some relaxation of the controls can be permitted in serious accidents. Persons involved should not get effective doses of more than 0.5 Sv except for life-saving actions, which can be rarely limited by dosimetric assessments. The equivalent dose to skin should not exceed about 5 Sv. Once the emergency is under control, remedial work should be treated as part of the occupational exposure incurred by a practice.

8.12 PRACTICAL IMPLEMENTATION OF THE RECOMMENDATIONS

Chapter 7 of ICRP 60 emphasizes the importance of the operational level of protection and shows how this should be developed from the requirements of regulatory agencies and the ICRP recommendations. ICRP now recommends that the designation of controlled and supervised areas should be decided at the design stage or locally by the operating management on the basis of operational experience and judgement. The classification of working conditions based upon expected dose is no longer recommended. The chapter gives advice on the measurement of doses (monitoring and record keeping) and on medical surveillance. It also discusses emergency planning and the bases for exemption from regulatory requirements. It deals with both practices and intervention.

Table 8.1: Radiation weighting factors[1]
(from ICRP 60)

Type and energy range[2]	Radiation weighting factor, W_R
Photons, all energies	1
Electrons & muons, all energies[3]	1
Neutrons, energy < 10 keV	5
10 keV to 100 keV	10
> 100 keV to 2 MeV	20
> 2 MeV to 20 MeV	10
> 20 MeV	5
Protons, other than recoil protons, energy > 2 MeV	5
Alpha particles, fission fragments, heavy nuclei	20

1. All values relate to the radiation incident on the body or, for internal sources, emitted from the source.

2. For the choice of values for other radiations, *see* Chapter 16.

3. Excluding Auger electrons emitted from nuclei bound to DNA (*see* Chapter 2).

Table 8.2: Tissue weighting factors[1]
(from ICRP 60)

Tissue or organ	Tissue weighting factor, W_T
Gonads	0.20
Bone marrow (red)	0.12
Colon	0.12
Lung	0.12
Stomach	0.12
Bladder	0.05
Breast	0.05
Liver	0.05
Oesophagus	0.05
Thyroid	0.05
Skin	0.01
Bone surface	0.01
Remainder	0.05[2,3]

1. The values have been developed from a reference population of equal numbers of both sexes and a wide range of ages. In the definition of effective dose, they apply to workers, to the whole population, and to either sex.

2. For purposes of calculation, the remainder is composed of the following additional tissues and organs: adrenals, brain, kidney, upper large intestine, small intestine, muscle, pancreas, spleen, thymus and uterus. The list includes organs which are likely to be selectively irradiated. Some organs in the list are known to be susceptible to cancer induction. If other organs subsequently become identified as having a significant risk of induced cancer they can be included with a specific tissue weighting factor or in this additional list constituting the remainder. The latter may also include other tissues and organs selectively irradiated.

3. In those exceptional cases in which a single one of the remainder tissues or organs receives an equivalent dose in excess of the highest dose in any of the 12 organs for which a weighting factor is specified, a weighting factor of 0.025 should be applied to that tissue or organ and a weighting factor of 0.025 to the average dose in the rest of the remainder as defined above.

Table 8.3: Nominal probability coefficents for stochastic effects
(from ICRP 60)

Exposed population	Detriment (10^{-2} Sv^{-1})[1]			
	Fatal cancer[2]	Non-fatal cancer	Severe hereditary effects	Total
Adult workers	4.0	0.8	0.8	5.6
Whole population	5.0	1.0	1.3	7.3

1. Rounded values.

2. For fatal cancer, the detriment is equal to the probability coefficient.

Table 8.4: Recoomended dose limits[1]
(from ICRP 60)

Application	Dose limit	
	Occupational exposure	Public exposure
Effective dose	20 mSv per year, averaged over defined periods of 5 years[2]	1 mSv in a year[3]
Annual equivalent dose in		
Lens of the eye	150 mSv	15 mSv
Skin[4]	500 mSv	50 mSv
Hands and feet	500 mSv	-

1. The limits apply to the sum of the relevant doses from external exposure in the specified period and the 50-year committed dose (to age 70 years for children) from intakes in the same period.

2. With the further provision that the effective dose should not exceed 50 mSv in any single year. Additional restrictions apply to the occupational exposure of pregnant women (*see* relevant section in chapter 5).

3. In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

4. The limitation on the effective dose provides sufficient protection for the skin against stochastic effects. An additional limit is needed for localized exposures to prevent deterministic effects (*see* relevant section in chapter 5).

APPENDIX 8.1

HISTORY AND ORGANIZATION OF ICRP

EARLY HISTORY

Within a few weeks of Roentgen's discovery of X-rays in 1895, the potential of X-rays for diagnosing fractures became apparent. But the occurrence of acute adverse effects - hair loss, anaemia, erythema, dermatitis and skin burns - within the next few years soon made persons aware of the need to avoid overexposure. Similar undesirable effects were reported after the discovery of radium and its medical applications. Notwithstanding these observations, protection of staff exposed to X-rays and gamma rays was poorly coordinated for many years. The first organized effort for radiation protection came in 1921 when the British X-ray and Radium Protection Committee issued detailed recommendations and instructions. The American Roentgen Ray Society also proposed general recommendations in the early 1920s on the basis of avoiding acute effects. In 1925, on the occasion of the First International Congress of Radiology, the need for quantifying 'exposure' was recognized. At the Second International Congress of Radiology held in Stockholm in 1928, the unit 'roentgen' was recognized as the unit for X-ray dose. It was at this Congress that the 'International X-ray and Radium Protection Commission' (the forerunner of ICRP) was set up with five members. It was concerned primarily with recommendations designed to provide protection to members of the medical profession in their work with X-rays and gamma rays from radium. The early recommendations of the Commission were patterned after the proposals of the British X-ray and Radium Committee. In 1934, the Commission recommended 0.2 R/day as the 'tolerance dose'. Prior to World War II, the Commission published recommendations at intervals of about 3 years. Activities of the Commission were halted during World War II.

FORMATION OF ICRP

The period from 1940 to 1950 was one of great expansion in radiation protection work consequent upon atomic energy developments. A great deal of experimental work was carried out and a wealth of information

was accumulated which laid the basis for the decisions of the post-war International Congress of Radiology in 1950. Many functional and organizational changes were brought into the Commission, to extend its scope and broaden the area of responsibility beyond the protection of the medical profession only.

The name of the Commission was changed to International Commission on Radiological Protection (ICRP). It consists of a chairman and not more than 12 other members. The selection of the members was made by the members of the International Congress of Radiology and by ICRP itself. Not less than two but not more than four members were to be changed at each Congress. The members were to be chosen on the basis of their recognized activity in the fields of radiology, radiation protection, physics, biology, genetics, biochemistry and biophysics, with regard to an appropriate balance of expertise, rather than on nationality. The Secretary of ICRP was elected by ICRP from among its regular members. Decisions were by a majority vote.

ICRP (1950)

ICRP (1950) spelt out for the first time the various effects which were to be considered in making its recommendations. It was clearly stated that the recommendations were to deal primarily with the basic principles of radiation protection and to leave to the various national committees the responsibility of introducing detailed technical regulations, recommendations or codes of practice suited to the needs of their individual countries.

The Commission was assisted by a number of Committees working in various specialized fields:

Committee I:	Permissible dose for external radiation
Committee II:	Permissible dose for internal radiation
Committee III:	Protection against X-rays up to energies of 3 MeV and beta and gamma rays from sealed sources
Committee IV:	Protection against electromagnetic radiation above 3 MeV and electrons, neutrons and protons
Committee V:	Handling of radioactive isotopes and disposal of radioactive waste

ICRP (1950-59)

The 1958 ICRP recommendations are important in many ways (and are dealt with in Appendix 8.2). A comprehensive report by Committee II was published as ICRP Publication 2 in 1959; it contained values of maximum permissible concentrations in body, air and water for about 240 radionuclides.

ICRP (1962)

In 1962 the Commission decided to reorganize the structure of its Committees. The previous committees were dissolved and four new committees were established:

1. Radiation Effects
2. Internal Exposure
3. External Exposure
4. Application of Recommendations

This reorganization did not change in essence the scope of programmes of the previous Committees I and II, but the old Committees III, IV and V were not included as such in the new structure. All problems relating to external exposure, both from quantum and particulate radiations of any energy, fell under the purview of Committee 3. Special *ad hoc* groups were set up from time to time to consider and report on specific problems.

PRESENT POSITION

During the last few years more organizational and functional changes have come to the ICRP. The selection of members is left to ICRP itself subject to approval from the International Executive Committee of the International Congress of Radiology. Not less than three and not more than five members are to be changed at every Congress, and the Scientific Secretary need not be a member of the Committee.

Meetings of the Commission with its Committees are held approximately every 2 years, and the Commission itself meets about once a year. In addition, Committees and Task Groups meet on their own.

ICRP PUBLICATIONS

Since its inception. this international body has brought out a number of publications. There had been 5 publications of the International X-ray and Radium Protection Commission. After 1950, when the Commission assumed its present form, there have been 64 ICRP publications till the end of 1993. Some of the earlier volumes were published in blue covers. They were part of ICRP's continuing review of background information to provide scientific bases for its recommendations; these did not imply recommendations for current action. The main recommendations were published in brown covers. This distinction between blue and brown covers is not followed at present. Since 1977 (starting from ICRP Publication 24), the ICRP brings out its reports in the journal *Annals of the ICRP*, published by Pergamon Press; the issues of *Annals of the ICRP* do not come out at regular periodic intervals, but as and when any ICRP report is ready. Several issues of the Annals may come out together, comprising a single ICRP publication.

Besides the regular reports, many of the earlier studies by ICRP's task groups or the study of consultants have appeared in various scientific journals from time to time. The list of ICRP publications to-date is given in Appendix 8.3.

APPENDIX 8.2

EVOLUTION OF ICRP RECOMMENDATIONS

EARLY HISTORY

It was in the early 1920s that the first limits concerning X-ray exposures were stated, and these were meant to avoid apparent injury. The International X-ray and Radium Protection Commission (the forerunner of ICRP) was formed in 1928. At this time the concept of 'tolerance dose' was accepted. The tolerance dose signified that radiation exposure which could be tolerated without causing obvious damage to the individual. The unit 'roentgen' was defined in 1928. Many studies led the Commission to recommend 0.2 R/day as the tolerance dose. (By this time national organizations in USA, England, Germany and Sweden had lowered the tolerance dose to 0.1 R/ day.)

FORMATION OF ICRP (1950) AND ICRP RECOMMENDATIONS (1950)

ICRP was formed in 1950. In its 1950 recommendations, ICRP replaced the concept of 'tolerance dose' or 'safe dose' by that of the 'maximum permissible dose' with the recognition that there could be some risk even at these levels. ICRP said: "Whilst the values proposed for maximum permissible exposure are such as to involve a risk which is small compared to the other hazards of life, nevertheless, in view of the unsatisfactory nature of much of the evidence on which our judgement must be based, coupled with the knowledge that certain radiation effects are irreversible and cumulative, it is strongly recommended that every effort be made to reduce exposure to all types of radiation to the lowest possible levels.....and that any unnecessary exposure be avoided." Note that even at this early stage ICRP had brought home the point of 'as low as possible'.

(How the ICRP arrived at the conclusion that the values proposed for maximum permissible doses are 'such as to involve a risk which is small compared to the other hazards of life' has not been spelt out. One had to

wait until ICRP 26 (1977) for explicit numerical comparison of radiation risks with other risks.)

In 1950 ICRP spelt out for the first time the various effects which were to be considered in making recommendations, namely:

(i) Superficial injury;

(ii) General effects on body, particularly the blood and blood-forming organs (e.g. anaemia);

(iii) Induction of leukaemia and other malignant tumours;

(iv) Other deleterious effects including obesity, impaired fertility and reduction of life-span;

(v) Genetic effects (This was the first time that genetic effects figured in the recommendations).

Among the important decisions of ICRP (1950) were:

(i) Replacement of 'tolerance dose' by 'maximum permissible dose';

(ii) Lowering of the maximum permissible dose from 0.2 R per day to 0.3 R per week;

(iii) Introduction of the concept of RBE; limiting neutron dose to one-tenth of that permissible for gamma radiation;

(iv) Consideration of internal hazards;

(v) Introduction of the concept of 'Standard Man';

(vi) Listing of maximum permissible concentrations in the body, air and drinking water for 10 radioisotopes.

THE PHASE 1950-1958

In 1955 the maximum permissible dose was officially defined as 'a dose of ionizing radiation, that in the light of the present knowledge is not expected to cause appreciable bodily injury to a person at any time during his lifetime. As used here, 'appreciable bodily injury' means any bodily injury or effect that a person would regard as being objectionable and/or competent medical authorities would regard as being deleterious to the

health and well-being of the individuals.' Further, the recommendations were extended to persons other than occupationally exposed workers.

In the latter half of the 1950s two different types of possible long-term somatic effects were recognized which could be considered in setting up permissible limits of exposure. The first type, of which leukaemia is an example, is a serious effect occurring in some individuals and therefore the aim of protection would be to reduce the incidence to the lowest possible levels. The second type, e.g., life-shortening, is an effect on every individual and therefore the aim of protection would be to reduce the degree of effect to the lowest practicable value. (It may be noted that these concepts are the vague forerunners of the two types of effects we now distinctly categorize, viz. stochastic and deterministic.)

ICRP RECOMMENDATIONS (1958) [ICRP PUBLICATION 1]

These considerations led to the important 1958 recommendations. The objectives of radiation protection are stated to be to prevent or minimize somatic injuries and to minimize the deterioration of the genetic constitution of the population. The permissible dose was defined as one 'which involves a risk that is not unacceptable to the individual and to the population at large.' (How to arrive at this decision as to 'what is not unacceptable' had been kept vague right from the earliest days of the ICRP recommendations till ICRP Publication 26 (1977), when a serious attempt was made to compare radiation risks with risks in other conventional occupations (in the case of occupational workers) and with risks, voluntary or involuntary, which the public is prepared to accept as part of modern life.) It was also clarified that the the maximum permissible dose is one which could be expected to produce effects 'that could be detected only by statistical methods applied to large groups'. (This concept is quite sound even today, provided perhaps if we replace 'could be detected' by 'could be detected, if at all').

ICRP (1958) distinguished between 'somatic' and 'genetic' injuries. Late somatic injuries include leukaemia and other malignant diseases, impaired fertility, cataract and shortening of life. ('Shortening of life' not attributable to cancers is not at present considered relevant at doses of interest in radiation protection.) The objectives of radiation protection were stated to be to prevent or minimize somatic injuries (There was no clear realization that deterministic effects can be prevented and stochastic effects minimized.), and to minimize the deterioration of the genetic constitution of the population. The 'permissible dose for an individual' is

'that dose, which, in the light of present knowledge carries a negligible probability of severe somatic or genetic injuries; furthermore, it is such a dose that any effects that ensue more frequently are limited to that of a minor nature that would not be considered unacceptable by the exposed individual and by competent medical authorities.' (There is again no clear understanding of the difference between stochastic and deterministic effects. Many of the concepts mentioned here have been either given up or radically modified. For instance, we do not speak at present of 'effects of a minor nature'.) 'Any severe somatic injuries (e.g. leukaemia) that might result from exposure of individuals to the permissible dose would be limited to an exceedingly small fraction of the exposed group; effects such as shortening of life-span, which might be expected to occur more frequently, would be very slight and would be hidden by normal biological variations.' (The concept enunciated in the latter half of the sentence regarding life shortening is not considered relevant today.) The permissible dose to the gonads for the 'whole population' is limited primarily by considerations with respect to genetic effects. It was again emphasized that 'the recommended maximum permissible doses are 'maximum' values; the Commission recommends that all doses be kept as low as practicable, and that any unnecessary exposure be avoided.' (Between the 1950 and 1958 recommendations, the phrase 'as low as possible' had been changed to 'as low as practicable'.)

Till 1956 permissible levels had been expressed in terms of a dose in a rather short interval of time (1 day or 1 week), that is, essentially in terms of an average dose rate. Implicitly it was assumed that if this average dose rate was low enough, no appreciable bodily injury would become apparent in the lifetime of the individual. The basic permissible dose (prior to 1958) was 0.3 rem/week. This corresponded to an accumulated 50-year dose of 750 rem and it was realized that this constituted a 'large' lifetime dose. It was however noted that the actual exposures of personnel were much below the existing permissible limits. The 1958 recommendations were expressed in terms of doses accumulated over a period of years, rather than in terms of a weekly dose.

With growing interest in atomic power plants, it was felt that the number of occupational workers as well as the potential exposure of the population as a whole would increase.

The concept of 'critical organ or tissue' was introduced; these were "those tissues that are most radiosensitive with respect to the ability of carrying out functions essential to the body as a whole". They were taken as the blood-forming organs, the gonads and the lens of the eye. The skin was not taken as a critical organ.

ICRP made it explicit that doses from natural background and from medical exposures were excluded from the maximum permissible doses. (This policy continues even now.) However, ICRP "recognizes especially the importance of the 'gonad doses' resulting from medical exposure and the attendant genetic hazard to the population" and recommended that "the medical profession exercise great care in the use of ionizing radiation in order that the gonad dose received by individuals before the end of their reproductive periods be kept at the minimum value consistent with medical requirements."

The 1958 recommendations covered the following categories of exposure:

A. Occupational exposure;

B. Exposure of special groups:

(a) Adults who work in vicinity of controlled areas;

(b) Adults who enter controlled areas occasionally;

(c) Members of the public who live in the neighbourhood of controlled areas.

C. Population at large.

Maximum Permissible Limits

A. *Occupational Exposure*

Maximum permissible accumulated dose to gonads, the blood-forming organs and lenses of the eyes at any age over 18 years:

$D = 5\ (N - 18)$, where D is dose in rem and N is age in years.

For a person occupationally exposed at a constant rate from age 18 years, this implies a maximum weekly dose of 0.1 rem which should be used for purposes of planning and design.

In one quarter (consecutive 13 weeks) the accumulated dose should not be greater than 3 rem.

Values for other organs are:	
Skin, thyroid, bone*	8 rem/13 weeks
Hands & forearms, feet and ankles	20 rem/13 weeks
Other organs	4 rem/13 weeks
Accidental exposure:	25 rem, once in a lifetime
Emergency exposure:	12 rem not too frequently;
but total should not exceed $D = 5\ (N - 18)$	

* The dose limit for bone was based on comparison with the maximum permissible body burden of ^{226}Ra.

B. *Special Groups*

Total annual dose to gonads, blood-forming organs and lens of eye:

Groups B(a) and B(b):	1.5 rem/year
Group B(c)	0.5 rem/year

C. *Population at Large*

No recommendation was made for exposure of individual member of the public but it was suggested that per capita genetic dose should not exceed 5 rem per generation excluding medical exposures and exposures due to natural background radiation. A linear non-threshold response was assumed for genetic effects.

As an illustration an apportionment of the genetic dose was given by ICRP as under:

Occupational exposure	1.0 rem
Special groups	0.5 rem
Population at large	2.0 rem
Reserve	1.5 rem

It was noted that at present the number of persons occupationally exposed in technologically developed countries was 0.1 to 0.2 % of the population, and that most of these persons received doses considerably less than the maximum permissible dose.

(Author's note; The concept of a 'critical group' was vaguely realized, though not explicitly formulated. The emphasis was on limiting the genetic dose to the population. This concept has now been abandoned.)

No specific recommendations were made regarding maximum permissible 'somatically' relevant dose to the population. It was indicated that the individual organ doses recommended for occupational exposure could be applied here also.

Some Points from ICRP Publication 2 'Report of Committee II on Permissible Dose for Internal Radiation' (1959)

This publication has discussed the basis for derivation of the maximum permissible body burdens (MPBB), and maximum permissible concentrations in air and water (MPC_a) and (MPC_w) for internally deposited radionuclides.

Two somewhat different criteria are commonly used in determining maximum permissible exposure values: (a) for bone-seekers like ^{90}Sr, ^{239}Pu, etc., which emit significant amounts of particulate radiation, the estimate is based on a comparison with ^{226}Ra and daughter products; and (b) for all other radionuclides, the MPC and body burden values are set to limit the weekly RBE dose received by the various organs of the body, e.g. 0.1 rem/week to the gonads and the total body, 0.6 rem/week to the skin and thyroid, and 0.3 rem/week to all other soft tissues. The first method is the result of a calculation designed to determine: (i) the amount (μCi) deposited in the bone that will deliver the same effective RBE dose as delivered by 0.1μCi of ^{226}Ra and its daughter products and (ii) the amount (μCi) deposited in the bone that will result in damage comparable to that observed from known deposits of ^{226}Ra in the bone. In some cases, this first method rests on rather extensive clinical experience or studies of biological damage. The method based on RBE dose rate is used generally when bone is not the critical organ or when direct experience is not available. The biological evidence supporting the limits on RBE dose to the various organs of the body is less direct than clinical observation or studies of biological damage, but is consistent with general experience involving radiation from both external and internal sources.

In 1941 an advisory committee of the National Bureau of Standards of USA first established the maximum permissible body burden for radium as 0.1 g (~ 0.1μCi). Humans have had years of experience with radium. Radium dial painters, patients treated medically with radium, and persons using public water supplies relatively rich in radium have furnished the best source of continuous human exposure from which to observe the effects of an internally deposited radionuclide.

ICRP PUBLICATION 6 (1962)

The changes were the following:

(i) The term 'Dose Equivalent' (DE) was introduced to take into account the differences in biological effectiveness of different radiations. A 'Quality Factor' (QF) related to the Linear Energy Transfer (LET) was to be used for this purpose. The quantity obtained by weighting the absorbed dose by the QF and other modifying factors is the dose equivalent. [The term Relative Biological Effectiveness (RBE) was to be used only for radiobiological experiments.];

(ii) Lens of the eye ceased to be a critical organ except for high LET radiation, and in the case of fast neutrons a modifying factor of 30 was used instead of 10;

(iii) Special consideration was given to occupational dose limits for women of reproductive age. The level of 3 rem/13 weeks normally allowed for gonad and whole body exposure was to be restricted to 1.3 rem/13 weeks, corresponding to 5 rem/year delivered at an even rate;

(iv) For pregnant women, after pregnancy had been established, the exposure should be such that the fetus may not receive more than 1 rem for the remaining period;

(v) The previous categories of exposure A and B were slightly revised to conform to the two classes of exposure used in the International Labour Office's Radiation Protection Convention, namely, (a) workers directly engaged in radiation work; and (b) workers not directly engaged in radiation work. In effect, this meant that the previous sub-categories B(a) and B(b) were merged, and the sub-category B(c) was dispensed with;

(vi) The definition of maximum permissible dose continued to be the same as in ICRP 1. The Commission stated that in recommending the maximum permissible dose, it has balanced the risk of the exposure against the benefit of the practice. As in ICRP 1, ICRP 6 reiterated its recommendation that all doses be kept as low as practicable, and that unnecessary exposure be avoided.

The revised maximum permissible doses (to gonads and blood-forming organs) were:

Occupational exposure $D = 5(N - 18)$

where D is the accumulated dose in rem in the gonads and blood-forming organs at any age above 18 up to age N years.

Adult workers in the vicinity of controlled area	1.5 rem/year
Individual member of the population at large	0.5 rem/year.

For other organs the 1958/1959 values were retained for occupational exposure. But for members of the population at large, the doses to other organs were limited to one-tenth of the corresponding occupational doses.

ICRP Publication 6 also gave a supplement to Report of Committee II on Permissible Dose for Internal Radiation (ICRP Publication 2).

ICRP PUBLICATION 9 (1965)

Some conceptual changes have been introduced in this publication. The objectives of radiation protection were stated to be 'to prevent acute effects and to limit the risk of late effects to acceptable levels'.

(Author's note: Although the terms 'deterministic' and 'stochastic' had not come into use in 1965, the objectives as stated here are the same as those explicitly spelt out in ICRP Publication 26 (1977), viz. to prevent deterministic effects and to limit stochastic effects.)

There was some change in the wording of the recommendation regarding keeping exposures to the minimum. It was stated: '..... the Commission recommends that any unnecessary exposure be avoided and that all doses be kept as low as readily achievable, economic and social considerations being taken into account.' (The wording has been changed from 'as low as practicable' to 'as low as readily achievable, economic and social considerations being taken into account'.)

The concept of 'risk' is dealt with in detail. Any exposure is assumed to entail a risk of deleterious effects. The 'acceptable dose' (with the same meaning as was implied by 'permissible dose') is that level of dose at which the assumed risk is deemed to be acceptable to the individual and to society in view of the benefits derived from activities involving exposure to radiation. While recognizing that the term 'maximum permissible dose' is not entirely satisfactory, ICRP decided to retain it.

Recommendations were made only for two categories of individuals, viz.

(a) Adults exposed in the course of their work;

(b) Members of the public.

It was explicitly stated that when populations are exposed, it becomes necessary to take into account not only the magnitude of individual risk but also the number of persons exposed, since the total burden is the sum of individual risks (even if it be small). (This is the forerunner of the concept of collective dose and collective detriment.)

Two distinct exposure situations were recognized, viz.

(a) Exposure from controllable sources (in which the occurrence of the exposure is foreseen);

(b) Exposure from uncontrollable sources.

In the former case, the term 'dose limit' is preferred as the dose can be limited in amount by control of the source, and by the development of proper operating procedures. In the latter case (an example of which is an accident) the exposure can be limited in amount, if at all, only by remedial actions. Under these conditions, it is no longer a matter of balancing an appropriate risk against any benefit. Instead, the question arises as to what remedial action may be available to limit the amount of exposure and increase the chances of recovery. Under such circumstances, the term 'action level' is more appropriate. (These concepts still hold good.)

Maximum Permissible Doses for Exposure from Controllable Sources

Occupational Exposure

Gonads and red bone marrow (and uniform irradiation of the whole body)	5 rem/year
Skin, thyroid, bone	30 rem/year
Hands and forearms; feet and ankles	75 rem/year
All other organs	15 rem/year

In a period of a quarter of a year, up to one-half of the annual dose limit (rounded upward to the next whole number) may be accumulated.

Within this framework some cases were considered separately, such as:

(a) Previous history unknown;

(b) Persons exposed in accordance with the former limits;

(c) Persons starting work at the age of less than 18 years;

(d) Exposure of women of reproductive capacity;

(e) Exposure of pregnant women;

(f) Planned special exposures.

Members of the Public

The annual dose limits for members of the public shall be one-tenth of the corresponding annual occupational Maximum Permissible Doses (except in the case of thyroids of children where the limit is restricted to 1.5 rem.)

Exposure of Populations

The genetic dose to the population should be kept to the minimum amount consistent with necessity and should certainly not exceed 5 rem.

Action Levels

No action levels have been suggested by ICRP as it has been considered to be the responsibility of the appropriate national authorities. However, the workers should be informed about the risks before they are asked to accept such exposures.

ICRP PUBLICATION 22 (1973) AND COST-BENEFIT ANALYSIS

ICRP 22 replaced the phrase 'as low as readily achievable' by 'as low as reasonably achievable'. It introduced the quantity 'collective dose' (previously called 'population dose') as a measure of the radiation-induced detriment. It also introduced the concept of cost-benefit analysis and gave the notation for such analysis. It suggested that it would be helpful to express the value of the collective dose in monetary units '...so that the advantage of reduction in dose can be compared directly with the detriment or cost of achieving this reduction.'

ICRP PUBLICATION 26 (1977)

ICRP 26 was indeed an epoch-making document in the annals of radiation protection, giving as it did a new philosophical and conceptual framework.

Among the major new concepts of ICRP 26 may be mentioned:

(a) Statement of the aim of radiation protection as being to prevent detrimental non-stochastic effects and to limit the probability of stochastic effects to levels deemed to be acceptable.

(b) Formulation of the basic tenets of the system of radiation protection as

(i) JUSTIFICATION: No practice shall be adopted unless its introduction produces a positive net benefit.

(ii) OPTIMIZATION: All (necessary) exposures shall be kept as low as reasonably achievable, economic and social factors being taken into account.

(Author's note: This would involve differential cost-benefit analysis.)

(iii) DOSE LIMITATION: The dose equivalents to individuals shall not exceed the limits recommended for the appropriate circumstances by the Commission.

(c) Explicit distinction between stochastic and non-stochastic effects.

(d) Introduction of the concept of Detriment.

(e) Introduction of the parameter 'Collective Dose Equivalent'.

(f) Enunciation that the recommended dose limitation is based on the principle that the risk should be equal whether the whole body is irradiated uniformly or whether there is non-uniform irradiation; introduction of tissue weighting factors for achieving this.

(Author's note: In the previous recommendations the dose limit was the same whether the whole body was irradiated or whether the gonads or the blood-forming organs alone were irradiated. This is clearly illogical, since in the case of whole body irradiation the risk is more than if the gonads or blood-forming organs alone were irradiated, because there will be additional risks from irradiation of other organs.)

(g) Detailed review of current knowledge of radiobiological effects, including the nature of dose-response relationships (particularly at the low doses and dose rates of interest in radiation protection); derivation of numerical risk coefficients for the induction of malignancies in various organs and of hereditary effects; enumeration of thresholds for important non-stochastic effects;

(h) Addition of hereditary detriment in the immediate offspring of the irradiated individual (i.e. in the first two generations of offspring) to the total of somatic detriment in setting up dose limits;

(i) Recognition that a valid method for judging the level of acceptability of risk in radiation work will be by comparing it with that for other occupations accepted as having a high standard of safety; consequently setting up of a limit of an annual mortality rate of about 1 in 10,000 for occupational workers;

(j) Drawing attention to the fact that apart from malignancies there will be very few injuries or illnesses in radiation workers; drawing attention to the further fact that in many conventional industries an accidental death involves an average loss of 30 years of life, and an approximately equal loss of working time from industrial accidents; on the other hand, a fatal malignancy from radiation would involve the loss of about only 10 years of life owing to the long latency in the development of the cancer, without appreciable associated time loss from accidents;

(k) Statement of the fact that genetic effects, while important, are unlikely to be of overriding importance;

(l) Attempt to set dose limit to members of the public on the basis of an annual fatality risk of 1 in 100,000 to 1 in a million as one which would be likely to be acceptable to any individual member of the public;

(m) For exposure of the populations, doing away with the genetic dose limit in view of the fact that past experience had shown that the earlier limit of 5 rem in 30 years is not likely to be reached, and that therefore its retention may lead to some laxity in protection enforcement;

(n) Recognition of types of exposure other than occupational and public, including:

(i) Exposures resulting from the use in everyday life of widely distributed products containing sources of radiation (such as electronic equipment that emit X-rays and those which contain radioactivity);

(ii) Exposure to practices in every-day life that cause an increase in the level of dose resulting from the natural background radiation (what is now called 'technologically enhanced natural background radiation') such as the use of certain materials in the construction of buildings and roads; high altitude flying; consumption of water and foodstuffs in which the concentration of natural radioelements is unusually high because of their origin, or has been enhanced, for example, by the use of fertilizers;

(o) Introduction of parameters for protection standards in operational radiation protection, including secondary limits, authorized limits, reference levels, investigation levels and intervention levels.

(p) Making a distinction, in the case of occupational workers, between Working Condition A (where the annual exposures might exceed three-tenths of the dose limits) and Working Condition B (where it is most unlikely that the annual exposure will exceed three-tenths of the dose limits).

A 'Controlled Area' is one where continued operation would give rise to Working Condition A and to which access is limited. A 'Supervised Area' is one with a boundary chosen so as to make it most unlikely that the annual dose limits outside this area will exceed one-tenth of the dose limits.

Annual Dose Equivalent Limits

A. *Workers*

(a) *Stochastic Limits*

$$\sum_T W_T H_T \leq H_{wb,\,L}$$

where W_T is a weighting factor representing the proportion of the stochastic risk resulting from tissue T to the total risk, when the whole body is irradiated uniformly, H_T is the annual dose equivalent in tissue T, $H_{wb,L}$ is the dose equivalent limit for uniform irradiation of the whole body, namely 50 mSv. The tissue weighting factors are given in Table App. 8.2.1.

(b) *Annual Non-stochastic Limits*

Lens of eye	0.3 Sv
All other tissues	0.5 Sv

(c) When external and internal exposures are received together, the stipulation is:

$$\frac{H_i}{H_{wb,L}} + \sum_j \frac{I_j}{I_{j,L}} \leq 1$$

where H_i is the annual dose-equivalent index, $H_{wb,L}$ is the annual dose-equivalent limit, H_j is the annual intake of radionuclide j, and $I_{j,L}$ is the annual limit on intake from radionuclide j.

(d) When women of reproductive capacity are occupationally exposed under the stipulated limits and when this exposure is received at an approximately regular rate, it is unlikely that any embryo would receive more than 5 mSv during the first two months of pregnancy. ICRP believes that this procedure will provide appropriate protection during the essential period of organogenesis.

(e) It is likely that any pregnancy of more than 2 months duration would have been recognized by the woman herself or by a physician. ICRP recommends that when pregnancy has been diagnosed, it should be ensured that the woman can continue to work only in Working Condition B.

B. *Individual Members of the Public*

(a) *Annual Stochastic Limits*

A lifetime dose that would correspond to 1 mSv per year of lifetime whole body exposure.

Whole body dose equivalent limit of 5 mSv in a year to critical groups. In ICRP's view this will provide the requisite degree of safety for the public under certain boundary conditions (which, it is reasonable to assume, are actually applicable in present day practices).

For non-homogeneous organ exposures (with organ doses H_T), the limitation is

$$\sum_T W_T \, H_T \leqslant H_{wb,\,L}$$

where $H_{wb,L}$ is the dose equivalent limit for uniform irradiation of the whole body for a member of the public (viz., 1 mSv per year).

(b) *Annual Non-stochastic Limits*

Any organ or tissue 50 mSv

C. *Exposure of Populations*

No recommendations. (Earlier genetic dose limit of 5 rem withdrawn.)

SOME FURTHER COMMENTS ON ICRP 26

(a) Note that the wording in the plea to reduce doses has been changed from 'as low as practicable' to 'as low as reasonably achievable, economic and social factors being taken into account'. ICRP does not choose to use the ubiquitous acronym ALARA.

(b) While discussing the extrapolation from human observations at high doses and high dose rates to the low doses and low dose rates of interest in radiation protection, ICRP 26 says: "It may be appropriate to reduce these (high dose/dose rate) estimates by a factor to allow for the probable difference in risk. The risk factors discussed later have therefore been chosen as far as possible to apply in practice for the purposes of radiation protection." But how exactly this reduction has been done is not specified.

(c) ICRP 26 has used only the absolute (additive) risk projection model and not the relative (multiplicative) risk projection model.

(d) In arriving at the dose limit for occupational workers ICRP could be faulted in taking recourse to the observation (gained as a result of operational experience in radiation work extending over 3 decades) that the 'average' doses to occupational workers were only one-tenth of the dose limit (of 5 rem/year) and calculating the corresponding fatality risk probability; this leads to the conclusion that radiation risk is comparable with that in other 'safe' industries, namely corresponding to an annual fatality risk of 1 in 10,000. More appropriately, computations should have been made for the maximum permissible dose limit. [This inconsistency has been duly taken care of in the new recommendations (ICRP publication 60).]

AMENDMENTS TO RECOMMENDATIONS OF ICRP 26 IN SUBSEQUENT MEETINGS OF THE COMMISSION

Subsequent to the publication of the 1977 recommendations, there have been further clarifications and amendments in 1978, 1980, 1983, 1984, 1985 and 1987. We shall note the major changes that pertain to the dose limits.

At the 1980 meeting of the Commission, the dose equivalent limit for the lens of the eye in the case of occupational workers was reduced from 0.3 Sv per year to 0.15 Sv per year.

The following quotation is from the statement from the ICRP meeting in 1985: "In the recommendation on effective dose equivalent limits for members of the public, made in its 1977 Recommendations, two values were mentioned. The use of the limit of 5 mSv in a year was endorsed, but only under (certain) conditions. For other circumstances the Commission recommended that it would be prudent to limit exposures on the basis of a lifetime average annual dose of 1 mSv." ..... "The Commission's present view is that the principal limit is 1 mSv in a year. However, it is permissible to use a subsidiary dose limit of 5 mSv in a year for some years, provided that the average annual effective dose equivalent over a lifetime does not exceed the principal limit of 1 mSv in a year."...."The recommended (non-stochastic) dose equivalent limit for both the skin and the lens (as well as for other organs) is still 50 mSv in a year for members of the public."

COMPARISON OF ICRP 26 AND ICRP 60

A. *Terminology and Definitions*

(a) 'Non-stochastic effects' are now called 'deterministic effects';

(b) 'Quality factor' Q is now called 'radiation weighting factor' W_R. Q was applied to the dose at a point, while W_R is now averaged over the tissue or organ. Numerical values of the radiation weighting factors are substantially different in some cases from the corresponding Q values for the same type of radiation;

(c) 'Dose equivalent' is now called 'equivalent dose' H_T. It is the absorbed dose averaged over a tissue or organ weighted by the radiation weighting factor. In ICRP 26 a 'modifying factor' N was introduced in addition to the quality factor Q (although this did not have a value different

from 1); this factor has been dispensed with in ICRP 60;

(d) The concept of 'tissue weighting factor' W_T is the same as in ICRP 26 but it was not given this name earlier;

(e) 'Effective dose equivalent' E is now called 'effective dose' E;

(f) Numerical values of W_T were given for 6 organs in ICRP 26, the rest of the organs being clubbed under 'the remainder'. ICRP 60 gives W_T values for 12 organs; consequently the organs relegated to 'remainder' earlier are much less now. Values of W_T are substantially different for many organs than those given in ICRP 26.

B. *Biological Effects of Radiation*

A lot more data have become available after ICRP 26 on the biological effects of radiation.

In particular, the risk estimates have been changed substantially as a result of (i) revised dosimetry of the Hiroshima-Nagasaki survivors; (ii) longer follow-up period (1950-1985) allowing solid cancers with long latent periods to manifest themselves (perhaps not fully yet); (iii) use of the multiplicative risk projection model for most cancers (as against the additive model which had been used by ICRP 26, but which is now restricted to leukaemia and bone cancer); (All the above 3 factors have increased the risk estimates substantially.) (iv) use of a dose and dose rate reduction factor (DDREF) of 2 to extrapolate risk estimats from high doses and high dose rates to doses below 0.2 Gy and dose rates below 0.1 Gy/h for low LET radiations.

There has been substantial addition to our knowledge of the various types of effects of antenatal exposure since ICRP 26, and this has been utilized in ICRP 60 risk estimates.

C. *Changes in Dose Limits*

Dose limits have been brought down significantly for occupational exposure (from 50 mSv for the annual effective dose to 20 mSv). The dose limit of 1 mSv per year for the public has been retained.

As per the present dose limits and revised risk coefficients (multiplicative model), the annual probability of radiation-induced cancer death would be 0.8 per 1000 for an occupational worker and 6 in a million for a member

of the public for exposures at the dose limits. The loss of life expectancy if cancer death occurs is around 13 years and the most probable age at attributable death is 78 years in both cases.

There has been some change in the case of occupational exposure of women. No special limits have been recommended, but once pregnancy has been declared, a supplementary dose limit of 2 mSv to the surface of the abdomen for the remainder of the pregnancy has been prescribed.

D. *Conceptual Framework of Radiological Protection*

The concepts of justification, optimization and individual dose limits have been retained.

A distinction is made between the systems of protection for (a) proposed and continuing practices (new terminology used in ICRP 60) and (b) intervention.

'Potential exposures' are to be considered as part of the assessment of practices (but they may also lead to intervention).

Distinction is made between 'source-related' and 'individual-related' systems of protection.

The concepts of 'risk constraints' and 'risk limits' have been postulated for potential exposures.

ICRP 60 explicitly recognizes 3 types of exposure, viz. occupational, medical, and public. (This was also there in ICRP 26.) The present definition of medical exposure is different in one important respect; it runs as follows: 'Medical exposure is confined to exposures incurred by individuals as part of their own medical diagnosis and treatment, *and to exposures (other than occupational) incurred knowingly and willingly by individuals helping in the support and comfort of patients undergoing diagnosis or treatment.*' (The underlined category was not included in the earlier definition.)

While the classification of working areas into 'controlled' and 'supervised' areas has been retained, the classification into working conditions A and B has been abolished.

E. *Bases for Estimates of Detriment*

Detailed attention has been given in ICRP 60 to the bases for judging the significance of the effects of radiation, including an elaborate discus-

sion of the meaning and expression of 'risk'and 'detriment', and definitions of a number of mathematical terms related to quantitative evaluation of risk. Calculations have been made for each of the parameters for radiation exposures of given magnitude and pattern. The detriment evaluation takes into account the probability of death, time lost if death occurs, reduction of life expectancy (a combination of the first two attributes), morbidity due to non-fatal cancers weighted for severity and for the period of life lost or impaired, and weighted contribution from hereditary effects. (ICRP 26 took into account only fatal cancers and serious hereditary effects in its evaluation of the detriment.)

Regarding hereditary effects, ICRP 26 added the hereditary risk to the first and second generation offspring to the stochastic risk to the exposed individual, the effects in later generations being considerd as part of the consequences for society. ICRP 60 now attributes the whole hereditary detriment to the detriment suffered by the exposed individual, thus avoiding the need for a two-stage assessment.

Table APP. 8.2.1: Tissue weighting factors
[from ICRP 26 (1977)]

Tissue	W_T
Gonads	0.25
Breast	0.15
Red bone marrow	0.12
Lung	0.12
Thyroid	0.03
Bone surfaces	0.03
Remainder*	0.30

* $W_T = 0.06$ for each of the 5 organs of the remainder receving the highest dose; exposure of remaining tissues can be neglected. (When the gastro-intestinal tract is irradiated, the stomach, small intestine, upper large intestine and lower large intestine are treated as four separate organs.)

APPENDIX 8.3

LIST OF ICRP PUBLICATIONS

X-ray and Radium Protection. Recommendations of the 2nd Congress of Radiology, 1928. Circular No. 374 of the Bureau of Standards, US Government Printing Office (January 23, 1929), *Br. J. Radiol.*, **1**, 359-63 (1928).

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Note:

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\# deontes publications that are out of print.

Part II

Biological Effects of Radiation

CHAPTER 9

BASIC RADIOBIOLOGY

The material presented in this chapter is mainly based on Annex B of ICRP 60, 'Biological effects of ionizing radiation'.

9.1 INTRODUCTION

Soon after the discovery of X-rays in 1895 and of natural radioactivity in 1896, clinical evidence, mainly from effects on the skin, indicated that ionizing radiation is harmful to human tissues. With the rapid increase in applications of nuclear energy following the discovery of nuclear fission in 1939, the problem of protection from the harmful effects of radiation, while maximizing the benefits, became increasingly important. Today we have a wealth of information concerning the biological effects of radiation, possibly greater than that associated with any other environmental hazard.

In the present and succeeding chapters we shall discuss in some detail the biological effects of radiation (which have already been briefly discussed in Chapter 3) with special reference to stochastic effects (mainly induction of cancer and hereditary effects) and deterministic effects, broadly in relation to radiation protection. Many effects such as those in radiation therapy and the late deterministic effects on specific tissues, such as fibrosis, which may result from such treatment, are not discussed here. This introductory chapter is devoted to basic radiobiology.

9.2 INTERACTION OF RADIATION WITH MATTER

We have already discussed the basic mechanisms of ionization and excitation. Directly ionizing particles such as alpha and beta rays produce ionization and excitation in the matter through which they pass as a result of electrical interactions between the radiations and the electrons in the atoms and molecules of the material. Indirectly ionizing radiations include X-rays, gamma rays and neutrons. In the case of X- and gamma rays, the photons eject electrons from atoms by the photoelectric, Compton and pair production effects. These secondary electrons can then produce

ionization and excitation in the matter through which they traverse. Neutrons eject protons and other charged particles from nuclei and cause the nuclei to recoil; both the ejected proton and the recoil nucleus can produce ionizations and excitations.

When ionizing radiations traverse a medium, the resulting electric interactions are random and follow the often haphazard tracks of the charged particles (primary or secondary) bouncing from one interaction event to another. At low doses only a small number of the atoms of the medium will be ionized or excited. Each of the interaction events involves the transfer of a small amount of energy (in packets of around 100 eV for low LET radiations) from the radiation to the medium. These transfers occur in a very short time ($< 10^{-16}$ s) but may be broadly distributed spatially in a discontinuous fashion. The average energy deposited along the track of the particle per unit length ('linear energy transfer', LET), which is related to the number of interaction events per micron of track, depends upon the type of particle and its energy.

The actual energy lost by a charged particle is subject to random fluctuations. The energy transferred to an atom or to a small volume like a biological cell has an average value and a distribution around this value. The average energy transferred per unit mass of medium is the absorbed dose.

The transfer of energy gives rise to physico-chemical processes such as the induction of free radicals (in $\sim 10^{-12}$ s); the free radicals can move rapidly some distance from the site of the original event and cause further chemical changes in the molecules of the medium before they are inactivated ($\sim 10^{-6}$ s). Molecular changes reflecting the breakup of chemical bonds can manifest themselves over varied periods of time and in a variety of ways.

9.3 BIOLOGICAL STRUCTURE AND FUNCTION

The basic unit of the living organism is the cell, its nucleus containing genetic information in nuclear DNA that is capable of providing instructions for cellular reproduction and intracellular protein synthesis. A 'milieu interior' is maintained within the cell and in relation to its extracellular environment, aided by a complex system of semipermeable membranes which regulate movement of water, nutrients and electrolytes in and out of the cell. Any disturbance of this equilibrium can threaten the cell's viability but the cells have evolved an elaborate system of repair processes, particularly for damage within the nuclear DNA.

In higher organisms, cells are organized into tissues and organs with specialist roles, whose functions are coordinated by the nervous and endocrine systems.

9.3.1 DNA Damage and Repair

Biological structures can be altered directly or indirectly (through free radicals) through events set in motion by the transfer of energy from ionizing radiations to the medium. Most vital is the damage to DNA (single or double strand breaks, recombinational changes, cross-links, alterations in sugar and base fractions, base substitutions, deletions, etc.). Chromosomal aberrations are also a result of DNA damage.

Many of the acute effects observed in the intact organism are mediated through the death of cells when they attempt to divide (cell reproductive death). The organism has a set of complex enzyme-mediated repair systems which are specific for different types of DNA damage, whereby the lesions induced in DNA by ionizing radiation, ultraviolet light and chemical agents are identified and removed, often in tens of minutes. When a single strand break occurs, the site of damage is identified, and the break repaired by annealing of the broken ends. If base damage occurs on the single strand, enzymatic excision occurs and the intact complementary DNA strand provides a template upon which to reconstruct the correct sequence of bases. These damages are removed with high fidelity (error-free repair), and there is no long-term consequence of the lesion. Error-prone repair processes may lead to small base changes (point mutations) at the site of the molecular lesions or more gross changes such as gene deletions or rearrangements; these may have long-term consequences for the cell and can result in reproductive death or stable genetic changes in the surviving cells (Friberg and Hanawalt, 1988).

Double strand breaks may also be repaired by simple annealing. But if base damage has occurred simultaneously in both the strands, the consequences are more serious since a template will not be available for reconstructing the sequence on either strand. The outcome could be cell reproductive death, or misrepair reflected in a point mutation or more extensive gene deletion. Increased frequency of misrepair of DNA double strand breaks has been observed in radiation-sensitive strains of cultured mammalian cells known to be deficient in DNA repair enzymes. The fidelity of DNA repair may be a major factor that determines the response to variable dose rate and radiation quality (Debenham *et al.*, 1987).

9.3.2 Cell Killing

The killing of somatic cells, resulting from irreparable damage to vital cell structures such as the chromosomes, becomes manifest in rapidly dividing cell populations in a few hours or days. In slowly dividing cells, death may not occur for months or years. The degree of cell killing increases with the dose. If enough cells are killed in an organ or tissue, its function is impaired, and in extreme cases the organism itself may die. In addition to alterations in the function of organs and tissues due to cell killing, functional disorders can also result from alteration of cellular processes such as membrane permeability and cell-to-cell communication.

9.3.3 Cell Modification

Ionizing radiations (as well as several other agents) can also lead to modifications in the DNA structure which leave the cell viable but with the potential for a neoplastic transformation, i.e. capability of the transformed cells for unlimited cellular proliferation. This change alone does not constitute a 'malignant transformation' (i.e. the ability of the cells to multiply and form tumours when injected into recipient animals), since other phenotypic changes (such as altered behaviour of cells in cell-cell interactions and the invasion of neighbouring tissues and metastasis to distant sites) also occur in malignant transformations. The multiple changes that occur in the development of a cancer proceed in sequential stages. The initial events in the genome and the production of a cell or cells with the potential to develop into a cancer are known as initiation. Both endogenous and exogenous factors may influence expression of the initial event. The initiated cell(s) must undergo further changes, usually after a long time and possibly after stimulation by a promoting substance, before becoming a cell with malignant potential. (One theory postulates that a further step, 'conversion', would be required before the promoted 'precancerous' cell becomes 'cancerous'.) Thereafter the division and multiplication of this cell give rise to an occult tumour in the 'progression' stage. The carcinogenic process including the growth of a primary cancer to a detectable size (about 1 cm diameter and containing billions of cells) and its spread to other tissues can take months in small animals and years in humans. The interval between exposure and the detection of a radiation-induced cancer is the 'latency period'. This period varies with the type of cancer and the age at exposure.

Changes in the genome, compatible with continued cell division, may also take place in the germinal tissues of the reproductive cells. They result in a variety of transmissible lesions, most often deleterious, which

are passed on to and may be manifest as hereditary disorders in succeeding generations.

9.3.4 Tissue Response to Cell Modification

Most neoplastic cell transformations do not progress to cancer due to a combination of circumstances:

- Virtually no unrepaired cells remain viable after more than a few divisions;
- Those capable of several divisions are frequently 'programmed' to differentiate into non-dividing functional cells;
- The required sequence of promotion and progression events in the cell's environment does not occur;
- Host defence mechanisms (immuno-surveillance, natural killer cell activity) exist to prevent selective cloning.

9.4 STOCHASTIC AND DETERMINISTIC EFFECTS

Since deposition of energy by radiation is a random process, even at very low doses it is possible that sufficient energy may be deposited into a critical volume within the cell to result in its modification or even its death. Hence there may be no threshold for these effects. Death of a few cells may have no consequences in tissue, but modifications in single cells may lead to malignancies or hereditary effects (stochastic effects). There may be no threshold for such effects. As the dose is increased, the frequency of such events increases but not the severity of the resultant changes.

With larger doses there will be substantial killing of cells, sufficient to result in detectable tissue changes. Although other mechanisms may be involved, cell killing plays a crucial role in the pathogenesis of tissue injury. Hence the response of tissues *in vivo* is determined by the characteristics of cell survival. For any defined non-stochastic injury, a given population of cells must be killed in order to reach the level of detection threshold, as shown in Fig. 9.1. The threshold dose can be taken as the dose that will cause the effect in at least 1-5% of the exposed individuals (ICRP 41, 1984). It will depend upon the sensitivity of the method for determination of the damage. (The tolerance dose is the maximum dose which a tissue can stand without developing a clinically detrimental ef-

fect.) The available knowledge about deterministic effects in human beings derives largely from radiotherapeutic experience.

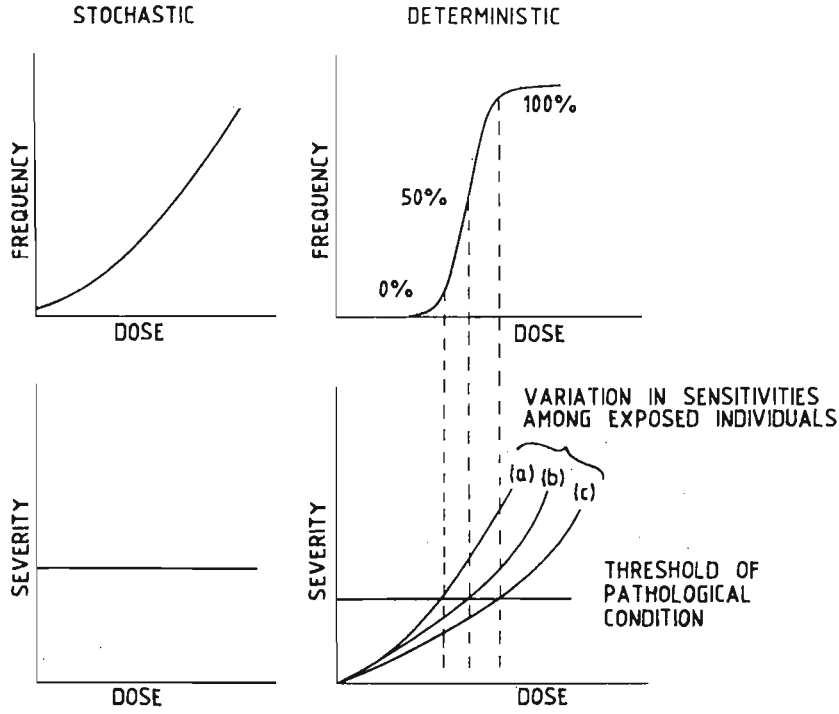

Figure 9.1. Typical dose-effect curves for stochastic and deterministic effects (from ICRP 41).

Since cell killing by radiation is itself a stochastic process, the term 'non-stochastic injury' resulting from the death of a large number of cells is not suitable, and ICRP now calls these as 'deterministic effects', meaning 'causally determined by preceding events'.

9.4.1 Deterministic Effects

Here the amount of cell killing is not compensated for by proliferation of viable cells. The severity of the effect depends on the dose and there will be a threshold below which the loss of cells is too small to detectably impair function. Examples of deterministic effects are: cataract of lens, non-malignant damage to skin, cell depletion in bone marrow causing haematological deficiencies, and gonadal damage leading to impairment

of fertility. In addition to cell killing there may be interference with tissue functions, including regulation of cellular components, inflammatory reactions involving modifications in cell permeability, and natural migration of cells in developing organs. Indirect functional effects include influence of pituitary irradiation on other endocrine functions. Deterministic effects can be manifested early (within weeks) or late (taking months to years).

More detailed discussions of deterministic effects can be found in Chapter 10, sections 1 and 2 [which summarize ICRP publication 41, 'Non-stochastic effects of ionizing radiation' (1984) and ICRP publication 58, 'The RBE for deterministic effects' (1989) respectively].

9.4.2 Cell Killing and In Vitro Survival Curves

Cell killing is the main, but not the only, process involved in deterministic effects. Unless the dose is high (many Gy), many cell types are not killed immediately after exposure but continue to function until they attempt to divide. While individual cell death is random, the composite effect of killing a high proportion of cells is deterministic.

The dose required to destroy a cell's reproductive integrity is much less than that required to destroy its metabolic or functional activity (Rubin and Casarett, 1968; Hall, 1988). The first observable effect of irradiation is a delay in the entry of cells into mitosis, resulting from their arrest in the G_2 phase of the cell cycle. The duration of the delay is dose dependent. A dose of 10-15 Gy delays division by the duration of one division cycle in mammalian cells (Denekamp, 1975). Some irradiated cells may die at the second or third divisions, especially at lower doses. In a few types of cells (e.g. small lymphocytes and oocytes) death normally occurs during interphase, before any attempt at division. Mature cells are relatively radioresistant.

Studies of cultured mammalian cells subjected to irradiation enable us to draw 'survival curves' (Fig. 9.2). For high LET radiations the dose-response curve is exponential, i.e. linear on a semi-log plot (Fig. 9.2(a)) characterized by a single parameter, viz. the slope, which is usually represented by its reciprocal, the dose D_0 required to reduce survival to 37% (37% is 1/e). For high LET radiations, we may write:

$$S(D) = \exp.\ (-D/D_0)$$

where $S(D)$ is the fraction of cells surviving after a dose D, and

D_0 is dose at 37% survival, or the reciprocal of the slope.

In the 'target theory' of radiobiology, this is interpreted to mean that there is only one sensitive target in the cell which, if hit, would lead to death. It can be shown that $1/D_0$ gives a measure of the dimensions of the subcellular target.

For low LET radiations, the curve (Fig. 9.2(a)) has an initial shoulder followed by a straight portion on the semilog plot. The curve is characterized by two of the 3 parameters: D_0 (dose required to reduce survival to 37% on the exponential part of the curve, i.e. the reciprocal slope of the straight portion of the curve); the extrapolation number n at zero dose; and D_q, the quasi-threshold dose, being the intercept of the straight portion of the curve on the dose axis (Fig. 9.2(a)).

For low LET radiation, this curve can be represented by

$$S(D) = 1 - [1 - \exp.(-D/D_0)]^n$$

For mammalian cells n is in the range 2-20 and D_0 in the range 1-2 Gy for low LET radiation. A more complex expression, describing the initial slope of the curve, is given by

$$S(D) = \exp.(-D/D_1).[1 - (1 - \exp.(-D/D_0)^n],$$

where D_1 is the reciprocal of the initial slope of the curve.

The 'target theory' interprets this equation as follows. It is postulated that there are n targets each of which has to be hit at least once to kill the cell (multi-target single-hit model).

The initial region between 0 and 5 Gy (and often over a broader dose range) can be described by a linear-quadratic equation

$$F(D) = \alpha D + \beta D^2,$$

and $$S(D) = \exp.(-\alpha D + \beta D^2),$$

where F is the frequency of lethal effects. This is shown in Fig. 9.2(b). The linear coefficient α (which has the dimensions of dose^{-1}), is in the range of 0.1 to 0.5 Gy^{-1} and β, the quadratic coefficient (with the dimensions of dose^{-2}) between 0.1 and 0.05 Gy^{-2}. The ratio α/β which has the dimensions of a dose and represents the dose at which the linear and quadratic components contribute equally to the damage, is in the range of 1 to 10 Gy (Hall, 1988). The linear term corresponds to a single hit (all

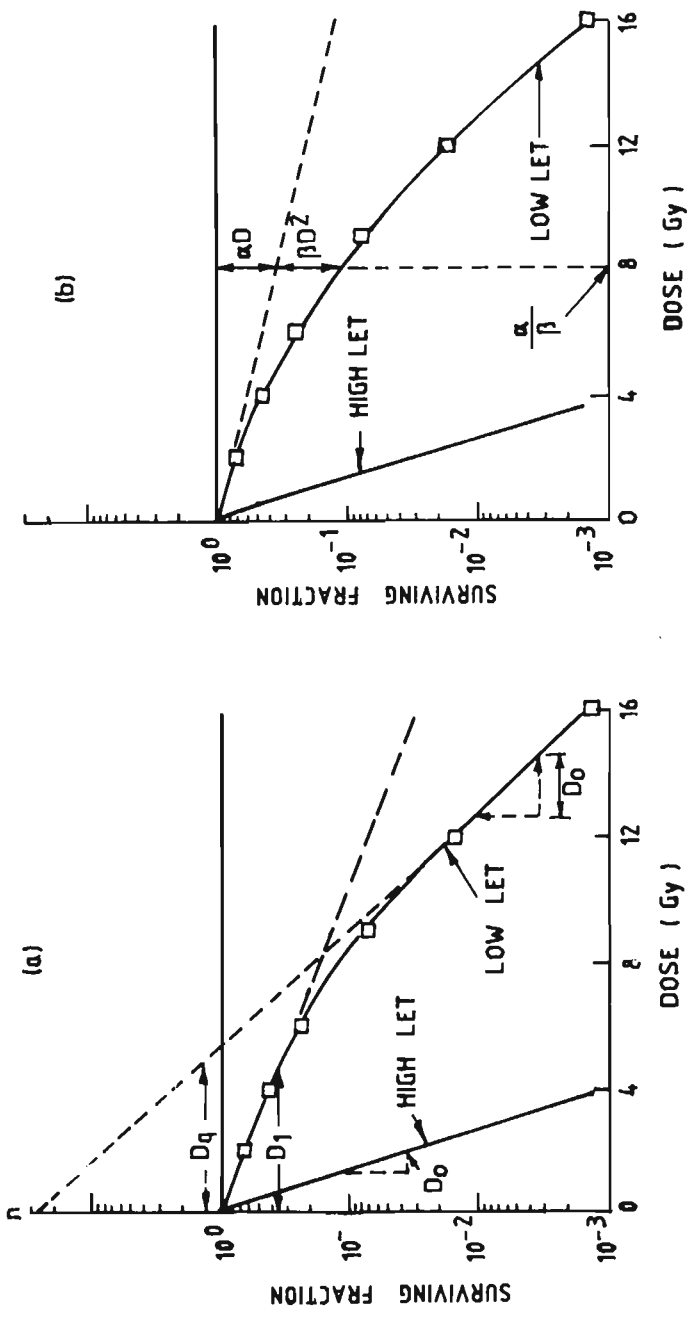

Figure 9.2. Survival curves for mammalian cells exposed to high LET and low LET radiation at high dose rates (> 0.1 Gy/min). The fraction of surviving cells is plotted on a logarithmic scale against dose on a linear scale (from ICRP 60).

or none effect on cell survival) and the quadratic term denotes effects resulting from an interaction between two separate but closely spaced intracellular lesions.

The initial increase in the slope with increasing dose in the survival curve for low LET radiations is interpreted as demonstrating that cells require to accumulate a certain number of damaging events within a short time for the cumulative effect to be lethal to the cell.

Tissues vary widely in the rates at which their constituent cells are normally replaced and in the population dynamics through which the production, differentiation, aging and loss of such cells occur. These differences affect the rapidity with which different tissues manifest the effects of radiation, since the expression of radiation injury in most cells is delayed until mitosis. Rapidly dividing tissues, in the main, have a defined stem cell compartment which gives rise to a proliferating cell compartment, and, in turn, to compartments of differentiating and functioning post-mitotic injury. The timing of radiation-induced injury depends on the life-span of the mature cells, which are comparatively radio-resistant, and it is thus relatively independent of dose. With fractionated or protracted exposures, proliferation of stem cells may compensate for cell killing and reduce the effectiveness of the radiation. Examples of rapidly proliferating tissues include the epithelium of the intestinal mucosa, the bone marrow, the gonads, and the epidermis (ICRP 41, 1984).

Other types of tissue do not have defined stem cells, and, in the main, are characterized by low cellular proliferative capacity. In such tissues, the timing of a response to radiation, although dose-dependent, may not occur until long after small doses. Far less protection by regenerative or compensatory proliferating is to be expected in tissues of this type, e.g. the liver, where parenchymal cell turnover is low, or blood vessels, where endothelial cell turnover is also low (ICRP 41, 1984; Wheldon *et al.*, 1982). Cells vary in sensitivity to cell killing, division delay and other progression changes during the cell cycle (Sinclair, 1968). Consequently, the surviving population will consist initially of mainly resistant cells but the distribution of cells at each cell cycle stage is modified. At the same time, damage in such cells is being repaired, and other undamaged cells will repopulate the tissue. Eventually, if the dose is not too large, the tissue should recover completely. Reduction of the volume of tissue irradiated considerably reduces the damage.

The dose-response relation is sigmoid (Fig. 9.1). The curve represents the frequency of the pathological conditions in a population (i.e. all sub-

groups with varying degrees of sensitivity as shown in the lower part of the figure). The severity will increase more steeply for the sensitive subpopulation for whom the threshold dose will also be lower. The frequency reaches 100% at a dose which exceeds the threshold of severity for all members of the population.

Various intrinsic and extrinsic factors (including degree of differentiation, rate of cell proliferation, age at irradiation, oxygen tension in the cell, temperature, genetic factors, physiological conditions, hormone balance, chemicals, stress, and injury) modify the radiation response.

9.4.3 Effects of Fractionation and Protraction of Irradiation

When a dose is split into two or more fractions or if it is delivered at a low dose rate, its biological effectiveness is usually reduced. The two main factors contributing to repair are 'repair of sublethal damage' (SLD) and replacement of lethally injured cells by repopulation. Other factors include recovery from 'potentially lethal damage' (PLD), 'slow repair', and cell replacement by migration of unirradiated cells (Withers, 1969; UNSCEAR, 1982).

If time elapses between exposures and thus between events, repair of 'sublethal damage' can occur and more radiation will be required to kill the same number of cells. This repair was demonstrated in experiments in mammalian cells involving two doses of radiation separated by intervals of time (Elkind and Sutton, 1960). A measure of the extent of accumulation and repair of sublethal damage during the interval between exposures is indicated by the magnitude of the extrapolation number n, or by the difference (D_2 - D_1) between the dose required to kill a given percentage of cells or cause a given level of injury when the radiation is delivered in two exposures, D_2, and the dose required to kill the same percentage of cells or cause the same level of injury when the dose is delivered in a single exposure, D_1 (Fig. 9.3). When the irradiation is given in many fractions, repair of sublethal injury occurs after each successive dose, and the multi-fraction survival curve is of the form shown in Fig. 9.3. Dose rates below 0.1 Gy/min of low LET radiation result in progressively less cell killing until a dose rate of about 0.1 Gy/h or less is reached for mammalian cells, at which point all the sublethal damage has been repaired and only cell death is the damage producing agent (Hall and Bedford, 1964).

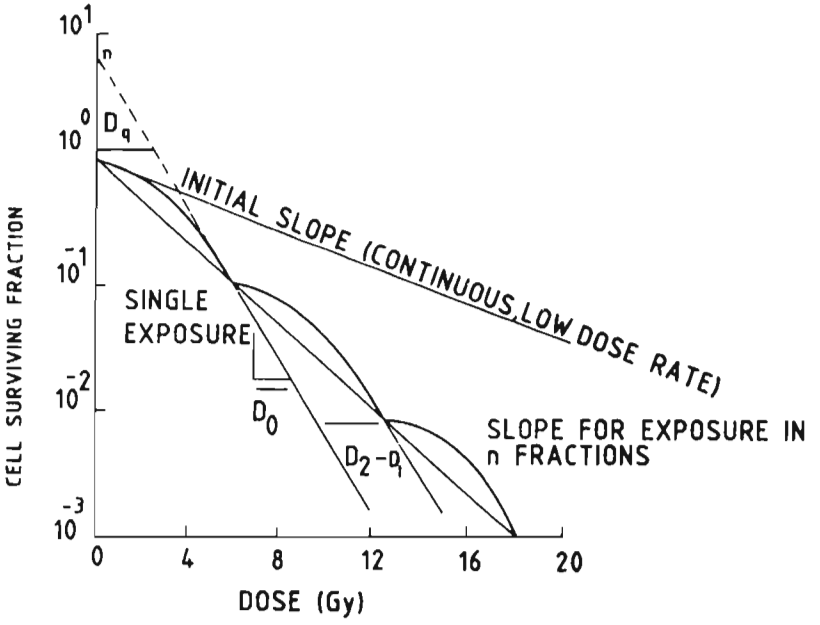

Figure 9.3. Single dose and fractionated dose cell survival curves following exposure to low LET radiations (from ICRP 41).

Irradiation causes a dose-dependent mitotic delay, after which there is renewed cellular proliferation. At a sufficiently small dose rate, cell replacement can fully counterbalance cell loss, depending upon the proliferative capacity of the cell type.

9.4.4 Iso-effect Relationship

The effect of fractionation on the severity of tissue damage is of relevance in radiotherapy (in relation to levels of healthy tissue tolerance), as discussed in ICRP publication 44 (1984), but not much in radiation protection.

A widely used empirical formula, due to Ellis (1968), for low LET radiation gives the relation between tissue tolerance and the pattern of dose fractionation.

$$\text{Total dose} = (\text{NSD})\, N^{0.24}\, T^{0.11}$$

where N is the number of exposures, T the overall treatment time in days, and NSD, the 'nominal standard dose' (tissue tolerance dose for brief

exposures). NSD can be taken to be the maximum dose that can be tolerated if given in a single exposure.

Another formula, due to Kirk *et al.* (1971), applicable for low dose rates, is

$$D_{inst.} = 0.8\dot{D}T^{0.71}$$

where $\dot{D}$ is the constant dose rate in Gy/day and $D_{inst.}$ is the equivalent acute dose.

We may infer from the linear-quadratic equation that the ratio α/β (which represents the dose at which the αD and βD^2 components contribute equally to the damage) is a useful parameter for describing the effects of fractionation and dose rate. The ratio α/β varies from 1 to 15 Gy, depending upon the type of tissue and particular response. Values below 6 apply to slowly proliferating tissues which give rise to late effects and higher values to rapidly proliferating tissues which give rise to early effects (Barendsen, 1982; Withers *et al.*, 1980).

With increasing LET the slope of the survival curve becomes steeper, and accumulation of sublethal injury contributes relatively less to lethality. Cell repopulation appears to be independent of LET. With high LET radiation, sublethal damage is repaired to a smaller extent between successive exposures. The formulas given above will therefore have to be suitably modified.

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CHAPTER 10: SECTION 1

DETERMINISTIC EFFECTS IN DIFFERENT ORGANS AND TISSUES

10(1).1 INTRODUCTION

This section is a summary of the deterministic effects in different organs and tissues as discussed in ICRP publication 41, 'Non-stochastic effects of ionizing radiation' (1984), supplemented by information from ICRP publication 58, 'RBE for deterministic effects' (1989), and ICRP 60. ICRP publication 44, 'Patient protection in radiotherapy' (1984) also briefly discusses some aspects of the subject. Section 2 of Chapter 10 discusses in a more detailed manner that part of ICRP 58 dealing specifically with the RBE aspects.

Much new information on deterministic effects is beginning to emerge from the unfortunate experiences during the Chernobyl accident. These include: cytogenetic studies of the doses received by the highly exposed group (Pyatkin *et al.*, 1989); haematological effects (Guskova and Baranov, 1989); and skin effects (Barabonova and Osanov, 1990).

10(1).2 SKIN

The main hazard is from beta particles and low energy X-rays. Alpha particles do not pose a hazard as they do not have enough penetration to irradiate cells of basal layers. Depths of concern are 300-500 μm (NCRP, 1989).

Skin reactions include a variety of effects, the frequency, severity and timing of which depend on the conditions of exposure. The following is the chronology:

Early Phase: Starts within hours and lasts a few hours. Transitory erythema (reflecting dilatation of capillaries resulting from histamine-like substances released by injured epithelial cells); the higher the dose, the earlier is the time of appearance of erythema.

Second Phase: Starts at 2-4 weeks. One or more waves of deeper and more prolonged erythema (threshold 3-5 Gy);

With increasing dose, epilation, dry desquamation, moist desquamation, cell death in epithelial and dermal layers resulting in necrosis (threshold 50 Gy);

Pain is often marked at the stage of exudataive radiation dermatitis and desquamation;

After a single exposure at a very high level, the initial erythema appears in a few minutes; then it will disappear and reappear several times, without any real interval between them; the final erythema produces a more sensitive reaction (ICRP 28, 1978);

Long-term: Takes months to years to manifest.

Changes in pigmentation, atrophy of epidermis, sweat glands and hair follicles, fibrosis of dermis, susceptibility to trauma and chronic ulceration, damage to vasculature (including telangactesia); threshold 30-40 Gy (Reinhold *et al.*, 1989).

Damage to germinal cells in the basal layers is critical to the pathogenesis of erythema and desquamation. Acute ulceration results from interphase death of fibroblasts and vascular epithelial cells, as can be seen with irradiation from 'hot particles'. Acute epithelial necrosis is caused by interphase death of post-mitotic suprabasal cells in the epidermis after exposure to low energy beta particles. With beta rays, the threshold dose for a visible reaction increases with decreasing field size (Wells *et al.*, 1982). In human skin, the threshold for low LET radiation in a 10 cm^2 field varies from 6-8 Gy delivered in a single brief exposure to more than 30 Gy delivered in highly protracted or fractionated exposure. For dry desquamation, moist desquamation and necrosis the thresholds are higher and they increase similarly with fractionation and protraction. The threshold for damage to hair follicles is lower than for erythema. The values are 3-5 Gy acute dose for temporary epilation and 7 Gy for permanent epilation; 50-60 Gy for protracted or fractionated exposure (Rubin and Casarett, 1968; UNSCEAR, 1982).

The dose D of low LET radiation to produce a given level of damage increases as the inverse cube root of the diameter:

$$D \propto (L)^{-0.33}$$

where L is the average diameter of the area (Cohen, 1966; Eads, 1972).

Different anatomic sites vary in decreasing order of sensitivity as follows: Anterior aspects of neck, antecubital and popliteal areas; anterior surfaces of extremities, chest, abdomen; face, not strongly pigmented; back and posterior surface of extremities; face, strongly pigmented, nape of neck; scalp; and palms and soles (Katz, 1941; Rubin and Casarett, 1968).

These effects are primarily due to injury to proliferating germinative cells with the basal layer of the epidermis and bulb of the hair follicle, the time of expression of injury coordinating with kinetics of turnover of cells in the corresponding cellular compartment of the epidermis (UNSCEAR, 1982).

Lesions of blood vessels are especially important in the evolution of lesions from exposure to higher doses. The initial effect, vasodilatation, is followed by degenerative changes. An indolent ulcer develops in the more severe cases within 2 to 3 months, involving subcutaneous tissue, blood vessels, and sometimes even bone, in addition to skin (ICRP 28, 1978).

The skin tolerance dose at 50 R/min. is 1650 R (16.5 Gy) for an area of 100 cm^2 for a single exposure, increasing to 5500 R (55 Gy) for 25 exposures in 5 weeks for the same area.

Pathogenesis of late changes such as atrophy and fibrosis results in part from depletion of fibroblasts and injury to blood vessels in the dermis (Law, 1981). Subclinical changes in dermal floor vessels have been observed after a dose of 10-30 Gy accumulated over 8-25 years in occupational workers as well as in atomic bomb survivors (Leny *et al.*, 1978; Tsuya *et al.*, 1971).

Radiotherapy experience shows that a cumulative dose of 55 Gy of conventionally fractionated X-rays will lead to a 1 - 5% risk of late (several months to years) severe dermal fibrosis and ulceration, while 70 Gy will produce a 25 - 50% risk of such late changes (Rubin and Casarett, 1968; UNSCEAR, 1982).

A report by a Task Group of Committee 1 of ICRP, entitled 'The biological basis for dose limitation in the skin' has been published as ICRP publication 59 (1991). ICRP 60 quotes the following figures for different threshold effects as suggested by ICRP 59:

Erythema and dry desquamation: 3-5 Gy;
Symptoms appear after 3 weeks;
Moist desquamation: 20 Gy;
Blistering appears after 4 weeks;
Cell death in epidermal and dermal layers resulting in tissue necrosis: 50 Gy;
Effect occurs after 8 weeks.

A summary of the contents of ICRP 59 which deals with both deterministic and stochastic effects on skin is given in chapter 11, section 4 of this Handbook.

Hot Particle Irradiation

In this case very high doses occur over a small area. Ulceration or breakdown with subsequent ulceration may result. Threshold dose for < 1 mm^2 particles is 70 Gy over an area of 1.1 mm^2 or 1 Gy over 1 cm^2 at a depth of 100-150 μm. However, below 250 Gy, ulcers are transient, lasting less than a week (Hopewell *et al.*, 1986).

10(1).3 DIGESTIVE SYSTEM

Squamous cell mucosa of oral cavity, pharynx, oesophagus, and anus resemble the skin in rate of response. Glandular mucosa of stomach, small intestine, and colon respond more quickly, and tolerate less radiation in a single exposure. Killing of a large number of germinative cells in mucosal epithelium can interfere with cell renewal to cause ulceration and ultimately denudation of affected parts of mucosa, giving rise to the prodromal syndrome (nausea, vomiting, diarrhoea) within an hour and leading to rapidly fatal dysentry-like syndrome if a large part of the intestine is exposed to acute doses above 10 Gy (Bond *et al.*, 1965; Hall, 1978). Long-term complications (after months to years) include fibrosis, stricture, intestinal perforation and fistula formation (Rubin and Casarett, 1968).

Radiotherapy experience shows that for an area of irradiation of about 100 cm^2 the threshold doses to produce ulceration/stricture in 5 years in stomach, intestine, colon, rectum and oesophagus are 45-60 Gy; the organs are listed in decreasing order of radiosensitivity. For similar effects in 25-50% of the cases the corresponding doses are 50 - 75 Gy (Roswit *et al.*, 1972; UNSCEAR, 1982).

Threshold doses for damage to other organs of the digestive tract are indicated in Table 10(1).1.

10(1).4 HAEMATOPOIETIC SYSTEM

Proliferating haematopoietic cells are among the most sensitive. Cytological changes are demonstrable in bone marrow and lymphoid follicles within minutes after whole body irradiation of 1 Gy (Bond *et al.*, 1965). Changes in peripheral blood count are also manifest soon after comparable whole body exposure, the lymphocyte count declining almost immediately. The changes result from interference with normal cell replacement - through the killing of circulating lymphocytes which undergo interphase death in minutes (Bond *et al.*, 1965; Maisin *et al.*, 1971; Proukakis and Lindop, 1971). After acute whole body doses above 1 Gy, maximal depression of leukocyte count is reached in the second to fifth week, its rapidity increasing with dose. The platelet count falls somewhat more slowly and erythrocyte count much more slowly in the absence of haemorrhage.

The severity and duration of the resulting pancytopenia depend on the fraction of haematopoietic tissue irradiated, dose, dose rate and LET. Regional exposure of marrow to a dose above 30-40 Gy causes localized necrosis followed by atrophy, fibrosis, and replacement with fat, without demonstrable effects on overall haematological function. No deaths are expected below 0.5-1 Gy. For a healthy adult human, the $LD_{50/60}$ dose (i.e. the dose which is lethal to 50% of the exposed population within 60 days) is 3-5 Gy midline dose (which approximates to the marrow dose) for low LET radiation (ICRP 60, 1991). Infection (due to granulocytopenia) and haemorrhage (due to thrombocytopenia) are the main causes of death for doses in this range (Bond *et al.*, 1965; Upton, 1969). LD_{100} is 7-10 Gy.

Tolerance is increased by fractionation, mainly due to repopulation (and some repair of sublethal damage). For exposures lasting months the tolerance dose is 3-10 Gy (Martin, 1983).

The conclusion is that the threshold for detectable depression of haemopoiesis exceeds 0.4 Sv and the threshold for fatal marrow aplasia exceeds 1 Sv.

There is suppression of immunological response due to damage to lymphoid tissue. The depression is profound enough at around LD_{50} doses to enable transplantation of allogenic bone marrow (Micklem and

Loutit, 1966; Thomas, 1982). Early stages in the immunological response, including the process of antigen recognition, are more sensitive than later stages, including antibody synthesis itself (UNSCEAR, 1972; Anderson and Warner, 1976).

Doses as low as 0.5 to 1 Gy can cause the death of some circulating lymphocytes in the blood, and the peripheral lymphocyte count is a useful biological dosimeter following whole body irradiation.

10(1).5 CARDIOVASCULAR SYSTEM

The heart is not highly radiosensitive. At 40 Gy (protracted) there is some myocardial degeneration. Doses above 60 Gy to the entire heart may lead to death from pericardial effusion or constrictive pericarditis. Tolerance is higher if only a portion of the heart is irradiated (Stewart and Fajardo, 1972).

Since the vascular system is important for the integrity and function of all tissues, damage to the blood vessels may lead to various types of tissue/organ damage. Blood vessels show changes after 40-60 Gy. Vascular permeability and blood flow increase in early phases, to be followed after months by degeneration of endothelial cells, thickening of basement membrane and sclerosis. Late changes include endothelial proliferation, thickening of the wall, narrowing of the lumen, and decreasing blood flow, accompanied by tortuosity of arterioles and arteries, atrophy of smooth muscle, loss of elasticity of arterial walls and focal vascular constriction and dilatation. The impairment of vascular function in advance of tissue atrophy suggests that vascular damage plays a major role in most forms of late radiation-induced injury (Rubin and Casarett, 1968; Berdjiş, 1971; Law, 1981).

10(1).6 EYE AND OTHER SENSORY ORGANS

The lens of the eye is among the most radiosensitive tissues. At high doses lens opacities (cataracts) develop within months, progress rapidly, and eventually cloud the lens completely. The development of cataract depends also on the age at exposure and on whether all or part of the lens is irradiated. At lower doses opacities take years to develop, remain microscopic in size and cause no significant impairment of vision (Merriam *et al.*, 1972). The equatorial part of the anterior epithelium is the most sensitive. The phenomenon involves damage to dividing cells in the

slowly proliferating cells of the anterior epithelium. Damaged cells and their breakdown products migrate posteriorly and accumulate beneath the capsule at the posterior pole of the lens bow; at a sufficient degree of damage they present a dot-like central posterior subcapsular opacity (which is characteristic only of radiation-induced opacities), but do not interfere with vision. Depending on the dose, the lesions may progressively involve the anterior cortex and nucleus of the lens and impair vision.

Neutrons have a high RBE for cataractogenesis due to lack of vascularity of the lens and consequent hypoxia. The earliest cataracts were observed in cyclotron workers in the 1940s; the threshold for cataractogenesis in their case was 0.7-1 Gy of mixed neutrons-gamma rays (Ham, 1953). In the atomic bomb survivors the threshold for opthalmologically detectable opacity was 0.6-1.5 Gy (Otake and Schull, 1982). A quality factor for neutrons in excess of ICRP's (earlier) recommended value of 10 is not warranted.

In radiotherapy patients a similar threshold is 2 Gy for single and 5.5 Gy for fractionated exposures of low LET radiation. It may be assumed that for low LET radiation the threshold for vision-impairing cataract is 8 Gy (prolonged). The lens appears to be less sparing on protraction of exposure than other organs (Merriam *et al.*, 1972). Other parts of the eye are relatively radioresistant, in the following order of decreasing radiosensitivity: lid of skin, retina, cornea and conjunctiva, lachrymal gland, and sclera (Merriam *et al.*, 1972; UNSCEAR, 1982).

It appears that the lenses of children are more radiosensitive than those of adults.

The middle and inner ear can withstand up to 50 Gy of fractionated exposures without serious effects on hearing (Rubin and Casarett, 1968, 1972).

10(1).7 NERVOUS SYSTEM

Radiotherapy experience has provided significant data in this field. The central nervous system has traditionally been regarded as radioresistant. But transitory electrophysiological changes in the brain can be evoked by relatively small doses (UNSCEAR, 1969). Even at high doses there is considerable repair of sublethal damage and a small degree of delayed repopulation, but there is no evidence of 'slow repair' in the spinal cord (UNSCEAR, 1982). Results are summarized in Table 10(1).2.

10(1).8 REPRODUCTIVE SYSTEM

The germ cells of the testis and ovary are highly sensitive whereas other cells of the reproductive system are relatively radioresistant (Rubin and Casarett, 1968). The killing of germ cells can impair fertility in both sexes, to a degree which is dose dependent.

In the testes, spermatogonia in an early stage of differentiation are more sensitive than spermatocytes, spermatids or spermatozoa. An acute exposure of 0.15 Gy causes significant depression of cell count (due to killing of late stage spermatogonia), but the sperm count does not start to decline until several weeks when the more mature germ cell stages are eliminated from the reproductive tract. Fertility will be restored if enough 'stem' spermatogonia survive to repopulate the seminiferous tubules, but recovery may take years after a high dose (Lushbaugh and Casarett, 1976; Hahn *et al.*, 1982). Sterility may be permanent above 3-5 Gy. This threshold does not increase with fractionation in humans, but animal experiments show a different picture. It is inferred that human testes can tolerate 1 mGy/day of occupational irradiation for an indefinite period without impairment of fertility.

There are 2 million germ cells in the human ovary at birth, of which 50% are atretric. The number of follicles rapidly declines from 400,000 at 12-16 years to 8000 at 40-44 years. This decline is due to atresia, since only 400 oocytes are ovulated during a reproductive lifetime of 35 years.

In the human ovary the mature oocyte is the most radiosensitive germ cell stage. Doses above 0.65-1.5 Gy cause prompt impairment of fertility. Below 2-3 Gy, fertility will eventually be restored due to surviving immature oocytes. Tolerance doses are 6-20 Gy for highly fractionated or protracted exposures (Lushbaugh and Ricks, 1972; Lushbaugh and Casarett, 1976). With increasing age, the threshold for permanent sterility decreases (since the ovary contains no proliferative oogonial stem cell pool to replace oocytes lost through aging and ovulation) from, say, 8 Gy at age 20 to 3 Gy at age 40. In radium dial painters the number of live-born children was slightly reduced at ovarian doses above 0.2 Sv (Polednak, 1980). No late effect of radiation on fertility has been detected in the atomic bomb survivors (Blot and Sawada, 1972).

10(1).9 URINARY TRACT

Table 10(1).3 summarizes the effects.

Children's kidneys appear to be somewhat more radiosensitive than those of the adult.

10(1).10 RESPIRATORY SYSTEM

The respiratory system has sufficient reserve to tolerate high levels of localized injury. Two types of damage are shown: early pneumonitis (starting within weeks to months after acute exposure), and long-term changes associated with pulmonary fibrosis (latent period around one year). Significant repair of sublethal damage also occurs.

Radiosensitive cells in the alveolar regions are the epithelial cells lining the alveoli and the endothelial cells of capillaries.

Radiation pneumonitis is characterized by interstitial oedema, desquamation of epithelial cells from alveolar walls and hyperplasia of pneumocytes. Following the acute phase of pneumonitis, progressive lung damage may occur after a latent period of 6 months. There may be irreversible fibrosis of alveolar septa followed by loss of alveoli and their replacement by collagen and connective tissue to form a scar. For total lung irradiation fatal pneumonitis can result in 2-6 months with an LD_{50} of 8-10 Gy for a single exposure and 20-30 Gy for fractionated exposure of low LET radiation (Wara *et al.*, 1973; Fryet *et al.*, 1978). The upper respiratory tract can tolerate a fractionated dose of 30 Gy; at higher doses the effects are mucositis, ulceration, atrophy and fibrosis (Van den Brenk, 1971).

10(1).11 MUSCULOSKELETAL SYSTEM

The higher absorption of low energy X-rays in bone, compared with that in soft tissue, results in a bone dose more than double that of similarly exposed soft tissue; there can be aseptic necrosis and fracture at doses tolerated by adjacent soft tissue. But for ^{60}Co gamma rays and megavoltage X-rays, the difference in dose is only 3-4%; radionecrosis in the latter cases is rare.

The musculoskeletal system is radioresistant in adults, but there is a heightened response in the proliferative state (children, healing of fracture). Even at 1 Gy there may be some retardation of growth in children due to sterilization of stem cells in epiphyses (Tefft, 1972; Blot, 1975). Above 20 Gy (fractionated) effects on children include scolosis, kyphosis, slipped upper femoral epiphysis and exostoses (Chapman *et al.*, 1980; Thomas *et al.*, 1983).

Tolerance dose in adults is 40 Gy (fractionated) for cartilage and 65 Gy for bone. But their susceptibility to subsequent trauma seems to be increased after a year or more (Parker, 1972).

For internally deposited alpha emitters, RBEs above 10 have been derived. The spatial and temporal distribution of dose depends on the chemical characteristics of the radionuclide. Types of change observed in bone are complex, including changes in trabecular bone patterns, patchy sclerosis, small and large bone infarcts. The estimate of RBE for deterministic effects in bone from internal emitters is very uncertain.

Mature muscle and connective tissue are relatively radioresistant. Contraction and delayed healing may occur above fractionated doses of 60 Gy (Rubin and Casarett, 1968).

10(1).12 ENDOCRINE SYSTEM

The endocrine glands have a low rate of cell turnover and hence are relatively radioresistant in adults. They can tolerate far larger doses when partially irradiated than for *in toto* irradiation.

The thyroid is the most sensitive, particularly in children. In the Marshall Islanders exposed to gamma radiation from fallout, there was an asymptomatic decrease in thyroid reserve (increased TSH, exaggerated TSH response to thyrotropin-releasing hormone) even at 4 Gy, while hypothyroidism accompanied by retardation of growth occurred at 7-14 Gy (Conard *et al.*, 1980; Larsen *et al.*, 1982). In adults the threshold dose for myxedema is 25-30 Gy (fractionated).

Hypothyroidism is a frequent complication of radioiodine therapy for thyrotoxicosis. For single administrations of less than 2 MBq (50 μCi) of ^{131}I per gram of thyroid, the incidence of hypothyroidism was 22%, and for doses above 6.5 MBq (175 μCi) per gram of thyroid, it was 55% (Becker *et al.*, 1971). For an absorbed dose to thyroid of 50 - 120 Gy, the incidence of hypothyroidism was 7.5% within the first year, with 3% becoming hypothyroid each year thereafter (Edsmyr and Einhorn, 1966).

Thresholds for permanent functional depression are 45 Gy (fractionated) for adult pituitary and 60 Gy (fractionated) for adult adrenals (Rubin and Casarett, 1968; Einhorn and Einhorn, 1972; Samaan *et al.*, 1982).

Though the female breast is relatively radioresistant in adults, it is highly susceptible in childhood, the threshold being 10 Gy of fractionated exposure (Rubin and Casarett, 1968).

10(1).13 DEATH AFTER WHOLE BODY IRRADIATION

The discussion in this section is based on ICRP 60.

High acute doses may result in death, which is mainly the result of severe cell depletion in one or more vital organ systems. The dose-response relation is sigmoid when plotted on linear axes, while for a probability-linear plot the shape is approximately linear [Fig. 10(1).1(a) and (b) respectively].

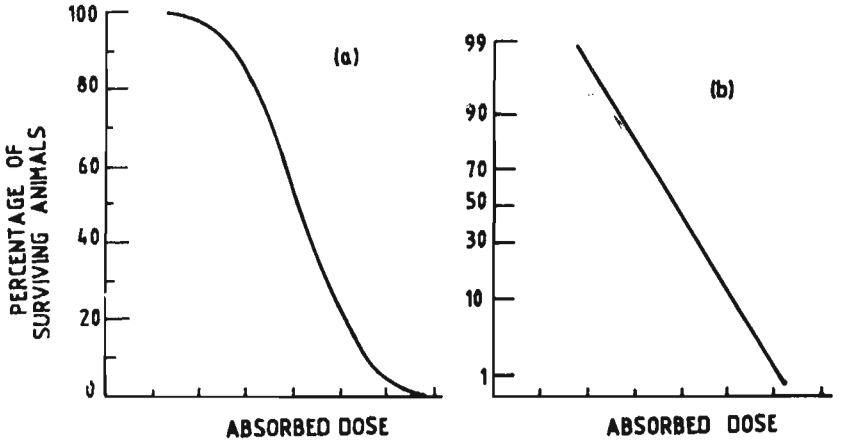

Figure 10(1).1. Typical dose-response curves for irradiated mammals: (a) linear ordinate; (b) probability ordinate (from ICRP 60).

No individual is expected to die at doses below 1 Gy; then as the dose increases the probability increases rapidly up to a certain dose beyond which the probability of death is virtually 100%. For a healthy adult human, the $LD_{50/60}$ value is 3-5 Gy midline dose (which approximates to the marrow dose) for low LET penetrating radiations. The chances of survival of individuals exposed to doses around or higher than the $LD_{50/60}$ can be improved by stimulating viable bone marrow stem cells, or by substituting new isologous marrow or concentrates of bone marrow stem cells from a suitable donor, together with appropriate medical care (fluid replacement, antibiotics, antifungal drugs, and barrier nursing) (UNSCEAR, 1988).

Values of $LD_{5/60}$ and $LD_{95/60}$ are useful end points in helping to establish the slope of the dose-survival curve.

At doses above 5 Gy, additional effects occur, including severe gastrointestinal (stem cell and capillary cell) damage which, when combined with bone marrow damage, cause death in 1-2 weeks. At about 10 Gy, acute inflammation of the lungs can lead to death. At even higher doses, effects on the nervous and cardiovascular systems occur and the individual dies of shock after a few days (NCRP, 1974). Doses for death at different times (for high dose, low LET radiation given over a few minutes) are given in Table 10(1).4.

10(1).14 CONCLUSIONS AND RECOMMENDATIONS

Decades of experience in the followup of radiotherapy patients (supported by animal experiments) indicate that for most tissues and organs there is a threshold dose for damage to normal function The threshold dose of conventionally fractionated X-rays for such damages varies from 2-3 Gy for permanent impairment of the fertility of the ovary, to more than 100 Gy for atrophy of involuntary muscle.

In some tissues (e.g., bone marrow and testes) damage results primarily from radiation-induced killing of proliferating stem cells, leading to interference with cell renewal and to depletion of functioning tissue elements below critical levels. In other instances, mainly involving slowly developing injuries (e.g. central nervous system, kidney, dermis, nerve, endocrine organs, heart), the aetiology is debated. It could be from damage to nutrient blood vessels (Law, 1981) or a late effect from depletion of the slowly proliferating parenchymal or supplying cells (Withers *et al.*, 1980).

If the tolerance values derived from radiotherapy experience are assumed to be applicable, with appropriate adjustments, it can be inferred that, presumably, the dose limits recommended by ICRP 26 provide a substantial margin of safety for all tissues, with the possible exception of the bone marrow, gonads and lens of the eye. In the case of the testes and the bone marrow, protraction of the exposure over decades should minimize the effect of occupational irradiation, because spermatogonial and haematopoietic cells are highly capable of regeneration and repopulation. For the ovary and lens, however, annual exposure at the dose limit might conceivably lead to cumulative doses approaching the thresholds for deterministic effects.

ICRP 41 gives a detailed table (not reproduced in this Handbook) on the threshold doses for clinically determined deterministic effects in various tissues, based on responses of patients to conventionally fractionated

therapeutic X- or gamma radiation (Rubin and Casarett, 1972). The type of injury to the tissue or organ as well as the doses causing the effect in 1 - 5% and 25 - 50% of irradiated patients are tabulated.

Table 10(1).5 gives the thresholds for deterministic effects in the adult human testes, ovaries, lens and bone marrow, as well as the annual dose limits as recommended by ICRP 26 (1977). The conclusions are given in the following:

10(1).14.1 Testes

Testes are not likely to be irradiated selectively. They can tolerate occupational irradiation at a dose rate of 0.3-0.4 Sv per year indefinitely without detectable impairment of fertility. Thus the ICRP 26 stochastic limit for testes when irradiated alone (0.2 Sv/year), and more so, the ICRP 26 whole body limit (0.05 Sv/year) would provide adequate protection.

If the testes were to receive the ICRP 26 annual dose limit of 0.2 Sv in a single brief exposure (a most unlikely situation), transitory depression of sperm count may ensue.

10(1).14.2 Ovaries

Ovaries are not likely to be irradiated selectively. The ICRP 26 whole body dose limit of 0.05 Sv/year provides adequate protection. In theory, however, ovaries, if irradiated alone at the ICRP 26 dose limit level of 0.2 Sv/year, may reach the threshold of 6 Gy for permanent sterility in 30 years.

10(1).14.3 Lens of the Eye

Exposure of the lens at the dose limit of 0.15 Sv/year for 30 years would not cause vision-impairing cataract (threshold 8 Sv), although it might give rise to opacities that could be detected opthalmologically in some exposed individuals.

10(1).14.4 Bone Marrow

Threshold level for fatal haematopoietic failure is estimated to exceed 1 Gy/year under conditions of low level irradiation, which exceeds the implied dose limit for exposure to marrow alone. For internal contami-

nation, there might be preferential irradiation of bone marrow from bone-seeking radionuclides and marrow dose rates of 0.33 Sv per year are possible. In practice, however, dose rates are limited to lower values because of simultaneous radiation of non-skeletal tissues and because relatively long periods of intake at the ALI are required for reaching the limiting dose rate from many long-lived bone-seekers. Hence, limiting the dose rates to the marrow from external or internal irradiation can be expected to cause no detectable impairment of haematopoietic function.

10(1).14.5 Dose Limits for Members of the Public

Stochastic dose limits for members of the public will suffice to prevent the production of deterministic effects.

10(1).15 REVISED DOSE LIMITS (ICRP 60)

As per ICRP 60 recommendations, the annual effective dose limits as well as the weighting factor for gonads have been revised. Consequently, the revised limits for annual occupational exposures for the tissues discussed above would be:

Gonads

If irradiated alone:	0.10 Sv/year;
If irradiated with the whole body:	0.02 Sv/year

Red marrow

If irradiated alone:	0.17 Sv/year;
If irradiated with the whole body:	0.02 Sv/year

Lens of the eye

If irradiated alone:	0.15 Sv/year;
If irradiated with the whole body:	0.02 Sv/year

It may be seen that the revised limits will ensure that even the cases of marginal damage that were postulated with the earlier limits for these organs would not occur for occupational workers. The level of protection for members of the public would be much higher.

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Table 10(1).1: Deterministic effects and threshold doses in certain organs of the digestive system

(based on ICRP 41)

Organ	Effect	Threshold dose (fractionated) (Gy)
Liver	Damage to centrilobular veins, thrombosis, portal hypertension; may progress to hepatic failure, ascites and death (Kraut *et al.*, 1972; Wharton *et al.*, 1973);	30
	Early loss of Kupfer cell function demonstrated in radionuclide scans; may develop into cirrhosis (Jeffrey *et al.*, 1980); Hepatitis in 75% of the cases	40
Salivary glands	Reduced salivary flow; damage to non-dividing acinar cells and their vasculature; necrosis, atrophy, fibrosis (Rubin and Casarett,1968)	50-70
Pancreas	Damage to non-dividing acinar cells and their vasculature (Rubin and Casarett, 1968; Cheng *et al.*, 1981)	70-80

Table 10(1).2: Deterministic effects and threshold doses in the nervous system
(based on ICRP 41)

Tissue	Effect	Threshold dose (fractionated) and latent period
Brain	Detectable physiological/ morphological changes in children (Ron *et al.*, 1982)	1-6 Gy
	Leucoencephalopathy, EEG changes, specially in children, functional disturbances (UNSCEAR, 1982)	10 Gy months
	Necrosis, demyelation, damage to vasculature, neurological symptoms; Transient headaches, drowsiness, nausea, increased risk of seizures, occurring weeks to months after exposure, but generally self-limiting (Boldrey and Sheline, 1966);	55 Gy 1-3 y
Spinal cord	Myelitis caused by demyelation, necrosis of neurons in white matter and damage to fine vasculature (Rubin and Casarett, 1968; Sheline *et al.*, 1980; Dritschillo *et al.*, 1981; Wigg *et al.*, 1981) Symptoms: tingling, paraesthesia, weakness, paralysis	30 Gy 6 months-2 y
Optic nerve	Response similar to spinal cord	
Peripheral nerves	(Rubin and Casarett, 1968)	60 Gy

Table 10(1).3: Deterministic effects and threshold doses in the urinary system
(based on ICRP 41)

Tissue	Effect	Threshold dose (fractionated) and latent period
Kidneys	Degenerative changes in fine vasculature and epithelium of nephrons; histological changes due to degeneration and depopulation of renal tubules (Rubin and Casarett, 1968; Mostofi and Berdjis, 1971; Maier, 1972; Law, 1981; UNSCEAR, 1982)	10 Gy 6-12 months
	Reduction in excretory function	20 Gy
	Fatal nephritis-like reaction; (Late damage characterized by hypertension, albuminurea renal failure)	> 40 Gy 6-12 months
Bladder	Cystitis, ulceration, fistula, fibrosis, contraction, urinary obstruction (Rubin and Casarett, 1968; UNSCEAR, 1982)	55 Gy
Ureters	Urinary obstruction	> 60 Gy

Table 10(1).4: Range of doses associated with specific syndromes and death in human beings exposed to acute low LET whole body radiation
(from ICRP 60)

Whole body absorbed dose (Gy)	Principal effect contributing to death	Time of death after exposure (days)
3-5	Damage to bone marrow ($LD_{50/60}$)	30-60
5-15	Damage to GI tract and lungs	10-20
> 15	Damage to nervous system*	1-5

* Damage to vasculature and cell membranes at high doses is important.

Table 10(1).5: Thresholds for deterministic effects in the adult human testes, ovaries, lens and bone marrow and the annual dose limits
(from ICRP 41)

Organ	Threshold			Annual equivalent dose limit (Sv) (ICRP 26, 1977)	
	Total eq. dose: single exposure (Sv)	Total eq. dose: protracted/fractionated exposure (Sv)	Annual dose rate: protracted/fractionated exposure (Sv/y)	if irradiated alone	if irradiated with whole body
Testes					
Temporary sterility	0.15	NA*	0.4	0.2	0.05
Permanent sterility	3.5	NA	2.0	0.2	0.05
Ovaries					
Sterility	2.5-6.0	6.0	> 0.2	0.2	0.05
Lens					
Detectable opacities	0.5-2.0	5	> 0.1	(0.15)	0.05
Visual impairment (cataract)	5.0	> 8	> 0.15	(0.15)	0.05
Bone marrow					
Depression of haemopoiesis	0.5	NA	> 0.4	0.4	0.05
Fatal aplasia	1.5	NA	> 1	0.4	0.05

Values listed, except those in parentheses, denote annual equivalent dose limits for stochastic effects, these values being limiting for the tissues in question.

* NA denotes Not Applicable, since threshold is dependent on dose rate rather than on total dose.

CHAPTER 10: SECTION 2

RBE FOR DETERMINISTIC EFFECTS

10(2).1 INTRODUCTION

ICRP has been recommending values for the 'Quality Factor', Q (now called the 'radiation weighting factor', W_R) for different types of radiation for protection purposes which are related to RBE and LET. These are applicable only for stochastic effects at low doses (below the thresholds for deterministic effects, which are usually in excess of 1 Gy), and low dose rates. In view of the specific purpose for which Q values were selected, they may not represent the highest values of RBE judged to be applicable to all effects in all tissues. Thus, for some of the effects, either stochastic or deterministic, RBE values for specific exposure conditions might be larger than Q values for the high LET radiation considered. For a given tissue exposed selectively, e.g. as a consequence of the intake of a radionuclide specifically retained in that tissue, dose limits or limits on intake based on Q values might not be adequate to prevent deterministic effects induced by high LET radiation if an effect in this tissue is caused with a very high RBE value for these conditions of exposure.

ICRP publication 58, 'RBE for deterministic effects' (1989), a report of Committee 1 of ICRP, reviews information on deterministic effects after doses per fraction in excess of 1 Gy from studies on laboratory animals and humans. Extrapolation to doses much smaller than 1 Gy requires the application of radiobiological models, from which RBE values at low doses of various high LET radiations can be derived for specific responses of different tissues. These values are useful because of their application to exposures in which a mixture of high and low LET radiations may cause enough damage to result in observable deterministic effects. They can be applied to estimate possible consequences of exposures to high LET radiations, and to judge, whether, for specific tissues, RBE values in excess of Q values should be taken into account to achieve adequate protection.

A generally applicable theory concerning the dependence of RBE on radiation quality is not available, although trends have been established.

The absolute effectiveness of low LET radiations is more strongly dependent on many factors, e.g. dose, dose fractionation, dose rate, and cellular conditions, than is the case for high LET radiations. Per unit dose, low LET radiations are most effective at high doses and high dose rates. Thus, RBE values for high LET radiations are relatively low under these conditions, i.e. in the range of 1 to 5. With decreasing dose and dose rate, the effectiveness per unit dose for low LET radiation decreases more rapidly than that of high LET radiation. With decreasing dose rate, there is increasing cellular repair and reduction of the contribution from accumulated damage. Consequently, RBE values increase with decreasing dose and dose rate. From theoretical considerations and experimental evidence, it can be stated that, as dose and dose rate decrease, RBE values approach a maximum value depending on the type of radiation and end point observed. These are designated as RBE_M and RBE_m for stochastic and deterministic effects respectively. For stochastic effects RBE values range from 1 to over 50 at low doses. RBE values in excess of 10 have not been observed for deterministic effects in a variety of tissues. For damage to certain molecules, e.g. proteins, and to some structures in cells, e.g. membranes, RBE values less than 1 for high LET radiations have also been observed (Henglein and Schnabel, 1966). RBE values range widely, depending on the type of radiation (mainly LET), exposure parameters (dose rate, fractionation), conditions of the system (cell cycle stage), presence of sensitizers (e.g. oxygen) or protectors, tissue irradiated, and specific end point observed.

10(2).2 RBE VALUES

Loss of reproductive capacity of cells plays a crucial role in the pathogenesis of most deterministic effects (ICRP 41, 1984). Responses of tissues generally show characteristics similar to those for clonogenic capacity of cells in culture (including repair of damage, oxygen effect, and post-irradiation cell proliferation kinetics). Hence RBE-LET relations can be inferred from cell culture studies. RBE values for non-lethal damage, e.g. mitotic delay or partially impaired proliferation, are not significantly different from those for loss of clonogenic capacity (Broerse and Barendsen, 1973).

RBE values for reproductive death of cells are frequently larger for cell types which are relatively resistant to low LET radiation, e.g. intestinal crypt stem cells, as compared to cells which are relatively sensitive, e.g. bone marrow stem cells (Broerse and Barendsen, 1973).

Table 10(2).1 gives the range of variation in RBE due to different factors for induction of cell reproductive death.

ICRP 58 gives a number of tables for RBE and RBE_m values for various types of irradiation (neutrons of energy 1-50 MeV, heavy ions with LET values reaching up to 1500 keV/μm), different mammalian systems including humans, and different end points. Table 10(2).2 summarizes the results in a very condensed form. Detailed reference to the sources of data are given in ICRP 58. The theoretical derivation of RBE_m values from the observed data is described in the next section. Table 10(2).3 summarizes the range of RBE values for different deterministic effects.

There is a wide range of variability in the RBE values. But the RBE_m values are all lower than 10, and considerably smaller, by a factor of 2-5, than values of RBE_M, with the single exception of kidney damage by 2.5 MeV neutrons (RBE_m = 12).

Electrons and photons above 1 MeV are less effective than at energies around 0.1 MeV or less, particularly for stochastic effects at low doses. Between low energy X-rays and higher energy gamma rays (or high energy electrons), there is a difference of around 2 at low doses (Sinclair, 1985). The difference in RBE values renders the choice of the reference radiation critically important for stochastic effects, but is likely to be less critical for deterministic effects which result from cell reproductive death. Radiotherapy experience shows that at absorbed doses of about 1 Gy, the RBE of electromagnetic radiations does not change by more than 20% over an energy range from 10 keV to 100 MeV (ICRU, 1986).

RBE values increase with LET until around LET values of 100-150 keV/μm, after which they seem to decrease.

(Author's note: In the formulation of Q-L relationships (L being the linear energy transfer), ICRP 60 has allowed for the reduced effectiveness of heavy ions with LET greater than 100 keV/μm.)

Substantial experimental evidence is available to indicate that for fractionated exposures, the RBE values as a function of dose per fraction for the same end point are similar for animals and humans.

For inhomogeneous distribution of radionuclides (as in the case of alpha emitters in the lung) the term 'RBE' does not strictly apply and 'RBE' values may be larger than 10 for reasons unrelated to LET. Cell reproductive death due to incorporation of ^{125}I in DNA gives RBE values as high as 50, although for randomly distributed Auger electron emitters the RBE was around 10.

10(2).3 RBE VALUES FOR EFFECTS ON CULTURED CELLS

As mentioned earlier, cell reproductive death is the effect which plays a crucial role in deterministic effects. Thus the RBE-LET relations can be assumed to be the same for both cell reproductive death and deterministic effects.

We know that the surviving fraction S of cells exposed to a dose D can be described by the equation

$$S = \exp. (- \alpha D + \beta D^2),$$

where α determines the initial slope of the survival curve at low doses and β determines the contribution to the frequency of cell inactivation by accumulation of damage.

If D_H and D_L are the doses of high and low LET radiation respectively to reduce the fraction of clonogenic cells to the same level, we can write

$$\alpha_L D_L + \beta_L D_L^2 = \alpha_H D_H + \beta_H D_H^2$$

$$\text{Now RBE } (D_H) = D_L/D_H$$

For very small values of D_H and D_L, it is easy to show that this equation leads to the following approximation:

$$\alpha_L = \alpha_H / \text{RBE } (D_H).$$

$$\text{For } D_H \longrightarrow 0, \ \text{RBE}_m = \alpha_H/\alpha_L$$

Knowing the values of the α, β parameters for high and low LET radiations at large doses we can derive RBE_m values.

As a first approximation it may be assumed that $\beta_H = 0$. A somewhat better approximation is to take $\beta_H = \beta_L$. The difference in RBE_m values calculated on these two assumptions is generally less than 20% except for $\text{RBE}_m < 3$.

RBE values are commonly within 10% of RBE_m values at doses of 0.1 Gy of X-rays or 0.01 Gy of high LET radiations.

Cell survival curves show that for large doses (surviving fraction around 0.01) the maximum RBE value is around 3, while for surviving fraction around 0.8, it is about 8 at 100 keV/μm (Barendsen, 1968). The pattern

of variation with LET is nearly the same, irrespective of the surviving fraction, increasing with increasing LET, reaching a maximum around 100-200 keV/μm, with a subsequent decrease. The decrease is presumably due to a saturation effect. RBE increases with decreasing dose, dose protraction and dose rate and approaches a maximum value, depending upon the type of radiation, below about 0.1 Gy of low LET radiation.

These data have been obtained for doses in the range above 1 Gy of low LET radiation and extrapolation must be applied for lower doses.

10(2).3.1 RBE Data for Different Types of Cultured Cells

There are considerable variations in the values of α, β and α/β between cultured cells of different origin; RBE and RBE_m values also vary correspondingly by a factor of at least 3 for the same high LET radiation for different cell lines. The range of RBE values for heavy ions is not considerably different from the range for lighter ions like alpha particles. Table 10(2).3 gives the range of RBE values for different deterministic effects for three categories of radiation and three ranges of doses or doses per fraction.

For neutrons the largest α values and the largest RBE_m values are observed at energies of 0.5-1.5 MeV (corresponding to $\bar{y}_F(1\mu m)$ values of 50-60 keV/μm). (For explanation of the quantity $\bar{y}_F$, see Chapter 16.) RBE_m for fast neutrons increases by a factor of 3 for values of $\bar{y}_F$ between 10 and 50 keV/μm. RBE values at doses of 5-10 Gy of low LET radiation are lower than corresponding RBE_m values by a factor of 2-3 (Barendsen, 1979).

If cell reproductive death is the basic mechanism resulting in tissue damage, the dependence of RBE on dose and type of cell would be expressed also for the effects on corresponding tissues. For a number of tissue responses, the critical cells have been identified. A good correlation is found between the characteristic parameters α and β and RBE values derived from cell survival curves for the responses of these critical cells on the one hand with equivalent parameters for the response of the tissues on the other. Examples of such correlation are discussed in ICRP 58 for early skin damage induced by X-rays, bone marrow failure, early damage to the intestinal tract, etc. The conclusion is thus strengthened that cell reproductive death is mainly responsible for tissue injury. Therefore, the LQ model and the associated model of extrapolation to low doses, which has been applied to cell survival curves, will be applicable to tissue responses also.

10(2).4 RBE VALUES FOR TISSUE RESPONSES

The mechanisms for the occurrence of deterministic effects and their characteristics have been discussed in Chapter 9, and Chapter 10, Section 1. Some further points are elucidated below.

The time interval between irradiation and the expression of tissue damage depends on the life span of the mature functioning cells. Three types of tissue can be distinguished:

(a) *Rapid cell renewal systems* with a life span of days to weeks. The cell population is maintained by supply of new maturing cells from a stem cell compartment. If all the stem cells are sterilized by a high enough dose, the latency period for tissue damage is determined mainly by the life span of maturing cells. Examples are the epithelium of GI tract, bone marrow cells, skin, oral mucosa, and testis.

(b) *Slow renewal systems* which show little cell proliferation under normal conditions because of the long life span of the differentiated functioning cells. These may not have an identifiable stem cell compartment. Examples are liver, kidney, bone and blood vessel endothelium. After significant damage, loss of cells after a long latent period can result in rapid replacement by induction of accelerated proliferation of surviving cells. The replacement can occur early or late, depending on the life span and the rate of loss of functioning cells.

(c) *Non-renewal systems* which consist of cells which are not replaced in the adult. Examples are nerve cells and oocytes. The difference between the two is that oocytes are very radiosensitive while nerve cells are radioresistant.

For maintaining function, tissues with long cell renewal times depend after fractionated or low dose rate exposure mainly on the ability of cells to repair intracellular injury, rather than on cellular repopulation. The ratio α/β is generally smaller for late responding tissues, indicating a relatively large influence of repair after low LET exposure (Fowler, 1983, 1984). For photon irradiation, iso-effect curves for late end points show a stronger dependence on the dose per fraction than those for early reactions. For high LET radiations, the iso-effect curves are much less dependent on dose per fraction. The net effect is that RBE may be higher for late effects (Withers *et al.*, 1982). Even for the same tissue, RBE values may be different for different end points.

In view of the available information, it is considered reasonable to assume that RBE values as a function of dose per fraction for the same end point are similar for animals and humans.

10(2).5 EFFECTS OF INHALED ALPHA AND BETA EMITTERS IN LUNG

The lung has more than 40 distinctive cell types. ICRP publication 31, 'Biological effects of inhaled radionuclides' (1980) (summarized in Chapter 15 of this Handbook) has reviewed dose-effect relationships for stochastic and deterministic effects. Some further data of a limited nature that have become available since the publication of ICRP 31 are summarized in ICRP 58. The main deterministic effects are early severe inflammation and late fibrosis.

The spatial and temporal distribution of dose in relation to the sensitive cells depends on a variety of factors, including nature of radiation, particle size, solubility, rate of clearance, etc. For lung cancer induction, an 'Equal Effectiveness Ratio' of 20-30 for alpha particles in relation to betas has been derived; this can be taken to approximate RBE_m.

Calculations for a hypothetical accidental release from a pressurized water reactor show that at 1 year the cumulated equivalent dose to lungs from alphas is less than 2% of the total. (External gamma radiation accounts for 60% and internal beta-gamma emitters 38%.)

There have been very few accidental exposures in the nuclear industry leading to deterministic effects. No acute effects in the human respiratory tract from radon and daughters have been reported (BEIR IV, 1988).

Limited animal data indicate that for beta irradiation lung damage depends both on the total dose and dose rate. For exposures protracted over days the mean lethal dose in dogs is 100 Gy, while if the exposure is over months, it increases to 550 Gy. For mortality from radiation-induced pneumonitis and fibrosis the equal effectiveness ratio is 7-10. Functional impairment will occur at doses between 25% and 50% of those resulting in mortality and the equal effectiveness ratio is similar to that for mortality.

10(2).6 RBE VALUES FOR DETERMINISTIC EFFECTS AND THEIR RELATION TO *Q* VALUES

As discussed earlier, RBE values increase with dose fractionation and reduction in dose rate. On the basis of the LQ model, the magnitude of this increase is determined principally by the value of α/β for low LET radiation. With this model, data available for a given dose fractionation can be extrapolated to other doses per fraction or dose rates. For example, for X-rays, there is a difference, by a factor of at least 2, in RBE between large single doses of 10-20 Gy and doses per fraction of 2-3 Gy. For chronic exposures at the dose limit level, RBE values are expected to attain a maximum RBE_m. However, deterministic effects are not in general observed after such low doses and RBE_m values can be derived only by extrapolation from higher dose rates and larger doses per fraction. RBE_m may be assumed to apply at dose rates of 0.1 Gy per hour and less of the reference radiation and 0.01 Gy of high LET radiation (Barendsen, 1982).

Because the range of RBE values covers at least a factor of 2, average RBE values should not be used for medical intervention purposes whereby an assessment is required of the probability that a given exposure will actually result in tissue damage.

We have seen that RBE_m values are normally in the range of 4-8 for most deterministic effects in tissues, and are less that RBE_M values for stochastic effects. The RBE_M values for fission neutrons are: 40-50 for cytogenetic effects; 35-70 for cell transformation; and 3-200 for tumour induction (Sinclair, 1985). Since *Q* values are based on RBE_M, they are high enough to provide a sufficient degree of protection for deterministic effects for radiation protection purposes (including choice of ALIs).

10(2).6.1 Accident Situations

In accident situations, exposure will result from a mixture of radiations of different quality. RBE values for mixed radiations can be computed from the relations observed for cell killing. If q, the fraction of the dose from high LET radiation to the total dose, is a substantial fraction of the total, we can derive an RBE for the mixed radiation, R, as follows:

$$R = D_e / D$$

where D_e is the equivalent photon dose (i.e. that which produces the same magnitude of effect as D), and D the absorbed dose. An expression

for R can be derived from the values of q, α, β and RBE_m. For small doses (where $D << \alpha/\beta$),

$$R = 1 + q(RBE_m - 1).$$

If the fraction of high LET radiation is known, the procedure discussed earlier can be applied to derive an effective RBE. However, if the high LET component constitutes only a few per cent of the total dose or if the total dose rate is less than 1 Gy per hour, RBE_m values for the concerned tissue will provide a sufficient approximation. RBE values, rather than Q values, should be applied in assessing medical management.

10(2).7 CONCLUSIONS

RBE values of high LET radiations for deterministic effects depend on a variety of factors, in particular on the type of radiation and the type of tissue, and vary widely. They increase with decreasing dose, dose per fraction, and dose rate. Maximum values derived by extrapolation to very low doses are denoted by RBE_m to distinguish them from RBE_M for stochastic effects.

RBE_m values are generally lower than 10, and therefore are considerably smaller, by a factor of 2-5, than values of RBE_M. A formalism has been developed for deriving RBE values for mixed high and low LET radiations. Application of RBE_m values will yield estimates of maximum values of equivalent dose, and these should be applied for planning medical intervention only when the contribution from high LET radiation is small.

Because RBE_m values for deterministic effects are considerably smaller than RBE_M values for stochastic effects (upon which the assessment of Q values is based), the application of Q values in cases where deterministic effects are important would result in an overestimate of the contribution from high LET radiation. The present Q factors are certainly adequate for the establishment of ALIs which are determined by deterministic effects.

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Table 10(2).1: Variations in RBE for induction of cell reproductive death
(from ICRP 58)

Factor causing difference	Factor of range in RBE
Difference in cell or tissue	3
Difference in ion charge and density	10
Difference in neutron energy	3
Dose, dose per fraction, or dose rate	5

Table 10(2).2: Calculated RBE_m values* for deterministic effects in mammalian systems
(based on ICRP 58)

Tissue or organ, type of effect	Neutrons 1-5 MeV mean energy	Neutrons 5-50 MeV mean energy	Heavy ions C, Ne, Ar
Skin:			
Early response	7-9	4-5	3
Late effects		3-6	
GI tract:			
Early response	5-7	3-7	1.3-5
Late effects	5-9	5-7	
Haematopoietic system:			
Early response	4	2-3	1.2-3
Eyes:			
Late effects	5	3	2-3
Nervous system:			
CNS, late effects	-	5-7	4-8
Brain, late effects		6-10	
Reproductive system:			
Testes, early effects	5-6	3	2
Ovaries, early effects	1	2-3	
Urinary system:			
Kidneys, late response	12	6-9	4-6
Bladder, late response	7-9		
Respiratory system:			
Late response	8	7	4
Vascular system:			
Late effects	-	4-8	-

* Rounded figures. Detailed references to the sources from which the information has been collated may be found in ICRP 58.

Table 10(2).3: Range of RBE values for different deterministic effects
(based on ICRP 58)

Deterministic effect	1-5 MeV neutrons	5-50 MeV neutrons	Heavy ions C, Ne, Ar
Single exposure, high dose rate	2-4	1-3	1-3
Fractionated exposure, 2-3 Gy per fraction	4-8	2-5	1-4
RBE_m	4-12	3-10	2-5

Detailed references to the sources from which the information has been collated may be found in ICRP 58.

CHAPTER 11: SECTION 1

INDUCTION OF CANCER BY RADIATION AND RISK ESTIMATES

11(1).1 INTRODUCTION

The material presented in this chapter is based on Annex B to ICRP 60, 'Biological effects of ionizing radiation', supplemented by material from two articles, one by Upton (1991) on 'Risk estimates for carcinogenic effects of radiation', and the other by Land and Sinclair (1991) on 'The relative contributions of different organ sites to the total cancer mortality associated with low dose radiation exposure', both of which were prepared as part of the assignment of a Task Group of Committee 1 of ICRP.

Induction of cancer and hereditary effects are the most important classes of stochastic effects of radiation. Stochastic effects result from alterations in normal cells caused by a radiation event. The probability of such changes occurring in a population of cells is proportional to the dose at low doses where, on the average, less than one event per sensitive target in a cell occurs. A dose of 1 mGy of 1 MeV gamma rays and 1 MeV neutrons gives rise to about 1 (and occasionally more than 2) and 10^{-2} tracks per cell nucleus respectively. From the point of view of carcinogenic mechanisms, the probability of energy being deposited in a particular 2 nm segment of DNA (there are about 2×10^9 segments in the DNA molecule) is 10^{-9} or less for both types of radiation. However, per unit length, more energy is deposited for neutrons than for gamma rays. Thus if alterations of a particular 2 nm segment may play a vital role in the subsequent carcinogenic process, the biological changes resulting from energy deposition in that segment due to neutrons will be greater. Raising the dose within the tens of mGy range increases proportionately the number of cells that can be affected by single events. At higher doses when more than one event is likely to occur per sensitive target of dimensions 2-100 nm, more complex dose-response relations (like linear-quadratic) can occur.

Initial events that result in a malignant transformation may themselves involve many steps in which radiation or any other external trigger is not necessarily the first. At some time after the initial event a clone of cells with malignant potential may arise, and after further events (in the cells or in their environment) a cancer may develop. These later changes are age-dependent for many cancers. The probability of development of an overt cancer is far lower than that of the initial events because of host defences and the failure of succeeding changes required for the expression of the malignant potential of initiated cells.

The minimum latency period for induction of acute myeloid leukaemia (and ^{224}Ra-induced osteosarcoma) in humans is 2 years and for other cancers 5-10 years (Rall *et al.*, 1985). ICRP 60 assumes an average of 10 years. The median latency period is 8 years for leukaemia and 2-3 times longer for most other cancers. The frequency of leukaemias and osteosarcomas declines after a peak at 5-7 years to small excess values after 20 years or more. For other cancers the relative risk remains approximately constant with time in those persons irradiated in adulthood. There may be a decreasing relative risk in persons exposed during childhood, as well as a decline in frequency with time for lung cancer from radon exposure (NCRP, 1984a,b; NAS, 1988), some cancers in patients irradiated for ankylosing spondylitis (Darby *et al.*, 1987) and in radiation-induced thyroid cancer (Shore *etal.*, 1985).

Experimental systems which use neoplastic transformations of cells in culture or induced tumours (benign and malignant) in animals as end-points are useful for studying the nature of the dose-response curves and the influence of different modifying factors. On the assumption that initiation leading to oncogenesis may occur through induced somatic mutations, studies in *in vivo* mutagenesis systems also give important information.

For low LET radiations, protracted (low dose rate) or fractionated exposures are less effective for many biological end-points including cancer than single high dose rate exposures. For high LET radiations, the effects at low dose rates or due to fractionation may be similar to those at high dose rates in some cases, while in others, low dose rate or fractionation is more effective [Fig. 11(1).1]. Certain chemicals like TPA (a component of croton oil) or asbestos may increase the rate of radiation-induced cell transformation (Han and Elkind, 1982) while others like vitamin A analogues may decrease it. The effects of high LET radiation are generally less influenced by such factors (Sinclair, 1987).

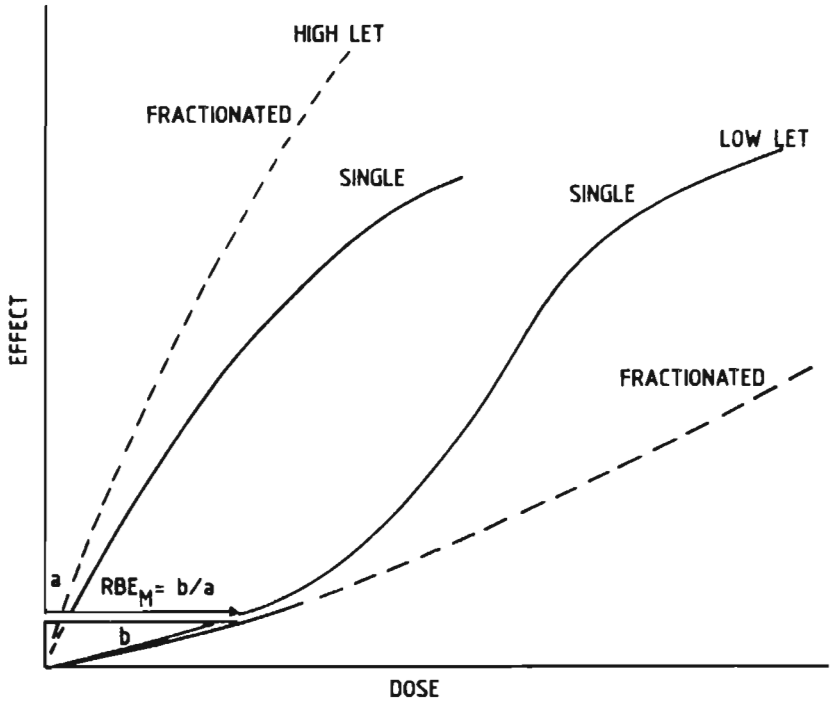

Figure 11(1).1. Shape of dose-response curves for high LET and low LET radiation plotted on linear axes (reproduced with permission from the Commission of the European Communities, Brussels).

The risk of cancer is assumed to be broadly proportional to the number of irradiated cells at risk in a given organ or tissue (but between species there is no correlation with body size). In certain cases an organ like liver or lung can be irradiated non-uniformly with 'hot' (very active) particles irradiating only a portion. The average tissue dose would be much smaller than the dose in the vicinity of the hot spot. Both experiment and theory indicate that hot spots are less effective carcinogenically than the same amount of material spread uniformly and delivering a lower but uniform dose (Little and O'Toole, 1974; NCRP, 1975; NAS, 1976).

11(1).2 CANCER INDUCTION BY RADIATION: DOSE-RESPONSE RELATIONSHIPS

Most human information has been obtained at doses above 0.1 to 0.2 Gy (and at high dose rates).

Cancer initiation is associated with the induction of lesions in the genomic DNA that result in specific gene losses and/or changes in gene structure and activity (Bishop, 1987; Ponder, 1988; Reik and Surani, 1989). Recent studies with radiation or chemically induced rodent tumours are beginning to shed light on the genes that might be involved in this initiation process (Janowski *et al.*, 1990; Sloan *et al.*, 1990; Kumar *et al.*, 1990). Mammalian systems possess enzymes that recognize and remove lesions from DNA. *In vitro* studies indicate that dose rate effects on cellular low LET radiation responses may be associated with the activity of certain DNA repair systems. Some cellular repair systems appear to operate more effectively after low dose rate exposure; it thus seems that radiation carcinogenesis will be subject to a dose rate factor. At very low doses, when the number of energy loss events in critical cellular target volumes is equal to or less than that of the targets themselves, dose rate dependent cellular processes become irrelevant. Overall, the dose rate effectiveness factor to be applied to estimates of cancer induction from data at lower doses should be lower than those required to be applied to observations at higher doses.

For a linear-quadratic dose-response [Fig. 11(1).2], at low doses and dose rates the response is effectively linear, the effect per unit dose being constant ($E/D = \alpha$, a constant). The effect increases more rapidly thereafter, i.e. the effect per unit dose increases linearly with the dose as the quadratic term becomes operative ($E/D = \beta D$). At still higher doses the effectiveness declines again due to cell killing, reducing the number of cells at risk.

ICRP 60 has briefly summarized different recent reviews of the question of dose and dose rate effectiveness factors.

The NCRP (1980) defined the dose rate effectiveness factor (DREF) as the ratio of the slope of the linear no threshold fit to high dose, high dose rate data, to the slope of the linear no threshold fit to low dose rate data [i.e. α_L (curve B) to α_1 (curve D in Fig. 11(1).2)]. It is evident from this curve that

$$\alpha_L D = \alpha_1 D + \beta D^2$$

(where curves A and B meet initially). Thus

$$\text{DREF} = \alpha_L / \alpha_1 = 1 + \beta/\alpha_1 \, D.$$

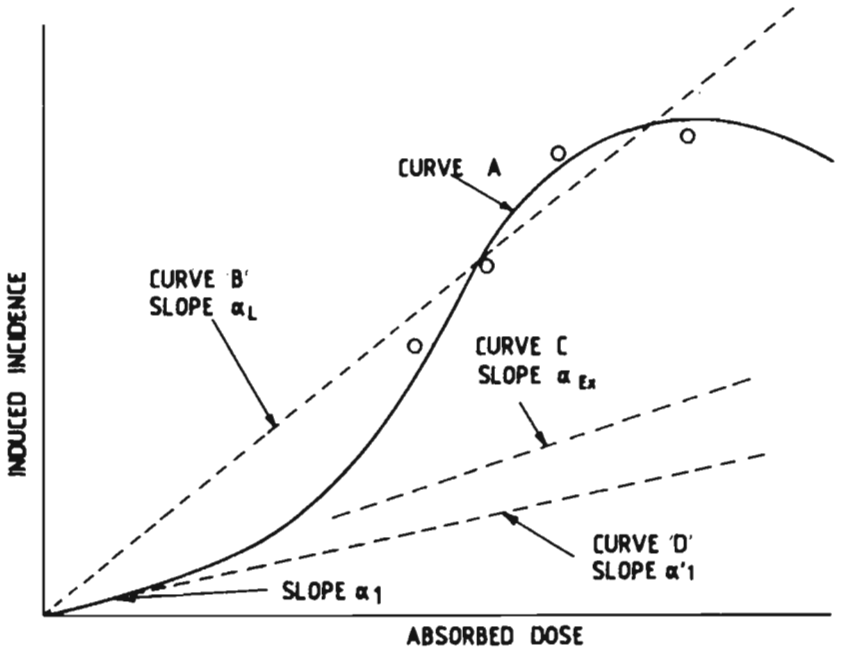

Figure 11(1).2. Schematic curves of incidence versus absorbed dose (reproduced with permission from the National Council on Radiation Protection and Measurements, Bethesda).

The slope of the experimental curves α_{Ex} (curve C) will approximate α_L when the dose, dose rate (and DREF) are high, and α_1 when the dose and dose rate are low (and the experimentally determined DREF close to unity). Thus the observed DREF in experimental situations will depend on the dose range and dose rate over which the studies are performed. It will be smaller if these ranges are small. At the maximum in curve A (which bends over due to cell killing), DREF will also be a maximum. ICRP 60 calls this factor as Dose and Dose Rate Effectiveness Factor (DDREF).

The NCRP report provides tables of data on DREF values in a wide variety of experimental systems, including tumours and life-shortening in animals. The report concluded that values of DREF in experimental systems varied between 2 and 10.

Recent information on the A-bomb survivors suggests that for leukaemia the dose response fits a linear-quadratic relationship best with a DDREF of 2 (NAS, 1990). For the solid cancers together, linearity provides the best fit (NAS, 1990). The most recent reanalysis (Pierce and Vaeth, 1989), however, suggests that there is little difference in dose- response relationship for any of the different cancer sites including leukaemia. Some human experience shows little evidence of fractionation effects while others indicate DDREF values of 3-4. Japanese data indicate a factor of not much more than 2. Most recent surveys such as UNSCEAR (1988), NUREG (1989) and BEIR V (NAS, 1990) recommend values of DDREF generally in the range of 2-3. Clinical data on fractionated *versus* single exposures do not give conclusive evidence for any specific DDREF. A brief discussion of the subject together with references to the literature will be found in ICRP 60.

ICRP 60 has recommended that for radiation protection purposes the value of 2 be used for the DDREF, while recognizing that the choice is somewhat arbitrary and may be conservative. ICRP 60 also concludes that most of the available data do not support the idea of a threshold for carcinogenesis. Nevertheless, on statistical grounds, a threshold cannot be ruled out with certainty; if thresholds do exist, their values must be less (perhaps much less) than 0.2 Gy for most human cancers.

11(1).3 CANCER INDUCTION AFTER EXPOSURE TO HIGH LET RADIATIONS

The dose-response relation is typically concave upwards for single doses of low LET radiation and concave downwards for high LET radiation (Fig. 11(1).1) for stochastic effects. In contrast, fractionated doses (or low dose rates) are less effective for low LET radiation, while for high LET radiation they are as effective as or more effective than single doses. Evidently, the RBE [*b/a* in Fig. 11(1).1] increases with decreasing dose but reaches a constant value, RBE_M, at low doses where both high and low LET radiation dose-response curves become linear.

In some cases high LET radiations (especially fission neutrons) have increased effectiveness at low dose rates and/or fractionation, even initially (i.e. an initial linear slope steeper for low dose rate compared to high dose rate). This has been termed the 'reverse dose rate effect' and is not yet well understood. The increased effectiveness is usually small, 1.5 to 2.5 times (Ullrich, 1984).

Experimental RBE_M values for fission neutrons and alpha particles show a wide range (10-50 or more) for various stochastic end-points [Table 11(1).1]. Alpha particles have values of RBE_M about the same as or somewhat less than those of fission neutrons. It is difficult to recommend a typical single value of RBE_M for use in deriving quality factors.

In cases where the radionuclide does not penetrate the cell, Auger electron emitters are very inefficient in producing biological effects because of the short range of the low energy electrons. For those Auger electron emitters which penetrate the cell but are not incorporated into the DNA, RBE values of 1.5-8 have been reported (Kassis *et al.*, 1988). But if Auger electron emitters such as ^{125}I are incorporated in DNA, RBE values of 20-40 have been found for end points such as cell transformation (Chan and Little, 1986), and calculations of energy deposition patterns confirmed that these high values are to be expected (Charlton, 1988; Baverstock and Charlton, 1988).

At low doses, 200 kV X-rays are about twice as effective as gamma rays for many end points (Bond *et al.*, 1978). Fast electrons may be even less effective than gamma rays (ICRU, 1986; Sinclair, 1985).

The choice of quality factors is based on appropriate 'average' RBE values for stochastic end-points with respect to a 'reference radiation' (broadly defined to include all low LET radiations, and taking the more relevant end-points into account). The quality factor is applicable only for stochastic effects in the dose range up to tens of mGy. Thus, the applicable RBE values to be accounted for in the assessment of Q are usually values of RBE_M only (Sinclair, 1985; ICRU, 1986; NCRP, 1990). For higher doses (several Gy) RBE values for deterministic effects must be considered. Reference may also be made to Chapter 10, Section 2 which summarizes the contents of ICRP Publication 58, 'RBE for deterministic effects' (1989).

11(1).4 ESTIMATES OF PROBABILITY FOR CARCINOGENIC EFFECTS

Since the publication of ICRP 26 (1977), new information on radiation-induced cancer risks in humans has emerged and new experimental data on both laboratory animals and cultured cells have become available.

These developments have been summarized in reports of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1977, 1982, 1986, 1988) and the Committee on the Biological Effects of

Ionizing Radiation (BEIR V) of the US National Academy of Sciences (NAS, 1990).

The principal new information comes from the continued assessment of more than 90,000 survivors of the A-bomb in Japan (76,000 with DS86 dosimetry).

(Author's note: DS86 stands for Dosimetry System 1986 which has superseded T65D, Tentative Dosimetry 1965. DS86 was developed by the Radiation Effects Research Foundation, RERF, a joint US-Japanese organization which is the successor to the Atomic Bomb Casualty Commission, ABCC.)

Estimates of the probability of cancer deaths for the period 1950-85 are increased over earlier estimates because of :

(a) Increase in the number of excess solid cancers observed in the additional follow-up period (~135 in 1975 compared with ~260 in 1985; the corresponding figures for leukaemia are 70 and 80) (Shimizu *et al.*, 1988, 1990). The longer follow-up is particularly relevant for cohorts of survivors irradiated early in life and who have now moved into age groups of increased baseline cancer rates (Preston and Pierce, 1988). The total follow-up period now corresponds to 2.2 million person-years. About 60% of the irradiated population survives at the present time.

DS86 has given values for the 'shielded kerma' (kerma to each individual after gamma rays and neutrons have passed through the shielding of house or other structure estimated for that individual at the time of the explosion). The organ dose depends on the shielded kerma but the ratio between them is different for each organ.

(b) The new dosimetry (DS86 *vs* T65D) (Roesch, 1987) which increases the probability values by between 1 and 2 times depending on the tissue site and the allowance made for neutron RBE. The difference between probabilities using DS86 and T65D dosimetries is based on the UNSCEAR determination of organ dose equivalents in 1977, which used T65D with a neutron RBE of up to 20, and the UNSCEAR determination in 1988 using DS86 with neutron RBE no longer critical. The tissue gamma dose is now estimated to be higher at Hiroshima and slightly lower at Nagasaki than heretofore. Further, the difference in overall risk between Hiroshima and Nagasaki observed earlier is no longer statistically significant.

(c) Preference for the 'relative' or 'multiplicative' rather than the 'absolute' or 'additive' risk projection model for projecting the observed

number of solid cancers to lifetime values. For example, the increase in solid cancers observed during the follow-up period 1975-85 has been roughly proportional to the increase in baseline rates associated with the aging of the cohort, while the excess relative risk of fatal cancer has remained comparatively constant over time, indicating the better validity of the multiplicative model.

Although the data for leukaemia mortality conform to a linear-quadratic function (NAS, 1990), the relationship for other cancers reveals no significant departure from linearity in the range below 3 Gy.

Further information has come from two other major populations, viz.

(a) 14,106 patients followed up in UK after radiotherapy for ankylosing spondylitis (average follow-up period 23 years, maximum 48 years), equivalent to 184,000 person-years (Darby *et al.*, 1985, 1987). There are marked differences between the Japanese and the ankylosing spondylitis samples and their exposures. Whereas the former was whole body exposure, in the latter case, irradiation was confined to the spine and the pelvis; further, doses within the bone marrow itself would be variable. Dose data for many of the individual patients are absent. Moreover, the study sample was a selected subgroup, and the average follow-up time was fairly short. It is not therefore easy to compare risk estimates in the two cases (Lewis *et al.*, 1988; Upton, 1991). The excess risk per unit dose has generally been smaller in the case of ankylosing spondylitics than for the Japanese survivors; however, they differ by less than a factor of 2 for leukaemia and about 2 for all cancers together;

(b) Study of cancers in women treated for carcinoma of cervix who developed cancers at other sites. Here the agreement of the risk estimates between this series and those for the Japanese survivors and the ankylosing spondylitics is less satisfactory (Boice *et al.*, 1985, 1987, 1988).

A number of other populations that have received radiation (mainly therapeutic, and also in some cases diagnostic) provide additional information:

(i) Children treated for leukaemia (Tucker *et al.*, 1984; Meadows *et al.*, 1985);

(ii) Patients treated for Hodgkins disease (Tucker *et al.*, 1984);

(iii) Patients treated with intravenously injected ^{224}Ra for tubercu-

losis and ankylosing spondylitis (Mays and Spiess, 1984; Spiess *et al.*, 1989);

(iv) Patients treated for ovarian cancer (Reimer *et al.*, 1978);

(v) Patients treated for tinea capitis. In this group which received scalp irradiation, an excess incidence of thyroid cancers has been reported; average thyroid doses would be rather small, < 100 mSv (Ron and Modan, 1984; Shore *et al.*, 1984; Modan *et al.*, 1989; Ron *et al.*, 1989; NAS, 1990);

(vi) Radiation-induced breast cancers in women receiving diagnostic multiple chest fluoroscopies in the course of therapy for tuberculosis and women receiving radiotherapy for acute post-partum mastitis and chronic breast diseases (Boice and Monson, 1977; Land *et al.*, 1980; Howe, 1984; Shore *et al.*, 1986; Miller *et al.*, 1989; Hrubec *et al.*, 1989; Hildreth *et al.*, 1989; Boice *et al.*, 1990);

(vii) Patients injected intravascularly with thorotrast for radiography (NAS, 1988);

(viii) Exposure of children *in utero* to diagnostic X-rays (Stewart *et al.*, 1958; Stewart and Kneale, 1970; Mole, 1974, 1990; Monson and MacMahon, 1984; Harvey *et al.*, 1985; Bithel and Stiller, 1988); and

(ix) Exposure of miners to radon and daughters in Canada, Czechoslovakia and USA (see Chapter 42).

Most of these studies do not provide sufficient quantitative information for general risk estimates but provide additional data to support risk estimates for cancers in specific organs. Studies on occupationally exposed groups have not also yielded any significant data on which to base risk estimates (Modan, 1991).

Chronic lymphocytic leukaemia and Hodgkins disease have not been observed to result from radiation exposure, in spite of their relatively high baseline prevalence among haematological cancers as a group.

Salient features of some of the larger such study populations are summarized in Tables 11(1).2. and 11(1).3.

The Japanese data provide the most comprehensive data base (76,000+ in the DS86 cohort) where both sexes and all ages are represented, dose

range is extensive, the exposure is to the whole body, and there is an internal control group. Risk estimates based on the study of other populations discussed earlier agree broadly with the Japanese data (Upton, 1991)

11(1).4.1 NEW LABORATORY FINDINGS SINCE 1977

These include animal experiments; cytogenetic and molecular studies on radiation- and chemically-induced neoplasms; importance of cellular repair and recovery processes in radiation response (Cox, 1982; Arlett *et al*, 1989; Thacker, 1991); role of specific chromosomal changes in radiation oncogenesis and their association with oncogene activation and/or gene losses (Silver *et al.*, 1989); radiation-induced chromosomal changes in human lymphocytes at low doses (< 0.1 Gy) (Edwards *et al.*, 1989); *in vitro* cellular studies indicating that mutations in a number of genes principally involve DNA deletion, and to some extent DNA base changes (Thacker, 1986; Glickman *et al.*, 1987); importance of double strand break (dsb) repair in cellular recovery and its influence on dose rate effects (Debenham *et al.*, 1987; Evans *et al.*, 1987; Wlodek and Hittelman, 1987); 'adaptive response' induced by low doses that reduces the frequency of chromosomal damage (Wolff *et al.*, 1989); the influence of dose rate, LET and extraneous modifying factors; the distribution of induced tumours in relation to time after irradiation and attained age.

11(1).5 METHODOLOGICAL FACTORS AFFECTING PROBABILITY ESTIMATION

11(1).5.1 Multiplicative and Additive Models for Projection of Probabilities

Since the period of observation of an exposed population sample rarely extends to a full lifetime, it is necessary to project the estimate of probability of cancer induction for the period of observation to the lifetime of the exposed population, in order to obtain the full lifetime risk. The absolute (or additive) risk projection model predicts a constant excess of induced cancer throughout life unrelated to the spontaneous rate of cancer while the relative (or multiplicative) model predicts that the excess of induced cancers will increase with time as a constant multiple of the spontaneous or natural rate of cancer and consequently increase with age in that population. Both forms of response occur after a minimum latency period.

The population surviving the Japanese A-bombs still contains many people irradiated in childhood or *in utero* who are now attaining the age when cancer and other diseases become prevalent. About three-fifths of the population survives at the present time. Thus, to obtain an estimate of the lifetime risk, the experience of the cohort so far must be projected forward in time, taking into account the age structure of the population and the age-dependent force of mortality from causes unrelated to radiation exposure, as well as from radiation-induced cancer.

Let $q_0(a)$ denote the age-specific death rate from all causes in a particular non-irradiated population, and let $h_{D,A0}(a)$ denote the age-specific, excess death rate per year associated with exposure to dose D at age A_0. (Note that for $a < A_0$, $h_{D,A0}(a) = 0$) The total death rate, then, is given by:

$$q_{D,A0}(a) = q_0((a) + h_{D,A0}(a)$$

$L_{D,A0}(a)$, the probability of surviving to age a (years), given exposure to dose D at age A_0, is given by:

$L_{D,A0}(a) = 1$ for $a < A_0$ (exposure at age A_0 implies survival until age A_0)

$$L_{D,A0}(a) = L_{D,A0}(a-1).\{1 - q_{D,A0}(a-1)\}$$

for $a = A_0 + 1, \ldots\ldots$

(survival at age a implies survival to age $a-1$ and precludes death at age $a-1$).

The annual probability of death from any cause at age a is

$$L_{D,A0}(a) \cdot q_{D,A0}(a)$$

and the annual probability of a *radiation-induced death* at age a is

$$L_{D,A0}(a) \cdot h_{D,A0}(a)$$

Thus the lifetime probability of a death due to radiation exposure $U_{(A0,D)}$ is

$$\sum_{a=A_0}^{max\ age} L_{D,A0}(a) \cdot h_{D,A0}(a)$$

In the *simple additive model* $h_{D,A0}(a)$ does not vary for $a \geq A_0 + m$, where m is the minimum latent period of 10 years or so:

$$h_{D,A0}(a) = \begin{cases} 0 \text{ for } a < A_0 + m \\ K_{D,A0} \text{ for } a \geq A_0 + m \end{cases}$$

In the *simple multiplicative model*, $h_{D,A0}$ (a) varies with a as a constant multiple of the baseline, age-specific cancer rate for a non-exposed population, $q_{0,(cancer)}(a)$:

$$h_{D,A0}(a) = \begin{cases} C_{D,A0}\, q_{0,(cancer)}(a) \text{ for } a \geq A_0 + m \\ 0 \text{ for } a < A_0 + m \end{cases}$$

In the above formulations $K_{D,A0}$ and $C_{D,A0}$ depend on D and A_0 but not on a, and A_0.

$q_0(a) = q_{0,(cancer)}(a) + q_{0,(non,cancer)}(a)$. $q_{0,(cancer)}(a)$ is the component of $q_0(a)$ that pertains to the specified cancer being considered.

(BEIR V (NAS, 1990) used a modified multiplicative projection model which included terms dependent on time since exposure, which enabled a decrease in risk with time at longer times to be included in the formulation.)

11(1).5.2 Cancer Incidence vs Mortality

Most epidemiological data refer to cancer mortality while data on incidence are relatively sparse. The incidence is usually a multiple of mortality for tumours, this multiple depending strongly on the level of medical care in each country.

11(1).5.3 Biological Factors Affecting Cancer Induction

The incidence of radiation-induced fatal cancer varies with age at exposure and age at attainment depending on the tumour type. In general, younger persons are more susceptible for most cancers. For example, for the female breast, susceptibility is greatest in the very young female, declining through adult life and virtually disappearing if exposure occurs after menopause (Tokunaga *et al.*, 1984; Shimizu *et al.*, 1988, 1990). A similar age-related trend is shown for thyroid cancer. For leukaemia, the risk is higher during prenatal development, infancy and childhood (same risk factor for all these 3 age groups) than in adolescence or early adult life; it also appears to increase in the older age group. Infants and children are 2-3 times more sensitive for thyroid cancer induc-

tion than adults (Shore *et al.*, 1985). For skeletal cancers, however, the susceptibility seems to be the same at all ages (Mays and Spiess, 1984).

In the atomic bomb survivors, the dose-dependent excess of cancers appears thus far to be approximately the same after prenatal irradiation as after irradiation during the first 10 years of life (Yoshimoto *et al.*, 1988; Upton, 1991).

For most cancers except leukaemia (especially for thyroid and breast cancers), females are somewhat more sensitive than males. The excess absolute risk for all cancers (including leukaemia) is about 20% higher for women. (There are sex differences in spontaneous cancer incidence also, probably related to hormone dependent promotive factors; for example, females are 3 times more susceptible to thyroid cancer.)

The minimum latency period for leukaemia and osteosarcoma is 2-5 years; the incidence reaches a peak within the first decade, and subsequently declines. For other cancers, the minimal latent period is 10 years, after which the relative risk remains constant. For the ankylosing spondylitics, the overall excess mortality reached a peak during the second decade after which it appeared to decline (Darby *et al.*, 1987); the cause for the difference between this group and the Japanese survivors is not clear.

11(1).5.4 Sensitive Sub-populations

There are no epidemiological data which identify adult sub-populations that are hypersensitive to radiation-induced cancers. In the case of exposure to UV light, patients with the DNA repair deficient genetic disorder, xeroderma pigmentosum (XP), show substantially increased susceptibility to sunlight (UV)-induced skin cancer. Patients with the leukaemia-prone genetic disorder ataxia-telangiectasia (AT) are extremely sensitive to low LET radiation. Cellular studies implicate DNA repair deficiency as the cause (Cox, 1982; Debenham *et al.*, 1987; Arlett *et al.*, 1989). Genetic disorders like retinoblastoma, where tissue-specific cancers may be associated with heterozygosity for 'cancer suppressor genes', may carry increased risk (Knudsen, 1986; Reik and Surani, 1989). It appears that there may be a synergistic effect between ultraviolet light and radiation in the induction of skin cancer (Shore *et al.*, 1984; Albert and Shore, 1986). A similar effect of smoking on the induction of lung cancer by exposure to radon and daughter products in mines has been reported (NAS, 1988).

It appears that in patients treated for cancer by combined therapy with drugs and radiation, the risk of a second, treatment-induced cancer is higher than in those treated with radiation alone (Coleman, 1982; Fry and Ullrich, 1986).

Radiation-induced tumours (such as breast cancer in women) tend to be expressed later in life when tumours from other causes also occur regardless of age at exposure. This suggests that radiation may initiate the process at a young age but completion requires additional steps later in life, some of which are hormone dependent.

11(1).5.5 Influence of Dose Rate, Fractionation and LET

In animals, the carcinogenic effectiveness of low LET radiation generally decreases with fractionation or protraction while that of high LET radiation tends to remain unchanged or even to increase with similar fractionation or protraction (NCRP, 1980; UNSCEAR, 1986; NAS, 1990). Comparable human data are fragmentary.

Patients given diagnostic doses of ^{131}I develop little excess of thyroid cancer, in contrast to the appreciable excess that would be predicted on the basis of the effects of comparable doses of external radiation (Holm *et al.*, 1988). Spatial and temporal differences in dose distribution may account for this discrepancy (NCRP, 1985), since the carcinogenic effects of therapeutic X-irradiation do not diminish on fractionation (Shore *et al.*, 1985).

In animals, the carcinogenic effects of radiation vary with LET depending on the neoplasm and conditions of irradiation (UNSCEAR, 1986). In general, RBE increases with decreasing dose and dose rate, falling in the range of 2 - 30 for duration-of-life exposure, although much higher values have been reported for induction of tumours by fission neutrons (ICRU, 1986).

In humans, data for neutron irradiation are lacking. RBEs for alpha particles for carcinogenic effects on the skeleton and lung are around 20 (ICRU, 1986; NAS, 1988, 1990; NCRP, 1990).

11(1).6 ESTIMATES OF FATAL CANCER PROBABILITIES

ICRP 60 gives a detailed and critical review of the cancer risk estimates (using different models) made by various bodies to-date, including

the United Nations Scientific Committee on the Effects of Atomic Radiation, UNSCEAR (1977, 1988), the US National Academy of Sciences Committee on the Biological Effects of Ionizing Radiation, BEIR I (NAS, 1972), BEIR III (NAS, 1980), BEIR V (NAS, 1990) and the US Nuclear Regulatory Commission, NUREG (1989).

UNSCEAR has used both the additive and multiplicative models. These two models result in somewhat different estimates of lifetime risks of fatal cancers; however, these differences have become smaller in progressively later reports [Table 11(1).4].

BEIR V, as mentioned earlier, used a modified multiplicative projection model. The results show a substantial difference with age at exposure for most cancer groups. Overall, the difference (in estimates) between the sexes is less than estimated by UNSCEAR, females being more sensitive by only 6%.

The estimates based on the additive and the multiplicative models have come closer together in later estimates. Furthermore, the estimates based on the multiplicative model have changed the least, varying by less than a factor of 2 since 1972.

ICRP 60 has developed its own methodology for arriving at detailed risk estimates for five populations (Japan, the USA, Puerto Rico, the UK and China) for the two sexes, different age groups, different organs, and different risk projection models.

The starting point is the age-specific coefficients available from the A-bomb study (Shimizu *et al.*, 1988). Calculations made for the Japanese population involve first a transfer from the observed data and then projection in time using the additive, multiplicative and 'NIH' models (the last being used by an *ad hoc* working group of the US National Institute of Health to develop radio-epidemiological tables). Estimates of the fatal cancer probability after 1 Gy of acute low LET whole body radiation were made for each organ for males and females, and for four age ranges (0-90, 0-19, 20-64 and 65-90 years) and for the five populations mentioned earlier. (For detailed results, reference may be made to ICRP 60; Upton, 1991; Land and Sinclair, 1991.)

The total risks are similar to those found by UNSCEAR. The largest differences in the relative probabilities for a given model between males and females for any important contributor organ are about a factor of 2 and for less important contributor organs a factor of 3. The total risk for all cancers differs between males and females at most by about 50% for

the multiplicative model, females having the greater risk. Sex differences in important organs are not greater (indeed about the same) for the populations of the USA, Puerto Rico, the UK and China (Land and Sinclair, 1991). In general, the sex ratios of radiation-induced cancers at specific sites appear to resemble those of the corresponding cancers in the general population. The relative risks for many epithelial cancers tend to be slightly higher in females. This fact, plus the substantial contribution of breast cancer to the total mortality from all cancers combined, accounts for the significantly higher projected cumulative lifetime excess in females. The relative probabilities vary with age group for a given model by a factor of 2 or 3.

When projections are made for the five different populations, large differences are evident in the contributions for the oesophagus, stomach and breast; however, contributions of all organs are within a factor of about 3 of the average value.

To reduce the effects of national population characteristics and arrive at a representative 'world population', ICRP 60 has simply averaged the relative probabilities of fatal cancer in specific organs as well as the total risk. The deviation of any population from the average ratio is within a factor of 3 to 4 for any organ and less for the average. For example, the total probabilities of fatal cancers for all organs (average of male and female, age 0-90 years, multiplicative model), come out to 10.7, 11.2, 9.5, 12.9, and 6.3 (10^{-2} Sv^{-1}) respectively for Japan, the USA, Puerto Rico, the UK and China, as against the average of 10.1 x 10^{-2} Sv^{-1}. ICRP 60 has further averaged the ratios obtained by the two projection models (multiplicative transfer and additive transfer) and used them as the basis for the relative probabilities of cancer in different organs for a normal world population of all ages from which to derive the detriment.

11(1).6.1 Fatal Cancers in Other Selected Organs

The excess mortality from cancers in A-bomb survivors for the period 1950-1985 resulted largely from leukaemia and cancers of the stomach, lung, female breast, colon and ovary. For certain other sites which have contributed fewer excess deaths in the A-bomb survivors, additional information from other sources is available; these include thyroid, bone, skin and liver, which are often selectively irradiated.

Table 11(1).5 gives the ICRP 60 values of fatal cancer risk (per 10,000 people per Sv) for various organs (rounded values, using a DDREF of 2).

11(1).6.2 Expected Years of Life Lost from Fatal Cancers in Different Organs

Calculations have been made of the expected years of life lost (l) for different ages, populations, sexes, etc. for site-specific and total cancers, as derived from the data of Land and Sinclair (1991). These ratios are similar to the distribution of probabilities of fatal cancer in each organ, except that for leukaemia the number of years lost is higher, reflecting the shorter latency period for leukaemia. For cancers of the breast and ovary, the length of life lost per specific cancer is based on the female data only and is not averaged for males and females. The value of l, the average life lost for all cancers, is obtained by dividing the expected years of life lost for all cancers by the total number of fatal cancers as a group, and comes out to be 15.0 years (14.6 for males, 15.4 for females). For bone surface, liver, skin and thyroid, the values have been arbitrarily set at the same value as l, i.e. 15.0 years. (The gonads are assigned a period of 20 years of life lost on average for severe genetic disorders.) One of the columns of Table 11(1).5 gives the relative length of life lost for various sites, based on the average value of l of 15.0 years.

A brief discussion on skin cancer may not be out of place (Fry *et al.*, 1991). Two types of skin cancer, basal and squamous cell carcinomas, have been associated with exposure to ionizing radiation. As yet, the evidence of an association between ionizing radiation and melanoma, the most malignant type of epidermal cancer, is inconclusive. The ratio of basal cell to squamous cell carcinomas in the Caucasian population exposed only to ultraviolet radiation (UVR) in sunlight is about 5:1 but for those exposed to ionizing radiation it is 10:1. The lethality of skin cancer is very low (0.01% for basal cell carcinoma and 1% for squamous cell carcinoma). The risk of excess radiation-induced skin cancer is influenced by exposure to UVR and is dependent on the skin pigmentation. The greatest risk is for those with a light complexion, the extreme example being Albinism. There is a fifty-fold range of susceptibility among races. The risk of both naturally occurring and ionizing radiation-induced skin cancer is low in black-skinned races. The risk estimates given by ICRP 60 are for Caucasians. It is necessary to make separate risk estimates for areas of the skin exposed to sunlight such as head and neck, and for areas such as the trunk that receive much less UVR. (Persons with the genetic condition Nevoid Basal Cell Syndrome have a high incidence of basal cell carcinoma.) The data for radiation-induced cancer seem to suggest that a relative risk model is appropriate.

The incidence of skin cancer is proportional to the area of skin exposed to ionizing radiation and also to UVR. ICRP 60 arrives at a fatal cancer risk estimate of 2 x 10^{-4} Sv^{-1} based on the relative risk model, taking into account the relative incidence and mortality of the two types of skin cancers. The incidence probability is taken to be 9.8 x 10^{-2} Sv^{-1}.

A summary of the contents of ICRP 59, 'The biological basis for dose limitation in the skin' (1991), dealing with both deterministic and stochastic effects, is given in Chapter 11, Section 4.

11(1).6.3 Morbidity and Detriment

In its evaluation of detriment, ICRP 60 considers the following factors: (a) the risk of fatal cancers in various organs; (b) allowance for differences in latency which will result in differences in values of expected life lost for fatal cancers in different organs; (c) allowance for the morbidity resulting from induced non-fatal cancers, and (d) allowance for the risk of serious hereditary disorders in all future generations.

In attaching a weight to the detriment due to the induction of a curable cancer, importance must be given to the ease of curing some cancers such as skin, the extreme difficulty of curing some others, and the trauma associated with the curative procedures. Some cancers like the breast are probably intermediate between these two situations. Thus to allow for the detriment associated with non-fatal cancers, the detriment of each cancer type includes a non-fatal component weighted according to the lethality fraction k. If in a given tissue there are F fatal cancers, the total number of cancers is F/k. The number of non-fatal cancers is then $(1-k)F/k$, and the total weighted detriment is $[F + k(1-k)F/k]$ or $F(2-k)$. The nominal weighted probability coefficient is then given by multiplying the corresponding fatality probability coefficient by $(2-k)$. For each organ the detriment will be the product of $F(l/l)$ and (2-k). Table 11(1).5 gives the relative contributions of various organs to the total detriment.

For a working population the total fatal cancer risk is taken to be 4 x 10^{-2} Sv^{-1} and the value of F for organs are 80% of those given in Table 11(1).5. The risk for severe genetic effects is estimated to be 0.6 x 10^{-2} Sv^{-1}.

Based on the data given in Table 11(1).5, ICRP 60 has arrived at tissue weighting factors. For simplicity, the values are rounded and grouped into a simple system of 4 classes of weights of adequate accuracy for calculations of effective dose. (The rounding involves no more than a

factor of 2 for any organ.) Tissue weighting factors are given in Table 2.2 of Chapter 2, and are summarized into groups in Table 11(1).6.

11(1).6.4 Uncertainties in Risk Estimates

After listing the various possible factors (which include model misspecification, population differences, dosimetry, sex, age and latency, shape of dose-response relationship, and of course, uncertainties in the base data themselves), ICRP 60 concludes that at this time it is difficult to arrive at a precise measure of overall uncertainty.

11(1).6.5 Cancer Risk following *in utero* Irradiation

This is dealt with in Chapter 13.

11(1).7 SUMMARY OF ESTIMATES OF PROBABILITIES OF BIOLOGICAL EFFECTS

Table 11(1).7 gives a summary of estimates of probabilities of various biological effects induced by radiation.

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Table 11(1).1: RBE_M for fission (or optimum energy, i.e. the most biologically effective energy) neutrons vs gamma rays for stochastic end points
(from ICRP 60, based on ICRU, 1986; Sinclair, 1985.)

Tumour induction	3-~200*
Life shortening (due to tumours)	15-45
Transformation	35-70
Cytogenetic studies	40-50
Genetic endpoints in mammalian systems	10-45

* These values have been subsequently modified to ~15 to ~60 (NCRP, 1990).

Table 11(1).2: Salient characteristics of some of the major study populations evaluated for derivation of numerical cancer risk estimates
(from Upton, 1991, based on NAS, 1990)

Population	Incidence or mortality	Cancer sites	Total cases	Total person-years	Reference
Atomic bomb survivors	Mortality	All	5,936	2,183,335	Shimizu *et al.*,1988
	Incidence	Breast	376	940,000	Tokunaga *et al.*,1987
Ankylosing spondylitis	Mortality	Breast	36	30,770	Darby *et al.*, 1987
		All except leukaemia and colon	563	183,749	Darby *et al.*, 1987
Canadian fluoro-scopy patients	Mortality	Breast	482	867,541	Miller *et al.*, 1989
Mass fluoroscopy	Mortality	Breast	74	30,932	Hrubec *et al.*, 1989
NY postpartum mastitis	Incidence	Breast	115	45,000	Shore *et al.*, 1986
Israel tinea capitis	Incidence	Thyroid	55	712,000	Ron & Modan,1984 Modan *et al.*, 1989
Rochester thymus	Incidence	Thyroid	28	138,000	Shore *et al.*, 1985

Table 11(1).3: Main characteristics of the A-Bomb survivor, ankylosing spondylitis and cervical cancer series
(from Upton, 1991, based on UNSCEAR, 1988)

Characteristic	Atomic bomb survivor series	Spondylitis series	Cervical cancer series
Nature of study	Prospective	Retrospective-prospective	Retrospective-prospective
Sample size	76,000	14,000	83,000
Sex composition	F = 59%	F = 17%	F = 100%
Age at irradiation (years)	0 - >90	>15	<30->70
Average follow-up (years)	28.8	23.0	7.6
Type of control	Internal	National rates	National rates and internal
Dosimetry	Individual (DS86)	Individual for leukaemia.1/15 random sample elsewhere	Mean dose of a sample
Irradiation	Instantaneous, whole-body	Fractionated, non-uniform, partial-body	Chronic, fractionated, partial-body
Dose distribution			
Mean dose (Gy)	0.24	1.9	Inhomogeneous
Range of doses (Gy)	(0.01-6.0)	(0-8.06)	
Person-years at risk	2,183,335	184,000	623,800

Table 11(1).4: Excess lifetime mortality from all cancers, attributable to 1 Gy acute uniform whole body low LET irradiation of the general population[a]

(based on Upton, 1991)

Source of estimate	Probability of death (10^{-2})	
	Additive risk projection model	Multiplicative risk projection model
BEIR I, 1972	1.2[b]	6.2[b]
UNSCEAR, 1977	2.5[b]	-
BEIR III, 1980	0.8[c]-2.5[b]	2.3[c]-5.0[b]
NUREG, 1985	2.9[c]	5.2[c]
UNSCEAR, 1988	4.0[b,d]-5.0[b,e]	7.0[b,e]-11.0[b,d]
BEIR V, 1990	-	8.85[b,f]

a. Values rounded; estimates based largely on follow-up of Japanese A-bomb survivors, analyzed with T65D dosimetry prior to 1988 and with DS86 dosimetry thereafter.
b. Linear dose-incidence model.
c. Estimate based on linear-quadratic dose-incidence model.
d. Estimate for Japanese population, based on age-specific risk coefficients.
e. Estimate for Japanese population, based on age-averaged risk coefficients.
f. Estimate for US population, based on modified multiplicative model.

Table 11(1).5: Relative contributions of organs to the total detriment from radiation exposure
(from ICRP 60)

	Probability of fatal cancer F (per 10,000 people/Sv)	Severe genetic effects (per 10,000 people/Sv)	Relative length of life lost $l/\bar{l}$	Relative non-fatal contribution (2-k)	Product $F(l/\bar{l})(2\text{-}k)$ (per 10,000 people/Sv)	Relative contribution
Bladder	30		0.65	1.50	29.4	0.040
Bone marrow	50		2.06	1.01	104.0	0.143
Bone surface	5		1.00	1.30	6.5	0.009
Breast	20		1.21	1.50	36.4	0.050
Colon	85		0.83	1.45	102.7	0.141
Liver	15		1.00	1.05	15.8	0.022
Lung	85		0.90	1.05	80.3	0.111
Oesophagus	30		0.77	1.05	24.2	0.034
Ovary	10		1.12	1.30	14.6	0.020
Skin	2		1.00	2.00	4.0	0.006
Stomach	110		0.83	1.10	100.0	0.139
Thyroid	8		1.00	1.90	15.2	0.021
Remainder	50		0.91	1.29	58.9	0.081
Gonads	-	100	1.33	-	133.3	0.183
Total	500				725.3	1.000

Table 11(1).6: Tissue weighting factors
(from ICRP 60)

W_T		ΣW_T
0.01	Bone surface, skin	0.02
0.05	Bladder, breast, liver, oesophagus, thyroid, remainder	0.30
0.12	Bone marrow, colon, lung, stomach	0.48
0.20	Gonads	0.20
	Total	1.00

Table 11(1).7: Summary of estimates of probabilities of biological effects
(from ICRP 60)

Effect	Population	Exposure period	Exposure modes	Probability
		Low-LET radiation		
Mental effects				
Reduction in IQ	Fetus	8-15 weeks of gestation	High dose, high dose rate	30 IQ points Sv^{-1}
Severe mental retardation	Fetus	8-15 weeks of gestation	High dose, high dose rate	40×10^{-2} at 1 Sv
Hereditary				
Severe hereditary effects, including multifactorial diseases	Whole population	All generations	Low dose, low dose rate	1.0×10^{-2} Sv^{-1}
Cancer				
Fatal cancers (total)	Workers	Lifetime	Low dose, low dose rate	4.0×10^{-2} Sv^{-1}
Fatal cancers (total)	General population	Lifetime	Low dose, low dose rate	5.0×10^{-2} Sv^{-1}
Fatal cancer (in specific organs)	Workers General population	Lifetime	Low dose, low dose rate	See Table 3.3
Skin (fatal)		Lifetime	High or low dose, low dose rate	2×10^{-4} Sv^{-1}
Aggregated health detriment (in specific organs)		Lifetime	Low dose, low dose rate	See Table 11(1).5
Tissue weighting factors				See Table 2.2
		High-LET radiation		
Cancer and hereditary risks are the same as for low-LET radiation using W_R to assess equivalent or effective dose.				See Table 2.1 for W_R values, $(1\text{-}4)\times10^{-4}$ WLM^{-1}
Radon				
Fatal lung cancers	Workers	Lifetime		(3-10) per Jhm^{-3}

CHAPTER 11: SECTION 2

RADIOSENSITIVITY AND SPATIAL DISTRIBUTION OF DOSE

11(2).1 INTRODUCTION

ICRP Publication 14, 'Radiosensitivity and spatial distribution of dose' (1969) contains the reports made by two Task Groups of Committee 1. Although ICRP 14 is over two decades old, since it has not been superseded, it was thought worthwhile to summarize its contents briefly.

11(2).2 REPORT OF THE TASK GROUP ON SPATIAL DISTRIBUTION OF RADIATION DOSE

The Task Group points out that the (then current) ICRP recommendations on non-uniform exposure for radiation workers do not provide a self-consistent scheme. Thus the risk when the whole body is irradiated must be greater than the risks to parts of the body given the same dose. Yet the dose limit for bone marrow is the same as that for the whole body. The division of the body into a large number of critical organs most of which have an individual dose limit only three times that of the whole body is not a consistent risk assessment scheme. There is also inconsistency concerning the recommendations for skin. The dose limit to the entire skin is 30 rems (300 mSv) in a year. However, in cases of considerable inhomogeneity, which include irradiation of small areas, ICRP recommends that the same value be applied, averaged over an area of 1 cm^2 in the region receiving the highest dose. Yet the hands and forearms, feet and ankles, involving a considerable area of the skin, carry a dose limit of 75 rems (750 mSv) in a year.

No logical self-consistent set of rules for non-uniform exposure is possible with the present 'critical organs' scheme.

(Author's note: The report mentions the desirability of a 'common scale of hurt or suffering', an idea elaborated in ICRP 27 (1977) and ICRP 45 (1985).)

The present report tentatively suggests a scheme for non-uniform exposure based on risk considerations, by subdividing the risk from uniform

whole body exposure into risks to various organs based on quantitative risk data presented in ICRP 8 (1966). ICRP 14 also recognized that the weighting factor for a non-fatal cancer, like that of the thyroid, should not be the same as that of a fatal one like leukaemia (an idea indicated in ICRP 26, but quantified only in ICRP 60). The report mentions the possibility of effects, other than malignancies, such as life-shortening from non-malignant causes.

(Author's note: This concept is not now considered relevant for dose levels of interest in radiation protection.)

One of the basic assumptions implicit in the present recommendations, which lies at the heart of the 'critical organ system', is that exposures of different parts of the body are not synergistic; this is reasonable if the dose is low enough.

In considering the problem of spatial distribution of dose, we can distinguish 3 classes of non-uniformity of dose, viz. (i) partial irradiation of an organ or tissue, when the part irradiated is representative of the whole organ or tissue (e.g. external irradiation of skin or bone marrow); (ii) partial irradiation where the part is not representative of the whole (e.g. internal irradiation from bone-seekers where certain locations and cell types are preferentially irradiated; irradiation of DNA by tritiated thymidine); and (iii) irradiation from radioactive materials in a particulate form.

The Task Group recommends that for partial irradiation of a tissue, where the part irradiated is representative of the whole organ or tissue, dose averaging can be carried out for local doses up to 100 rems (1 Sv) in a year and possibly higher. If only a fraction f of a tissue is exposed, the dose allowed can be $1/f$ of the dose limit for the whole organ. This applies to both skin and bone marrow.

A 'significant area' for skin of 1 cm^2 applicable to irradiation of small areas would seem to be reasonable on grounds of operational convenience, and the Task Group suggests that the present limit of 30 rems (0.30 Sv) in a year, to 1 cm^2 of skin, be increased to at least 100 rems (1 Sv) in a year, provided that the irradiation of the same area year after year is avoided, as far as possible.

In the case of preferential irradiation of certain groups of cells in an organ or tissue, ICRP 14 points out that the parameter 'distribution factor', DF (to convert absorbed dose into equivalent dose) was intended to take care of that circumstance. But this requires a knowledge of the

doses received by various cell types as well as their relative radiosensitivities. Such information is most scanty. Until more information is available, the current ICRP practice of using a mean dose throughout the organ has to be followed. The only exceptions are in the case of the epithelium of the gut which is taken as a critical tissue in calculating the dose from radioactive material in the gut content and the epithelium of the bronchus when calculating the dose from inhaled radon and daughter products.

The Task Group discusses exposure from radioactive material in particulate form, with special reference to the problem of non-uniform deposition of plutonium with high concentrations in the pulmonary lymph nodes following inhalation of plutonium particulates. It concludes that the same energy absorption might well be less effective when distributed as a series of 'hot spots' than when uniformly distributed. Thus a mean tissue dose would probably introduce a factor of safety.

(Author's note: The correctness of this conclusion has been validated subsequently; see, for example, ICRP 60).

It also concludes that there is no case for attempting to set up highly elaborate rules for spatial non-uniformity of exposure. Dose limits are put forward as largely arbitrary norms of good practice and there is virtue in recommendations which are administratively simple and have a degree of flexibility.

11(2).3 REPORT OF THE TASK GROUP TO CONSIDER THE RELATIVE RADIOSENSITIVITY OF DIFFERENT TISSUES

In view of the extensive body of information that has been obtained subsequent to the publication of this report, the report is not discussed here, except to indicate its contents.

The kinds of somatic damage which have to be considered in setting dose limits are cataract, impaired fertility, tumour induction, and defective development of the fetus. The possibility of impaired function of organs other than the gonads is uncertain and can be disregarded. The evidence for life-shortening from effects other than tumour induction is inconclusive.

The dose-response for each different kind of effect should be considered on its own merits. In humans there is good evidence that the relation is steeply sigmoid for opacification of the lens and impaired fertility (in

the latter case there is a long tail due to a small fraction of persons in each sex who are much more sensitive than the average) with definite thresholds. Available scanty evidence is in agreement with the working hypothesis that cancer induction in man is linear with dose.

There is no general hypothesis about carcinogenesis which would allow predictions of tissue sensitivity to tumour induction by radiation. Empirical evidence, however, enables a classification of organs into broad classes of radiosensitivity.

No attempt has been made to define the concepts of 'organ' and 'tissue' which have no unambiguous boundaries in some cases. (Author's note: Even ICRP 60 does not explicitly mention the distinction between the two.)

The sensitivity of individual organs to tumour induction in the fetus and the child is not necessarily greater than in the adult, and in some organs it seems as if benign tumours characteristically follow irradiation in childhood, whereas malignant tumours follow irradiation in adult life.

The desirability of developing a unified 'scale of degree of suffering' is pointed out, but no attempt has been made to construct it.

The concept of critical organ is administratively convenient and in some circumstances logically justifiable, but it does not allow summation of risks according to the relative radiosensitivities of the irradiated tissues.

The report has five detailed appendices, dealing with (i) Radiation cataract in man; (ii) Radiation and human fertility; (iii) The relative sensitivity of different tissues to tumour induction by radiation: the human evidence; (iv) The derivation of numerical values for dose limits: an example for discussion; and (v) Further information which could be helpful.

CHAPTER 11: SECTION 3

A REVIEW OF THE RADIOSENSITIVITY OF THE TISSUES IN BONE

11(3).1 INTRODUCTION

ICRP Publication 11 is titled 'A review of the radiosensitivity of the tissues in bone' (1968). Although the report is nearly 3 decades old, since it has not been superseded, it was thought worthwhile to summarize its contents briefly. ICRP 19, 'The metabolism of compounds of plutonium and other actinides' (1972), ICRP 48, 'The metabolism of plutonium and related elements' (1986) and ICRP 20, 'Alkaline earth metabolism in adult man' (1973) also deal with the subject of radiosensitivity of the tissues in bone and are of relevance in this connection. The former two are summarized in Chapter 23, Section 3, while ICRP 20 is summarized in Chapter 23, Section 2. ICRP 30, Part 1, 'Limits for the intake of radionuclides by workers, part 1' (1979) gives a detailed account of bone dosimetry. Chapter, 23, Section 1 summarizes the methodology and computational technqiues discussed in ICRP 30, Part 1. It will be seen that many of the basic principles enunciated in ICRP 11 have been retained in ICRP 30.

11(3).2 THE LOCATION AND RELATIVE NUMBERS OF RADIOSENSITIVE CELLS AND OF CELLS NOT CONSIDERED TO BE RADIOSENSITIVE IN BONE AND BONE MARROW

The available biological data suggest that induction of malignant changes in bone poses the most significant hazard since other crippling degenerative changes have not been seen when the dose is less than the carcinogenic dose. This statement is based on two extensive studies of persons with body burdens of radium (Evans, 1966, 1967; Finkel *et al.*, 1964) and also studies on experimental animals. Many of the dial painters had ingested mixtures of ^{228}Ra and ^{226}Ra. In the Chicago series, radiographic changes of moderate severity were not seen below 0.1 μCi (3.7 kBq) but increased sharply when the current body burden exceeded 0.32 μCi (11.

kBq) ^{226}Ra. The lowest body burden at which malignancy was noted was 0.167 μCi (6.2 kBq) (Finkel *et al.*, 1964). In the Boston series no clinically observable symptoms occurred for residual body burdens less than 0.5 μCi (18.5 kBq) Ra PRE (PRE = pure radium equivalent; this is to normalize the bone dose from a mixture of ^{228}Ra and ^{226}Ra in terms of dose from ^{226}Ra alone). The effects noted were osteomyelitis, spontaneous fractures, osteogenic sarcomas and carcinoma of the paranasal sinuses or mastoids (Evans, 1966). In the two series a total of at least 41 cases of osteosarcoma and 20 of paranasal sinus carcinoma were noted but only one confirmed case of myeloid leukaemia.

11(3).2.1 The Cells at Carcinogenic Risk

Carcinogenic risk is associated with the proliferative potential of the cells. In bone such cells are: (i) osteogenic cells on bone surfaces; (ii) haematopoeitic marrow; and (iii) certain epithelial cells close to bone surfaces.

11(3).2.1.1 Osteogenic Cells of Bone

Bone is a specialized form of connective tissue, much of which is hard owing to the deposition within a soft organic matrix of a complex mineral substance largely composed of calcium, phosphate, carbonate and citrate. The surfaces of bone are covered with non-mineralized layers of connective tissue called periosteum on the external surface and endosteum on the surface lining the bone cavities and the Haversian canals throughout the mineralized bone in which run the blood vessels. Within the mineralized bone are smaller spaces, lacunae, in which lie single bone cells, the osteocytes, connected with one another throughout the matrix by fine processes running in the bone canaliculi (Fig. 11(3).1). The bone cells are of different types. The stem cells differentiate into the non-dividing functional cells, viz. the osteoblasts (responsible for bone formation) and the osteoclasts (responsible for bone removal). The layer of cells on bone surfaces (the osteogenic layer) varies in thickness and proliferative potential. Adjacent to the bone surfaces are the osteoblasts. Behind these are the osteoprogenitor cells or preosteoblasts. The area of bone covered by the periosteum is less by a factor of more than 10 than that covered by the endosteum, since the latter covers the surface of all trabeculae. Except in the young, resorption and apposition are more active on trabecular surfaces than on periosteal surfaces and within Haversian systems. The cells at greatest carcinogenic risk are the osteoprogenitor cells, particularly those on the endosteal surface and associated with marrow spaces.

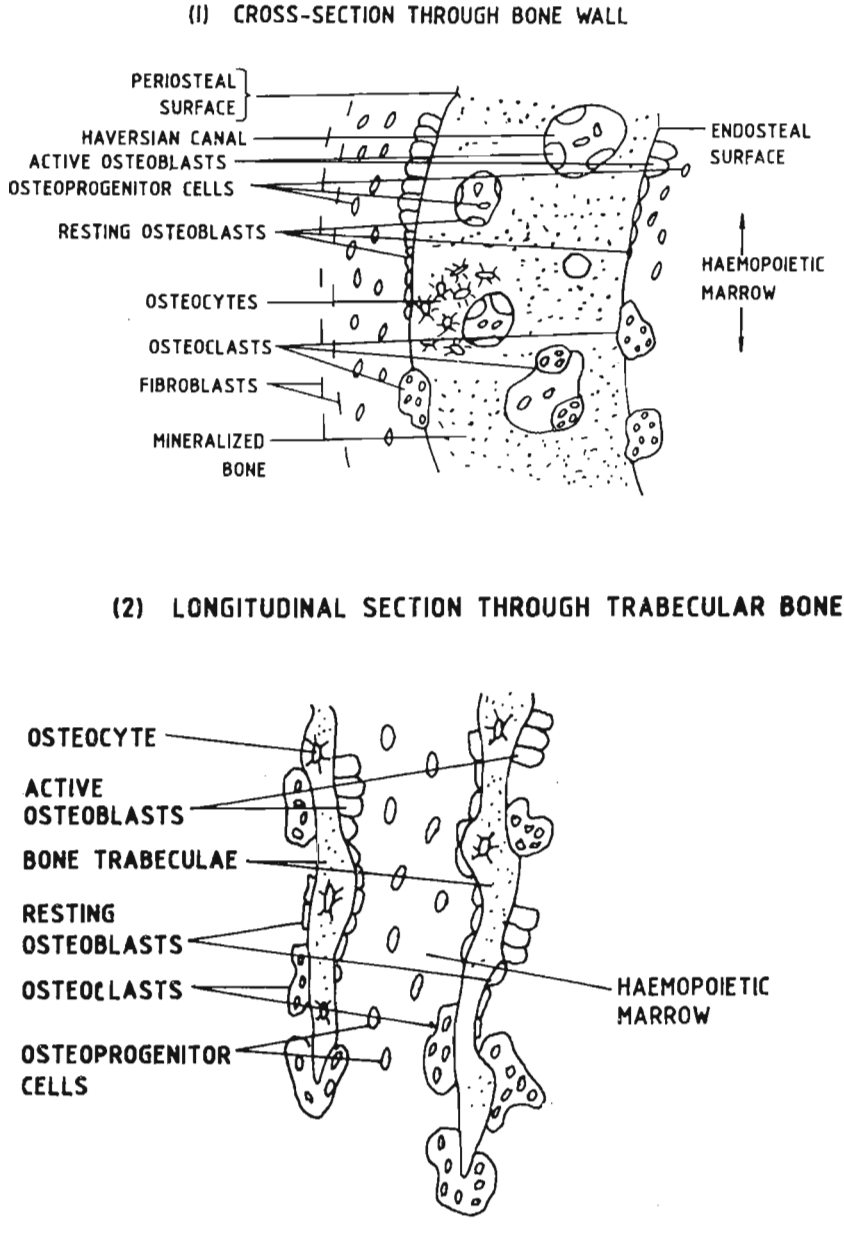

Figure 11(3).1.Diagrammatic representation of bone structure (not to scale) (from ICRP 11).

11(3).2.1.2 The Haematopoeitic Cells of the Bone Marrow

Haematopoeitic stem cells originate in the bone marrow. In children they are randomly distributed in active marrow throughout bone and in adults in active marrow within trabecular bone.

11(3).2.1.3 Epithelial Cells Close to Bone Surface

Cells at risk are the stem cells with greatest proliferative potential. In human skulls carcinomas arise in the mastoid air cells and paranasal sinuses; these are among the few areas in the human skeleton where epithelium is directly adjacent to bone. The total thickness of the epithelium and connective tissue in paranasal sinuses is 50-100 μm and within the range of alpha particles emerging from the underlying bone (Hasterlik *et al.*, 1964).

Death of osteocytes, osteoclasts and cells of the vascular endothelium from large doses of radiation may be responsible for fractures, fibrosis, osteoporosis and osteosclerosis (Jee, 1962).

11(3).3 THE LOCALIZATION OF BONE-SEEKING RADIONUCLIDES

The radionuclides that concentrate particularly in bone may be divided into four groups to describe their pattern of distribution, viz (i) the alkaline earths; (ii) plutonium and thorium; (iii) americium and other transuranics (about which less is known); and (iv) phosphorus and carbon.

The alkaline earths concentrate mainly in bone. Phosphorus and carbon occur in all tissues but are concentrated in bone minerals. The behaviour of rare earths and transuranics depends partly on the form in which they are present when they reach the circulation. In the monomeric form and in microgram amounts they are bound rapidly to transferrin to form a stable complex concentrating mainly in bone. In the polymeric form the concentration would be in liver and spleen.

11(3).3.1 Alkaline Earths

Calcium, strontium and radium are associated with mineral bone and behave similarly in their distribution pattern. Even in the adult, bone is always undergoing apposition (laying down of new mineral matrix and its calcification) and resorption (removal of mineral and matrix), so that the distribution of currently administered alkaline earths is uneven throughout

the bone. In addition to apposition and remodelling, there is a constant exchange between the minerals on the crystalline surfaces and the elements in the circulating blood, one part of the exchange being rapid and the other part slower.

Radium studies show that in the young, owing to growth and remodelling, a greater proportion of the skeleton will be active at any one time, and to that extent show greater unevenness of distribution. Regions which were actively growing at the time of administration show 'hot spots' in autoradiographs, which may contain half of the activity in a volume of about 1% of the bone volume, the rest of the activity being uniformly dispersed (Rowland, 1963). With passage of time, burial of the hot spots will result in the bone dose accumulated on the bone surfaces not being many times greater than the diffuse dose (Marshall, 1962; Rowland, 1963). For continuous uptake the distribution is more uniform. Radon and daughters may irradiate the epithelial surfaces in the paranasal sinuses.

The integrated dose from areas of low initial uptake where little remodelling takes place may become more significant in course of time than the dose from areas of higher initial uptake which are subsequently resorbed or buried (Vaughan and Williamson, 1967). Experience with radium patients indicates that remodelling plays an insignificant part in the distribution of radium in the adult skeleton. Radium and strontium are both lost from the adult skeleton largely through exchange with calcium (Lloyd, 1965; Kshirsagar *et al.*, 1966).

^{90}Sr (+ ^{90}Y) can be regarded as evenly distributed in adult bone. Because the average beta particle range is large compared with the dimensions of the Haversian system or trabecular cavities, hot spots would not produce much non-uniformity of dose to endosteal osteogenic cells or to bone marrow (Owen and Vaughan, 1959).

11(3).3.2 Plutonium and Thorium

It is probable that little inhaled plutonium reaches the bloodstream and therefore the skeleton, but cuts and abrasions may result in absorption from the site of injury by binding to transferrin, especially if the plutonium is in the monomeric form (Taylor, 1966). Plutonium concentrates mainly on endosteal surfaces adjacent to marrow cavities (Jee, 1964). It is greatest on resorbing or resting areas. Initially, there is no diffuse distribution throughout the bone, though this occurs later in new bone owing to uptake from blood. Initially, after injection of plutonium nitrate, there are large aggregates throughout the marrow (which are probably in

macrophages). Some months later the aggregates are of medium size and the total quantity in the marrow is much reduced. The large concentration in macrophages and endosteal surfaces (at all times after injection) irradiates both osteogenic cells and the bone marrow (Vaughan *et al.*, 1967). Plutonium is lost from bone surfaces by osteoclastic resorption. For continuous administration the marrow concentration relative to bone may be higher.

If ^{228}Ra (which has a large number of short-lived daughters) is injected as citrate, it concentrates on endosteal surfaces like plutonium. ^{228}Ra in the contrast medium Thorotrast is taken from the blood by cells of the reticuloendothelial system and large aggregates are retained in the liver and spleen (Marinelli, 1964).

11(3).3.3 Americium and the Trivalent Transuranics

^{241}Am (III) is taken up on all bone surfaces, particularly those that are resorbing or quiescent, but is not particularly concentrated on endosteal surfaces (Williamson and Vaughan, 1964). It does not concentrate in the bone marrow after injection as the nitrate. The other transuranics probably behave similarly to americium.

11(3).3.4 Phosphorus and Carbon

Like the alkaline earths, phosphorus becomes incorporated in the bone mineral with a similar pattern of distribution. It is also incorporated in the nucleoprotein fraction of all cells (Marshak, 1949), particularly those of the lymph nodes, spleen and bone marrow. The pattern of ^{32}P distribution in bone is affected by growth and remodelling in the same way as that of the alkaline earths, but its short half-life reduces the importance of remodelling.

^{14}C is present throughout the body including the bone mineral apatite and in the nucleoprotein fraction of cells.

11(3).4 BIOLOGICAL DATA ON TUMOUR FORMATION AND DEGENERATIVE CHANGES

A review of the biological data suggests the following conclusions:

(a) Experience with radium patients shows that both osteosarcoma

and carcinoma, but not leukaemia, are a hazard from bone-seeking alpha emitters.

(b) Alpha emitters, like ^{239}Pu, that concentrate on endosteal surfaces are more carcinogenic than those, such as radium, that are dispersed throughout bone.

(c) Experimental osteosarcomas the origin of which can be histologically determined arise on endosteal surfaces.

(d) With continuous administration of a long range beta emitter like ^{90}Sr (+ ^{90}Y), there is a suggestion that leukaemia rather than osteosarcoma may be the important hazard.

(e) The retention in the marrow of ^{239}Pu, ^{228}Ra and ^{32}P, in addition to irradiating haematopoeitic cells, will add to the irradiation received by the endosteal osteogenic cells.

ICRP 11 gives a detailed review of the data, both clinical and experimental, on which these conclusions are based.

The characteristic tumour in both humans and animals following the administration of large amounts of bone-seeking radionuclides is an osteosarcoma or a squamous cell carcinoma. Leukaemia in humans from internally deposited radioisotopes is much less common. The observations of Evans and Hasterlik *et al.* on tumour incidence from radium burdens in humans have been discussed earlier. Evans found that the time of onset for sarcoma was 7-43 years (median 23 years) after first exposure and for carcinomas 19-51 years (median 39 years).

A high incidence of osteosarcoma has been reported from Germany, particularly in children, following administration of ^{224}Ra for tuberculosis (Spiess *et al.*, 1962).

Radiation-induced leukaemia in patients treated with ^{32}P or X-rays for polycythemia is equivocal, since leukaemia may be a part of the disease process in polycythemia.

ICRP 11 compares experience in humans of external irradiation with internal irradiation from bone-seekers and concludes as follows: If endosteal osteogenic cells or epithelium close to bone surfaces are selectively irradiated from internally deposited bone-seekers, osteogenic sarcoma or carcinoma will result, but if haematopoeitic tissue is equally at

risk with X-rays, gamma rays or neutrons giving whole body irradiation, leukaemia is the more likely hazard.

11(3).5 FACTORS TO BE CONSIDERED IN DETERMINING THE DOSE LIMITATION FROM BONE-SEEKING RADIONUCLIDES

At the time of preparation of ICRP 11, the maximum permissible body content of bone-seekers was set by the average 'effective energy' deposited in the skeleton compared with that deposited by 0.1 μCi (3.7 kBq) ^{226}Ra (regardless of bone structure), a quality factor of 10 for alphas and a relative damage factor (n) of 5 for all bone-seekers other than radium.

(Author's note: ICRP Publication 2 (1959) introduced the relative damage factor 'to make some allowance both for the greater relative effectiveness of some radionuclides as well as for the fact that many of them have a more heterogeneous distribution than radium'.)

If we are to advance beyond present concepts (that is, those prevalent at the time of ICRP 11) on setting dose limits for bone-seeking radionuclides, the first consideration should be to examine the doses delivered.

Endosteal osteogenic tissues and haematopoeitic marrow are the tissues at risk respectively for bone tumour and leukaemia induction. Calculation of the endosteal tissue dose and the mean marrow dose requires a knowledge of the dimensions of trabecular cavities, the linear extent of the trabecular parts and the cortical thickness of different bones of the human skeleton (Spiers, 1966).

Table 11(3).1 gives computations of dose rates at the following sites:

(a) Dose rate D_0 to a small (as compared with particle range) tissue-filled cavity in bone matrix;

(b) Dose rate D_S to endosteal cells near the surface of bone trabeculae in the marrow cavities (averaged over the region from 5 to 10 μm from the trabecular surface);

(c) Dose rate D_M in trabecular cavities averaged over the whole cavity.

ICRP 11 discusses possible new approaches to the setting of dose limits for bone-seekers. These are given in the following section.

11(3).6 SUMMARY AND CONCLUSIONS OF ICRP 11

(a) Carcinogenesis (including leukaemogenesis) is the dose-limiting factor for bone.

(b) Tissues considered at risk are:

(i) osteogenic tissue, particularly on endosteal surfaces;
(ii) haematopoeitic marrow;
(iii) epithelium on bone surfaces.

(c) Bone-seeking radionuclides may be considered, as far as distribution is concerned, in four groups:

(i) the alkaline earths distributed throughout mineral bone;
(ii) plutonium and thorium concentrated particularly on endosteal surfaces;
(iii) americium, and probably other rare earths and transuranic elements, distributed on certain bone surfaces but not particularly on endosteal surfaces;
(iv) phosphorus and carbon with a wide distribution in all cells as well as in mineral bone.

(d) Under certain circumstances plutonium and thorium have a significant marrow distribution.

(e) Osteogenic cells on endosteal surfaces can receive a high dose owing to the pattern of retention of plutonium and thorium in the marrow. Both haematopoeitic marrow and osteogenic cells are at risk.

(f) In man it appears that the tissue mainly at risk from external irradiation is the haematopoeitic marrow, whereas from radium it is the osteogenic tissue and epithelium adhering to the bone. It also seems probable that in humans the marrow will be the tissue mainly at risk from the hard beta emitters, ^{90}Sr + ^{90}Y and ^{32}P. The tissues at risk from soft beta emitters may be the same as those at risk from radium.

(g) There are differences in dose, dose rate and dose distribution in bone and bone marrow when single administration is compared with repeated administrations of bone-seeking radionuclides.

(h) It seems logical to consider a fresh approach to the calculation of dose limits for humans. Such an approach now seems feasible for beta

emitters but further work is required for alpha emitters. Nevertheless, the following suggestions are put forward:

1. Separate beta emitters from alpha emitters and divide the former into three classes:

(a) For beta emitters with range large compared with bone trabecular spaces (i.e. with average energies > 0.4 MeV), calculate the marrow dose and relate it to 5 rem (50 mSv) per year;

(b) For lower energy beta emitters (0.04 MeV to 0.4 MeV), calculate the endosteal dose and relate it to 15 rem (150 mSv) per year. In this energy range QF would be 1 and no *n* factor would be involved. (150 mSv per year was the one used by ICRP at that time for unspecified tissues and ICRP 11 felt that it seemed to be applicable to endosteum).

(c) For very soft beta emitters (< 0.04 MeV) and therefore having ranges of the same order as alpha particle ranges, calculate the endosteal dose (using an appropriate QF but no *n* factor) and relate it to the endosteal dose rate from 0.1 μCi ^{226}Ra.

2. Compare alpha emitters with ^{226}Ra on the following basis:

(a) Where it is possible to calculate the dose to endosteal tissues, compare this with the endosteal dose from 0.1 μCi ^{226}Ra. No QF is required and no *n* factor is involved.

(b) In cases, where, because of inadequate information on deposition, the endosteal dose cannot be calculated, the maximum permissible body burden is to be calculated, as also now, from the appropriate equation given in ICRP publication 2 (1959).

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Table 11(3).1: Dose rates in rad/year for uniform skeletal burden of 1 μCi in 7000 g of wet bone
(from ICRP 11)

Nuclide	Radiation	Mean E (MeV)	D_0	Endosteal dose rate		Mean marrow dose rate D_M	Ratio D_S/D_M
				Over distance 5-10 μm D_S	Over whole alpha range		
^{226}Ra series	alpha	11[a]	36	10.5[e]	5	1.5	~7[e]
	beta	0.3	0.7	-	0.1	-	
^{228}Ra	alpha	19[b]	62	18[e]	9	2.5	~7[e]
	beta	1.1	2.7	-	-	~ 0.5	-
$^{90}Sr + ^{90}Y$[c]	beta	1.1	2.7	1.13	-	0.55	2
^{45}Ca[d]	beta	0.086	0.21	0.12	-	0.034	3.5
^{14}C[d]	beta	0.054	0.13	0.07	-	0.0-10	7
	X-rays 250 kV						1.5
	Gamma rays ~1 MeV						1.0

a. Based on ICRP assumptions for ^{226}Ra retention.
b. Based on ICRP assumptions about retention of ^{224}Ra following ^{228}Th in the ^{228}Ra series (ICRP 2); the absolute dose rates, however, do not affect the D_S/D_M ratio.
c. Based on data from Engstrom *et al.* (1957) - sandwich model calculations with modifications as in Spiers (1966).
d. Calculations based on single-cavity data but averaged over distribution of marrow cavity in adult skeleton.
e. Ratio depends very much on assumptions as to location of endosteal cell.

CHAPTER 11: SECTION 4

THE BIOLOGICAL BASIS FOR DOSE LIMITATION IN THE SKIN

11(4).1 INTRODUCTION

ICRP Publication 59, 'The biological basis for dose limitation in the skin' (1991) is a report of a Task Group of Committee 1 of ICRP.

The scope of the Task Group's work was to include: (a) a review of available dose-effect data for cancer induction and deterministic effects in the skin; (b) a review of the evidence concerning which cells are at risk, to determine at what depth dose measurements should be made; and (c) a re-examination of dosimetry considerations and weighting factors for skin, with reference to the effects of 'hot particles' and ultraviolet radiation.

The information collated in ICRP 59 was used by ICRP 60 to set dose limits and the weighting factor for skin.

Despite the increase in data and the understanding of biological effects on the skin in recent years, many questions remain. For example, to estimate the risk of radiation-induced skin cancer more accurately, it must be known what the nature of the dose-response relationship is, and how dose rate, fractionation, and age at exposure influence that relationship. It is not known what risk projection model is most appropriate, but neither a simple absolute nor a constant relative risk model is likely to be correct. In addition, there is little understanding of the mechanism underlying the interaction between ionizing radiation and exposure to sunlight, which is an important factor in the risk of radiation-induced skin cancer.

11(4).2 BRIEF HISTORICAL ACCOUNT OF THE EVOLUTION OF RADIATION PROTECTION STANDARDS WITH SPECIAL REFERENCE TO SKIN

ICRP 59 starts with an elegant brief account of the evolution of radiation protection standards with special reference to skin [drawn extensively

from the accounts of Taylor (1981, 1984) and Sowby (1986)]. The material is reproduced here fairly exhaustively.

Damage to the skin was historically the first radiation effect noted after exposure to X-rays. The doses were high to the skin of the physicians and technologists in the early part of this century. The first radiation-induced solid cancer was a skin cancer, and it occurred at a remarkably short time after the first of many exposures (Frieben, 1902).

The first quantitative recommendation for radiation protection proposed by Mutscheller in 1925 was called the tolerance dose and was expressed as 1/100 of a threshold skin erythema dose per month for whole body exposure to X-rays (Taylor, 1981).

In the early days of diagnostic radiology and radiotherapy, the clinician was exposed to radiation levels that increased the risk of cancer and damage to the skin. Fluoroscopy and the handling of radium were the activities that involved considerable levels of exposure. Some of the early radium workers incurred so much damage that parts of their fingers had to be amputated.

In 1928 the newly formed International X-ray and Radium Protection Commission (the forerunner of ICRP) recommended that gloves worn during fluoroscopic examinations should have a protective value of not less than 0.5 mm lead. Radium was to be surrounded with as much protective materials as possible, and long forceps were to be used for handling the radium. The Commission gave no advice on dose limits to the skin in 1928 or 1931 because of the difficulties in setting reasonable limits. In 1934, the threshold erythema dose was translated into an exposure measured in roentgens by the US Advisory Committee on X-ray and Radium Protection (which later became the present-day NCRP, National Council on Radiation Protection and Measurements). The US Advisory Committee considered the tolerance dose to be 0.1 R per day measured free in air. Later in 1934 the International X-ray and Radium Protection Commission introduced a value of 0.2 R per day. These tolerance doses were changed to 0.3 rem per week in 1948 in the US and by the ICRP in 1950. Considering the relative frequency of skin lesions in those early days (Grigg, 1965), it is surprising that there were no dose limits recommended for skin and underlying tissues until the Tripartite Conferences (1945-1954). (The participants at these Conferences were from Canada, the United Kingdom and the United States.) In 1949, at a Tripartite Conference, it was recognized that exposures at a low intermittent rate over a period of years could cause skin cancer (Taylor, 1984). It was

stated that all the cancer cases had shown typical radiation-induced effects in the skin before cancer occurred, and, in most cases, many years before. The cancers were reported to be usually squamous cell carcinomas. For radiations of low penetration it was concluded that skin was the critical tissue and cancer was the risk of concern.

Even in 1950 ICRP was somewhat reluctant to define the nature of radiation-induced damage to the skin. However, it did recommend a dose limit of 1.5 R/week for the skin in contrast to the 0.3 R/week for any other tissue. The 1.5 R/week referred to the basal layer of the epidermis of the hands and forearms, which was considered to be at a depth of 70 μm or 7 mg/cm^2. NCRP recommended 0.6 rem/week to the skin, the dose limit applying to whole body irradiation, and the dose being measured in the basal layer of the epidermis. The fact that the limit for the skin was set at twice that for other tissues was justified on the basis that skin cancer was curable and leukaemia was not. However, the limit for the skin was intended to prevent additional skin cancer. ICRP at this time introduced a limit of 1.5 rem/week for radiations with very low penetrating power, in addition to the 0.6 rem/week for more penetrating radiations. The significant area was considered by ICRP to be of the order of 1 cm^2.

In 1958 ICRP introduced some flexibility, and instead of 0.6 rem/week to the skin, the limits were set at 8 rem in 13 weeks for skin and 30 rem in a year to the whole body. For partial body exposures such as the hands, feet, forearms and ankles, the former limit of 1.5 R/week for radiation of low penetrating power was changed to 20 rem/quarter and 75 rem/year.

These recommendations did not take into account the size of the area irradiated. ICRP publication 6 (1964) considered that an area of 1 cm^2 was too small when considering the dose from contamination with radioactive material, and an area of 30 cm^2 was substituted. But the 1 cm^2 was retained for averaging the dose to the skin from other external sources. ICRP publication 9 (1966) considered the problems of such averaging.

ICRP publication 14 (1969) concluded that if the criterion for the dose limit for the skin was cancer, the limit could be raised to 100 rem/year or even 500 rem (a dose that is consistent with the risk estimates and weighting factors adopted 9 years later). If the aim was to prevent the occurrence of radiodermatitis, the limit should be lower than 100 rem. Concern was expressed that the dose limit of 75 rem/year to the extremities could result in a life-time dose of over 3000 rem and some risk of skin cancer. It was suggested that the ICRP whole body limit for skin of 30 rem should

apply to the extremities. ICRP 14 expressed concern about the use of a single depth for the basal layer, which was considered the site of the cells at greatest risk, and recommended that the basal layer should be considered to be at a depth ranging from 50 to 100 μm.

In ICRP publication 26 (1977), the basal layer was still considered the tissue most at risk, and 70 μm was considered a reasonable average for the depth of that layer. It is not known precisely what cells in the epidermis are at risk for basal and squamous cell carcinoma, but the basal layer of the epidermis is the likely site of the origin of at least some of the cancers but not all the non-cancer effects. However, the deterministic effects on the skin were considered to be the major concern, and the severity at which these were considered unacceptable was based on criteria related to cosmetic aspects.

It was thought that an annual dose equivalent limit of 0.5 Sv would prevent these deterministic effects. It was recommended that the approach of averaging the dose over a restricted area should be avoided. In the case of contamination, the dose could be averaged over areas of about 100 cm^2 and the dose limit applied to the average dose equivalent over the area.

In 1978, ICRP considered the possibility of fatal cancer induced by ionizing radiation while maintaining that deterministic effects be the basis of the dose limits. The risk of mortality from radiation-induced skin cancer was estimated to be 10^{-4} Sv^{-1} (mean dose over the skin). An annual dose limit of 5 Sv and a weighting factor of about 0.01 would be consistent with the above risk estimate. ICRP publication 41 (1984) on non-stochastic effects of ionizing radiation included reference to studies that suggest that serious skin damage will occur only with accumulated doses of 30 Gy when the exposure is protracted. Minor changes in the skin can be detected with special techniques at doses below 30 Gy of low LET radiation, but there is no evidence that these changes are of clinical consequence.

Kocher and Eckerman (1988) have drawn attention to what they consider inconsistencies in the manner in which ICRP has considered skin in relation to the effective dose and dose limits based upon it. They urged that the dose to the skin should be considered routinely for inclusion in the effective dose in the context of protection of individuals and groups. They noted that 'even with a weighting factor of only 0.01 the dose to the skin could be a significant contributor to the effective dose including skin for practical exposure conditions.' They pointed out that in the case of

many exposures, the risk to the skin can be ignored, but, for exposure in a uniformly contaminated cloud that might occur with ^{85}Kr, the dose to the skin could contribute 60% of the stochastic risk if included in the effective dose with a W_T of 0.01.

Even today the interest is in getting answers to questions like: Over what area should doses be averaged? What are the cells at risk? Are stochastic or deterministic effects dose limiting?

Since 1977 there has been a considerable addition of data for both stochastic and deterministic effects in the skin and advances in the understanding of the underlying mechanisms. This has improved the basis for recommendations of dose limits for the skin.

11(4).3 THE STRUCTURE OF THE SKIN

11(4).3.1 General

The skin is one of the major organs of the body. In the 70 kg adult it has a weight of around 2.1 kg and surface area of about 2 m^2. It has a highly complex structure designed to serve many vital functions: providing a physical barrier against the hazards of the environment; controlling fluid or electrolyte loss in varying climates; effecting thermoregulation (by dissipating heat via surface blood vessels or by evaporation of fluids secreted onto the surface of the skin by specialized structures); acting as insulator for retention of heat (insulation by subcutaneous fat); sensing the external environment; aiding physical and chemical communications; and playing a role in the body's immune system.

The skin is composed of a series of layers which can be broadly grouped into two structures: (i) the *epidermis*, which are the outermost layers; and (ii) the *dermis*, which is the deeper layer. The dermis is infiltrated by *skin appendages* which are specialized structures formed by an infolding of the epidermis [Fig. 11(4).1].

11(4).3.2 The Epidermis

The epidermis is composed of viable and non-viable layers. The outermost layer, the stratum corneum, is composed of many (15-20) layers of dead cells, and constitutes around 25% of the total epidermal thickness

Figure 11(4).1. Photomicrograph of human skin showing its organization into distinct layers; (1) epidermis; (2) papillary layer of the dermis; (3) reticular layer of the dermis; and (4) subcutaneous layer (from ICRP 59).

(Holbrook and Odland, 1974; Potten *et al.*, 1983). Its thickness depends upon the part of the body, being much thicker in the palms of the hands and soles of the feet. Beneath it is a thin (4-5 cell layers) transitional layer, the stratum granulosum, lying between the non-viable and viable layer of the epidermis.

The viable layers of the epidermis are composed of the stratum germinativum (a single layer of cells, called the basal layer) and the stratum spinosum (made up of a variable number of cells, which are rich in cell-to-cell contacts, the desmosomes, which bind the cells firmly together). It is the structural and proliferative organization of cells in these two layers which largely determines the response of the epidermis to radiation-induced injury. In many respects, the organization of the thick epidermis of humans has many parallels with that of the pig (Montagna and Yun, 1964; Weinstein, 1965); both differ in many respects from that of rodents which have very much thinner epidermis.

The basal layer, which is separated from the dermis by a basement membrane, is considerably undulated, and in many regions distinct rete ridges or pegs can be seen. In the viable epidermis, stem cells are restricted to the basal layers, predominantly towards the bases of rete pegs, although cell divisions do occur in suprabasal layers.

There are considerable variations in the thickness of human epidermis with respect to body site (Konishi and Yoshizawa, 1985; Whitton and Everall, 1973). On the face and trunk the median thickness is 20-40 μm. In general, on the arms and legs it is 40-60 μm, although there are some considerably thicker areas on the hands and feet. The mean values of the thickness in various parts of the body are given below (Whitton and Everall, 1973):

Face	50 ± 22 μm
Trunk	42 ± 12 μm
Arms and legs	60 ± 19 μm
Back of hands	85 ± 26 μm
Wrists	81 ± 18 μm
Fingertips	369 ± 112 μm
Sides of fingers	223 ± 93 μm
Backs of fingers	138 ± 47 μm

The degree of undulation in the basal layer increases with increasing epidermal thickness but as a rough guide the basal cells in the epidermis lie within ± 25% of the average epidermal thickness at that site.

In addition to the epidermis, basal cells are also found in association with the epithelium of the skin appendages. More than 50% of the basal cells are at a depth of > 200 μm, distributed in the shaft of the hair follicles (which lie continuous with the basal layer of the epidermis) at varying depths within the dermis (Osanov *et al.*, 1976).

11(4).3.3 The Skin and the Immune System

Information about the role of the skin in immune response has come to light in recent years. In the epidermis Langerhans cells are involved in processing antigens. Unlike other cells in the epidermis, these cells have Fc-IgG and C receptors that are characteristic of immunocompetent cells (Stingl *et al.*, 1977). Certain populations of lymphocytes appear to be attracted to the skin and are thought to play a role in the immune response. Keratinocytes secrete the 'epidermal thymocyte activating factor' (ETAF) which resembles interleukin-I (IL-I) (Luger *et al.*, 1981, 1982; Sauder *et al.*, 1982).

11(4).3.4 Melanocytes

Skin colour ranges from black through brown and yellow to white, and the inability to produce melanin results in the state of albinism. In white skin not exposed to sunlight, the presence of melanin is not evident except at the sites of moles. The colour of the skin is dependent more on the amount of melanin produced by melanocytes than on their number. With exposure to sunlight differences are revealed in the distribution of quantity of melanin produced. In some individuals, especially those of Celtic background, melanin appears in small patches or freckles. It is the gaps in pigmentation between the freckles that allow penetration of ultraviolet radiation (UVR), especially in the 280-320 nm wavelength (UV-B).

11(4).3.5 The Dermis

The dermis is largely composed of a network of collagen fibres and bundles which constitute about 75% of dermal tissue by dry weight; it is metabolically active and highly vascularized. Other components of the dermis are fibres of elastic tissue and an amorphous ground substance. Structurally, the dermis can be subdivided into two layers: the superficial papillary dermis, which is comparable in thickness to the epidermis, and the deeper and considerably thicker reticular dermis. The total thickness of the dermis in humans varies with the body site, but is usually in the range of 1.0 to 3.0 mm (Ordman and Gillman, 1966; Sejrsen, 1967;

Freedman *et al.*, 1968). The skin of the back is one of the thickest sites, having 2.5 mm thickness, and, in general, sites on the trunk are thicker than on the limbs. There is a significant linear decline in skin thickness with age in adults (Tan *et al.*, 1982). In the majority of sites the ratio of the dermal to epidermal thickness is around 10:1 (Rose *et al.*, 1977).

The papillary dermis is metabolically active and richly vascularized. Its main function is thermoregulation; about 90% of the blood flow is associated with thermoregulation (Goetz, 1949). The reticular dermis is the primary structural and mechanical component of the skin. The production of collagen is one of the main functions of the fibroblasts which are found throughout the dermis. The collagen is arranged in bundles which intersect at oblique angles to the skin surface, which gives the skin its unique mechanical properties (Montagna and Yun, 1964).

The vascular supply to the skin of humans (and the pig) is predominantly via segmental musculocutaneous arteries each of which supplies a relatively small area of skin. This is in contrast to that in the small laboratory rodents where single direct cutaneous arteries are the only blood suppliers to large areas of the dermis (Forbes, 1969; Smahel and Clodius, 1971).

The dermis is traversed by sensory nerves and by motor nerves of the autonomic nervous system. Their functions are: (a) sensory - probing the environment; (b) partial control of the skin pigmentation; (c) partial control of the proliferative activity; and (d) control of muscles.

11(4).3.6 The Skin Appendages

None of the appendages, which include the hair follicle, sebaceous glands, eccrine (sweat) glands, and apocrine glands, appears to play a major role in the radiation response of the skin. A brief account of skin appendages may be found in Chapter 17, Section 2.

11(4).4 RADIATION EFFECTS ON SKIN

The terminology used to describe the responses of skin to radiation and the time of onset of the responses are listed below (Hopewell, 1990);

(a) Dry desquamation (3-6 weeks): an atypical keratinization of the skin due to the reduction in the number of clonogenic cells within the basal layer of the epidermis;

(b) Moist desquamation (4-6 weeks): the loss of epidermis due to the sterilization of a high proportion of clonogenic cells within the basal layer of the epidermis;

(c) Secondary ulceration (> 6 weeks): secondary damage to the dermis as a consequence of dehydration and infection when moist desquamation is severe and protracted due to the reproductive sterilization of the vast majority of clonogenic cells in the irradiated area;

(d) Dermal necrosis (> 10 weeks): necrosis of dermal tissues as a consequence of vascular insufficiency;

(e) Dermal atrophy (> 26 weeks): a thinning of the dermal tissues associated with the contraction of the previously irradiated area;

(f) Telangiectasia (> 52 weeks): an atypical dilatation of the superficial dermal capillaries;

Special Cases:

(g) Acute ulceration (< 14 days): an early loss of the epidermis, and, to a varying degree, deeper dermal tissue, that results from the death of fibroblasts and endothelial cells in interphase;

(h) Acute epidermal necrosis (< 10 days): interphase death of post-mitotic keratinocytes in the upper viable layers of the epidermis. This type of lesion may occur with high dose, low energy beta radiation.

11(4).4.1 Pathophysiology of Radiation-induced Skin Changes

Following radiation exposure, the skin shows several distinct phases of damage, their frequency and severity depending on the conditions of exposure. Four principal phases of damage have been recognized:

11(4).4.1.1 Early Erythematous Reaction

An early transient erythema is seen in man within a few hours of irradiation of large fields with acute doses of > 2 Gy of X-rays (such as are normally involved in radiotherapy). It usually subsides after 24-48 hours. This response in the dermis is believed to be related to an early phase of inflammation in which the increased permeability of the capillaries plays a major role (Jolles and Harrison, 1965).

11(4).4.1.2 Main Erythematous Reaction

This response reflects a varying severity of loss of the basal cells from the epidermis and matrix cells from the hair follicles. There is a reddening of the skin (hyperaemia) representing a secondary inflammatory reaction to the death of basal cells and the subsequent development of epidermal hypoplasia.

The rate of the development of the early skin reaction is independent of the total dose but may vary with protraction of the exposure. After a single dose of a conventional fractionation schedule the peak reaction occurs between 3 and 6 weeks and is related to the natural turnover rate of the basal cell layer. The severity and the rate of recovery of the early reactions are dose-related. Dry desquamation occurs at moderate doses; this represents an atypical thickening of the stratum corneum. Moist desquamation (epithelial denudation) occurs at higher doses. Hair loss (epilation) may also result after 3-6 weeks.

Following irradiation with doses around the threshold for moist desquamation, repopulation of the basal layer of the epidermis is predominantly by the proliferation of surviving clonogenic cells within the irradiated area (Morris and Hopewell, 1988; Osanov, 1983). There is a 2-3 fold increase in mitotic index of the cells of the basal layer, within the canals of hair follicles, by the 20th-day post-irradiation, providing evidence for the important role played by these cells in the recovery of the epidermis after irradiation (Morris and Hopewell, 1989; Osanov *et al.*, 1976). These findings have implications for radiological protection because of the presence of a significant proportion of epithelial basal cells within the canals of the hair follicles. These will be spared in exposures involving low to intermediate energy beta emitters.

In pigs irradiated to a dose corresponding to ED_{95} for moist desquamation, the maximum level of erythema was seen between 3 and 6 weeks after irradiation. Transient moist desquamation developed after 4-6 weeks, followed by a residual dry, scaly desquamation at 5-6 weeks. Moist desquamation is more prolonged after $^{90}Sr + ^{90}Y$ (E_{max} = 2.27 MeV) than with lower energy ^{170}Tm (E_{max}= 0.97 MeV), despite the significantly lower skin surface dose. The more rapid onset of repopulation after ^{170}Tm irradiation is due to the high level of basal cell survival within the hair follicle canals (Hopewell, 1986).

With severe and prolonged moist desquamation secondary ulceration can develop.

After acute ulceration, secondary ulceration, and dermal necrosis, healing occurs by invasive fibrosis, leading to scar formation.

Moist desquamation is the reaction to be prevented after acute exposure of an area of > 5 mm diameter of the skin to radiations of moderate to high energy.

The estimated threshold doses for moist desquamation increase slightly with a reduction in the area of skin irradiated with $^{90}Sr + ^{90}Y$ due to cell migration from the edges of the exposed sites (i.e. 17.5-25 Gy for 40-5 mm diameter sources respectively). Higher estimated threshold doses (35-39 Gy) and a reduced area dependence were noted for comparable ^{170}Tm sources with E_{max} of 0.97 MeV, due to the migration of cells from within the hair follicle sheaths (Hopewell *et al.*, 1986).

The influence of the energy of the beta radiation is marked. The ED_{50} doses to produce acute effects in 50% of the exposed population were found to be 30, 70, and 340 Gy measured at a depth of 16 μm for beta radiation of maximum energy 2.27 MeV ($^{90}Sr + ^{90}Y$), 0.97 MeV(^{170}Tm), and 0.225 MeV (^{147}Pm) respectively (Charles, 1990; Hopewell *et al.* 1986).

11(4).4.1.3 Late Erythema

This is associated with dermal ischaemia, and possibly necrosis, between 8 and 20 weeks post-irradiation. With increasing fractionation this late phase becomes relatively less significant compared to the main erythematous reaction and moist desquamation (Hopewell *et al.*, 1978).

In the Chernobyl accident victims, this late phase was a significant complication in a group of people exposed to high energy beta radiation. The dose at 1500 μm depth was estimated as > 20 Gy, i.e., 30% of the dose at a depth of 70 μm (Barabanova and Osanov, 1990).

The late phase of erythema varies in severity, but at its maximum it is characterized by an erythema with a distinct bluish coloration, associated with edema. The latency period for necrosis in man is around 10 weeks.

11(4).4.1.4 Late Skin Damage

Late skin damage is characterized by dermal atrophy, a thinning of dermal tissue, and an associated reduction in the linear dimensions of an irradiated area.

Telangiectasia is a well recognized late reaction in patients receiving fractionated doses in radiotherapy. The latent period for these vascular abnormalities is 1 year, but they increase in a dose-related fashion in both incidence and severity up to at least 10 years (Turesson and Notter, 1986). Based on animal and patient studies involving fractionated irradiation, these effects are seen with an increasing frequency after a single equivalent dose of around 10 Gy, i.e. 35-40 Gy given in 2 Gy fractions.

In addition, late dermal injury may become manifest as a necrotic ulcer which is slow to heal. Necrosis is usually precipitated by trauma in atrophic skin where the recovery of the vasculature to injury may be markedly impaired.

11(4).4.2 'Hot Particle' Irradiation

'Hot particles' are small highly radioactive particles, whose size varies from a few microns to a millimetre or so. They can produce very high localized skin doses. The type of reaction is very different from that for large field irradiation with X- or gamma rays. Acute ulceration can be produced whose size and depth will depend on the skin surface dose and the energy of the particles. Following the appearance of a small pale circular area with a slight bluish tinge (frequently surrounded by a halo of erythema), the full lesion develops within 2 weeks (Hopewell, 1986). This is due to interphase death of fibroblasts and endothelial cells. The estimated threshold doses (based on animal experiments) for < 2 mm particles are around 75 Gy, measured over an area of 1.1 mm^2 at a depth of 16 μm. The corresponding threshold dose, expressed as an average over an area of the order of 1 cm^2 at a depth of 100-150 μm is in the region of 1 Gy. Doses below 220 Gy (at 16 μm depth over 1.1 mm^2) result in ulcers that will heal within a week (provided that infection is avoided). Erythema over a larger area will also occur (Darley *et al.*, 1991; Hopewell, 1990). The migration of viable basal cells from lower dose areas prevents the development of moist desquamation in the case of hot particle irradiation.

In Chernobyl, beta doses above 150 Gy (at 70 μm depth) produced a severe necrotic or ulcerative skin lesion with a latent period of 5-10 days (Barabanova and Osanov, 1990).

11(4).4.3 Irradiation by Low Energy Beta Rays and Alpha Rays

In the case of irradiation with beta particles of very low energy (< 0.225 MeV), there may be about 80% reduction in the dose across the

epidermis, leading to significantly higher doses to the cells in the upper viable layer of the epidermis than to the basal layer (Hopewell, 1986). Acute epithelial necrosis may be produced as a consequence of the interphase death of post-mitotic suprabasal cells in the epidermis less than 10 days after skin doses exceeding 200 Gy. With alpha particles, the variation in dose with depth will be much more pronounced, with no appreciable dose to the basal layers.

11(4).4.4 Field-size Effects

It has been a long accepted practice in radiotherapy to reduce the total dose to the skin as the treatment area is increased. For small fields (around 6 cm x 4 cm) the 'tolerance dose' is around 20 Gy for a single dose, and increases to 60 Gy for a 5-week fractionation. The corresponding values for a large field (15 cm x 20 cm) are around 11 and 35 Gy. Small fields can tolerate moist desquamation which is associated with prompt healing, while large fields only tolerate a dose that produces dry desquamation; moist desquamation is considered unacceptable over a large area (Ellis, 1942; Paterson, 1948).

11(4).4.5 Radiation Effects on Langerhans Cells

It is known that exposure to UVR (especially UV-B, 280-320 nm) can modify a number of immunological responses (Parrish, 1983). Exposure to ionizing radiation reduces the number of Langerhans cells in a dose-dependent manner.

It is not clear whether the surface markers are altered or the cells undergo interphase death. Delayed-type hypersensitivity appears to be reduced by soft X-rays (Fry, 1990).

11(4).4.6 Radiation Effects on Melanocytes

Both melanoblasts and melanocytes are relatively radio-resistant. After irradiation, the skin may appear less pigmented and the hair become grey or white (effects usually associated with the killing of melanocytes). In contrast, increased pigmentation may occur at lower doses with both ionizing radiation and exposure to sunlight (probably due to increased melanin synthesis rather than an increase in the size of melanoblasts). In mice the threshold for depigmentation is about 30 Gy (Chase, 1949, 1951). The effect of depigmentation appears only in hairs that grow subsequently

to irradiation. The survival curve for melanoblasts has a wide shoulder and a D_0 of about 2 Gy (Potten, 1968).

11(4).5 EXPERIMENTAL RADIATION CARCINOGENESIS

Most of the human data are for the induction of epidermal tumours, basal and squamous cell carcinomas, and it is these types of tumour that are of experimental interest. Epidermal skin cancers are very rare in rats and mice that have not been exposed to UV or ionizing radiation. The difference in the type of skin tumours and their susceptibility to induction by ionizing radiation between species has limited the generalizations that can be made. In mice the evidence supports a threshold type of dose-response relationship, but the initiation of potential cancer cells may be a non-threshold response.

The experimental studies give unequivocal evidence that the cancer risk is not increased by non-uniform irradiation. In other words, 'hot particles' do not cause more skin cancers per unit average dose than are caused by uniformly distributed doses to the skin.

The effect of dose rate has not been adequately studied. The experimental results suggest that reduction in dose rate does significantly reduce the carcinogenic effect to some degree. It might be that the reduction of malignant transformation is less than might be expected because of the greater survival of potential cancer cells.

The experimental studies have demonstrated the interactions that can occur between X-rays and other agents. Repeated exposures to UVR appear to act synergistically with X-rays. The evidence supports the finding of higher skin cancer incidence in UVR-exposed skin in patients treated with X-rays.

11(4).6 RADIATION-INDUCED SKIN CANCER IN HUMANS

There is little evidence that melanomas are associated with ionizing radiation. (Several, but not all, studies suggest that there may be a weak association between radiation exposure and malignant melanoma incidence, but the evidence is not convincing; we shall therefore not consider melanoma as a radiation-induced cancer risk type.)

The great majority of non-melanoma skin cancers are epithelial in origin. The dominant aetiological factor is UVR from sunlight, and the incidence rates vary markedly among populations.

11(4).6.1 Variations in Susceptibility to Non-melanotic Skin Cancer

It is worth noting that there is considerable under-reporting of skin cancer.

There is about a 50-fold range in skin cancer incidence across racial and ethnic groups. Among some white populations skin cancer is by far the most common form of cancer, but it is infrequent among darkly pigmented ethnic groups because of the protection from UVR (mainly UV-B, 280-320 nm).

The overall natural incidence (in unit of 10^{-5} y^{-1}) of non-melanotic skin cancer in the US white population is around 233, while the corresponding figure for US blacks is 3.4 (Scotto *et al.*, 1983). For Asians the figure is 3.1 (Quisenberry, 1963).

Among whites the overall ratio of basal cell carcinoma (BCC) to squamous cell carcinoma (SCC) is 4.6 (6 in females, 4 in males). For Asians it is 0.25-0.85, and for blacks < 0.1 (Scotto *et al.*, 1983; Gordon and Silverstone, 1976).

Although there is a preponderance of both BCC and SCC on the head and neck, relatively more SCCs occur on the arms and hands among whites. Metastases of BCC are rare, while the rate of metastases for SCC is more.

A substantial excess of SCC has been found following immunosuppressive treatment among transplant patients (Kinlen, 1982). There does not appear to be any literature on ionizing radiation-induced skin cancer among immunosuppressed patients.

11(4).6.2 Epidemiological Studies of Skin Cancers in Irradiated Populations

ICRP 59 gives a detailed account of the findings of various surveys. The groups studied include persons subjected to irradiation of thymus, head and neck, tonsils, scalp ringworm (blacks and whites), benign dermatoses, cervical cancers, testicular cancers, multiple fluoroscopy for TB, mastitis, and warts. The total number of persons studied in all these series together amounts to over 57,000, and the total follow-up period over 1 million person-years. (For details, reference may be made to ICRP 59.)

Mention may be made of an elevated incidence of skin cancers, primarily BCC of the face, in uranium miners who had alpha particle irradiation of the skin. The excess risk was concentrated among the workers with 10+ years of mining experience, for whom the doses to the basal cell layer were estimated to be 10-20 Gy or more (Sevcova *et al.*, 1978, 1984).

In addition, surveys have also been made of occupationally exposed groups like radiologists and workers at nuclear facilities, which have yielded some information. Little information on skin cancer incidence is available from the Japanese atomic bomb survivors or from the ankylosing spondylitics.

There is a large preponderance of BCC over SCC (10:1) in radiation-induced skin cancers. However, these subjects have had radiation exposure primarily in the head and neck where BCC predominates naturally. Among early radiation workers, who frequently had exposure to the hands, there were numerous reports of SCC. For UVR-induced cancers among Caucasians the ratio of BCC to SCC is 5:1.

11(4).6.3 Interaction of Ionizing Radiation and UV Radiation in Skin Cancer Induction

In the case of patients treated with PUVA (psoralen plus UV-A; UV-A comprises the 320-400 nm band) for psoriasis, it was found that a history of radiotherapy was common among those who developed skin cancer (Henseler *et al.*, 1987; Stern *et al.*, 1988). Generalization of the results of such studies to other populations and other types of UV radiation, for example, is complicated by the fact that the study subjects are not representative of the general population, having had a pre-existing skin disease and high intensity UV-A (320-400 nm) exposure to treat the disease.

There was also an excess of BCC among Caucasian whites (but no skin cancer among the blacks) treated for scalp ringworm (Harley *et al.*, 1983; Shore *et al.*, 1984). Among the whites those with a light complexion who freckled and sunburned easily showed a higher risk.

It appears that UVR is synergistic with ionizing radiation in promoting skin cancer among UVR-susceptible people such as those with light complexions. (A rigorous test of synergism has not been carried out.)

11(4).6.4 Genetic Susceptibility to Non-melanotic Skin Cancer

Patients with Nevoid basal cell syndrome (NBCS) often develop large numbers of spontaneous BCC, and it appears that such patients may be more susceptible to radiation-induced skin cancer. Patients with albinism may also be more susceptible. In both conditions, radiation-induced cancers account for only a small fraction (< 1%) of UVR-induced skin cancer.

Ataxia telangiectasia (A-T) is an autosomal recessive disorder with ataxia, immune deficiency, and a higher incidence of neoplasia. Although A-T patients manifest acute clinical hypersensitivity to X-ray therapy and their cells are hypersensitive to ionizing radiation in terms of cell killing (Morgan *et al.*, 1968), there is no clear evidence for excessive radiation-induced skin cancer in A-T patients.

There is also no clear evidence for greater susceptibility among patients suffering from retinoblastoma, xeroderma pigmentosum, psoriasis, and vitiligo.

11(4).6.5 Lethality of Radiation-induced Skin Cancers

ICRP 59 makes the following assumptions. The best estimates in the literature of lethality from SCC were 1.4%, 0.56%. and 0.7%, with the more recent estimates being lower. The estimates of lethality from BCC indicate that it is a very low rate, probably of the order of 0.01%. If we use mortality estimates of 1% for SCC and 0.01% for BCC, and a BCC:SCC ratio of 5:1 (based on the ratios of naturally occurring BCC and SCC) for radiation-induced cancers also, then the overall weighted mortality from radiation-induced skin cancer may be estimated as about 0.2%. If the BCC:SCC ratio is greater than 5:1, as most of the radiogenic cancer data suggest, then the average fatality rate would be even lower, but 0.2% has been chosen to be conservative. (The probable value could be as low as 0.05%.)

11(4).6.6 Variations in Risk with Respect to Body Site Irradiated

The bulk of the data suggest that the risk of radiogenic skin cancer per unit dose and per unit skin area is several times higher in skin areas which receive appreciable UV exposure (head, neck and arms) than in areas which do not (trunk and legs). It is therefore inappropriate to scale up

results from a study of head and neck irradiation, for instance, to the whole body by simply multiplying by the ratio of the skin areas. Separate risk estimates have therefore been derived by ICRP 59 for the UVR-exposed areas (around 3000 cm^2) and the relatively unexposed areas (around 15,000 cm^2) (Brodkin *et al.*, 1969; Laylee, 1963; Lynch and Blocker, 1977).

11(4).6.7 Choice of Skin Depth Appropriate for Skin Dosimetry

This choice is a matter of some controversy, in part because of the difference in the thickness of the skin at different body sites and in part because there is no complete agreement on the target cell for different types of carcinomas. The observation of excess BCC in uranium miners subjected to alpha particle irradiation of the skin is consistent with the prudent assumption that the basal layer of the epidermis is the site of the target cells for skin cancer induction.

Some deterministic effects occur at shallow depths but the depth at which the most serious effects arise is 300-500 μm. Nevertheless it would be conservative to use the shallow depths for monitoring specifications.

Where dermal damage is limiting, then the dose should be evaluated in the dermis. For poorly penetrating radiations, it is possible that the dose to the epidermis could be so much greater than the dermal dose that the risk of cancer exceeds the risk of deterministic effects. In the case of such exposures the dose should be monitored at a depth of 20-100 μm which is the range of depths of the majority of the skin.

Thus the target cells for the induction of cancer and the deterministic effects in skin are at different depths and, therefore, dose measurements may be required at two depths, 20-100 μm and 300-500 μm for epidermal and dermal effects respectively. Dosimeters, such as thermoluminescent devices, for measuring the dose at the greater of these depths have been available for some years. More recently, dosimeters for evaluating doses at 50-100 μm have become available.

The thickness of a routine personal skin dosimeter is of importance in providing the necessary energy response of the detector. The area of the dosimeter, for exposures other than due to 'hot particles', is not a particularly important parameter from a biological standpoint but may be relevant to questions related to the radiation sensitivity of the device. Most currently used thermoluminescent dosimeters, such as those of the sachet

type with areas of about 1 cm^2, for example, would be appropriate and could also be used to provide 'hot particle' dose assessments using the criterion of average dose.

In order to provide information of prognostic or therapeutic value in the event of an acute overexposure, information on the epidermal dose, and the shallow and deep dermal dose would be required (Osanov, 1983). Depending on the depth doses involved, this will require the use of appropriate dosimeters such as with at least 2 detector elements.

11(4).6.8 Shape of the Dose-Response Curve for Radiation-induced Skin Cancer

Although it has been traditionally thought that there was little, if any, risk of skin cancer below 10 Gy, there are now several sets of data indicating excess skin cancers following doses of a few Gy, with one suggesting a risk below 1 Gy (Shore *et al.*, 1984). The evidence does not indicate that the risk per unit dose is greater at higher doses than at lower doses. It can be assumed that the effects of multiple radiation exposures are additive.

11(4).6.9 Relative Risk vs. Absolute Risk Models

What risk model, with respect to time since irradiation and age at risk, is appropriate? The limited data available on the temporal pattern of skin cancer following irradiation suggest an RR model, and are incompatible with an AR model. The RR model may overestimate the risk (Little *et al.*, 1991). The temporal pattern of risk has been analyzed in few studies, and hence it is unclear if the RR function may diminish at older ages. The conservative assumption that the RR remains constant throughout life has therefore been chosen by ICRP 59. One limitation in comparing the models is that the available data have all been based on irradiation of juveniles; appropriate tabulations of data are not available to examine the temporal/age pattern following irradiation in adulthood.

11(4).6.10 Numerical Values for Risk of Radiation-induced Skin Cancer

A major weakness of the epidemiological data lies in the lack of control groups in several of the studies. This is particularly important for skin cancer studies, because (a) rates for the general population are generally inadequate, if available at all, and (b) special surveillance of the

irradiated population sample is likely to lead to the detection of a larger number of skin cancers at an earlier time than they would otherwise have been diagnosed, and hence the study rates may tend to be artificially inflated.

There is considerable variability in the magnitude of the risk coefficients derived from different sources. A few studies have used a 10-year minimum induction period, which may underestimate the risk. Further, follow-up times have not often been long enough.

The absolute risk estimates [10^{-8} (PY Gy)$^{-1}$ cm^{-2}], based on an analysis of the results of studies on the irradiated populations mentioned earlier, were 22 for UVR-exposed skin and 1.3 for UVR-shielded skin, which equals 6.7 x 10^{-4} (PY Gy)$^{-1}$ for total UVR-exposed skin and 1.9 for total shielded skin, or 8.7 x 10^{-4}(PY Gy)$^{-1}$ for whole body irradiation (Shore, 1990). The excess relative risk was 61% per Gy for UVR-exposed skin and 0.5% per Gy for UVR-shielded skin, or 55% on average for total body irradiation. It should be noted that these risk estimates apply principally to Caucasians, and are expected to be lower for other races.

Table 11(4).1 gives the final values arrived at by ICRP 59 for the risk of incidence as well as of mortality of radiation-induced skin cancer, estimated by both the absolute and relative risk models.

No reduction in risk has been assumed for protracted exposures even though such a reduction is likely.

The question of RBE for skin cancer by high LET radiations is essentially unanswered.

11(4).6.11 Life-time Risk Estimates

A life-table method was applied by ICRP 59 to the best available estimates of natural and radiogenic skin cancer risk. The mortality rates used were those for US white males and white females for 1983 (National Center for Health Statistics, 1987) with minor modifications. For the skin cancer rates an average of rates from the two US surveys conducted in 1971-72 and 1977-78 by Scotto *et al.* (1974, 1983) was used (again with minor modifications). A flexible life-table programme was devised based on the formulae and general approach of Cook *et al.* (1978). A simplifying assumption was made that death occurred at the age of cancer diagnosis; this would slightly overestimate the risk, because there would be no op-

portunity for intercurrent mortality from other causes to intervene between diagnosis and the potential death from skin cancer.

If a cumulative whole body dose of 1 Gy is spread out evenly over a working lifetime (ages 18-64 inclusive), then life-time excess skin cancer incidence for males under the AR model is 2.1×10^{-2} and excess skin mortality is 4.1×10^{-5}. The corresponding estimates for females are 2.6×10^{-2} and 5.1×10^{-5}. Under the RR model the life-time excess risk for males is 0.12 for skin cancer incidence and 2.3×10^{-4} for mortality. The corresponding estimates for females are 0.08 and 1.6×10^{-4}.

It may be noted that the constant relative risk model predicts greater excesses than does the constant additive risk model. This is because the natural rates of skin cancer increase sharply at older ages, so that the lifetime projection of the RR model produces larger numbers by multiplying those high natural rates. Secondly, the excess of cancers is larger for males than for females in the RR model, but relatively smaller for males under the AR model. Natural skin cancer rates at most ages are higher for males than females (Scotto *et al.*, 1983), which accounts for the result that the RR excesses are also larger. However, the greater longevity of females than that of males accounts for the higher female risks under the AR model.

The working population's average loss of life expectancy associated with the skin cancer risk was found to be small, varying from 0.1 to 9 days for the various worker exposure scenarios (1 mSv per year for ages 0-100 years; 1 Sv, evenly spread over ages 18-64 years; and 0.5 Sv per year for ages 18-64 years). As expected, the loss in life expectancy specifically for the skin cancer cases themselves was somewhat greater, ranging from 9 to 15 days. The average loss of life expectancy for an actual person who would die of skin cancer was 12-25 years, depending upon the irradiation scenario as well as upon the choice of the model (AR or RR); the RR model gives the lower value for the loss of life expectancy.

It is worth pointing out that the clinical treatment of skin cancer is relatively inexpensive and generally involves minimal trauma, and the loss of only a few working days. If treatment is given early then it should give rise to minimal scarring. However, there may be some disfigurement, and there is a tendency for recurrences and multiple primary lesions.

11(4).7 DOSE LIMITS AND THE FACTORS INFLUENCING RISK ESTIMATES

The main conclusions are summarized below:

11(4).7.1 Acute Exposure of Large Areas: Deterministic Effects

The effects on the skin of greatest concern in radiation protection are those from exposures to beta particles of various energies and low energy X- and gamma rays, because damage that may be caused by more penetrating X- or gamma rays will generally be restricted by the limitation on the effective dose. (Local irradiation by 'hot particles' poses a special problem.) There have not been any reports of deterministic effects resulting from alpha exposure.

The major acute deterministic effects are:

(i) Erythema and dry desquamation at moderate doses;

(ii) Moist desquamation which results after high dose acute exposure to moderate to high energy beta radiations or low energy X-rays;

(iii) Acute ulceration which may be seen with 'hot particle' irradiation; and

(iv) Acute epithelial necrosis after exposure to low energy beta particles.

The threshold for acute exposure of large areas is about 20 Gy. Protraction of the irradiation decreases the effect and at a dose rate of 0.4 Gy/hour no acute tissue breakdown was found with total body doses of about 100 Gy (Hopewell, 1989).

Dermal atrophy and damage to the vasculature (including telangiectasia) are the main late effects (Reinhold *et al.*, 1990). Dermal atrophy, detected as induration of the skin, a minor detriment, can occur at doses below the threshold for acute breakdown of the skin and thus could be considered the limiting effect. The threshold for fractionated exposures in man is 30-40 Gy.

In order to prevent late detrimental cosmetic effects due to chronic dermal exposure, clinical evidence thus indicates that a life-time dose to skin should be less than 30 Gy. This can be compared with the ICRP 60 occupational annual dose limit to skin of 0.5 Sv, which implies a life-time

dose limit of about 20 Sv. The dose should be evaluated in the upper dermis at a depth of 300-500 μm.

11(4).7.1.2 Stochastic Effects (Skin Cancer)

The upper limit of the risk of skin cancer is estimated to be 2×10^{-4} Sv^{-1}. The choice of a tissue weighting factor of 0.01 reflects this risk estimate and the contribution to the effective dose from exposure of the skin. The skin dose should be evaluated at a depth of 20-100 μm, averaged over the whole body. The health detriment associated with non-fatal skin cancers is considerably less than for other non-fatal cancers and the tissue weighting factor of 0.01 should provide adequately for the total detriment.

11(4).7.2 Partial Body Skin Exposures

11(4).7.2.1 Deterministic Effects

The deterministic dose limit for chronic skin exposure (0.5 Sv in a year) should be considered independent of the area exposed. In the case of skin contamination, care needs to be exercised to avoid averaging of the dose over areas which are much larger than the most highly contaminated regions, thus reducing the apparent dose which is related to the induction of potential skin damage.

11(4).7.2.2 Stochastic Effects (Skin Cancer)

The stochastic risk for the exposure of limited areas of skin is dependent upon the area of the skin involved and the exposure of the area to UVR. The whole body skin cancer risk, particularly in the case of the RR model, is dominated by the risk for those areas, such as the face, neck and arms, that are exposed to sunlight. If all of the skin that is exposed to UVR and none of the skin shielded from UVR, was exposed to ionizing radiation, the risk would be comparable to the risk for whole body exposure. The risk for areas that are some fraction of the total area exposed to UVR, such as the fingers, can be estimated from the ratio of the relevant area of the skin on the fingers to the total area exposed to UVR, about 3000 cm^2. In the case of exposure to ionizing radiation of skin shielded from UVR, the risk would be significantly less than the proportional risk based on the risk estimates for whole body exposure.

11(4).7.3 'Hot Particle' Exposures

'Hot particle' exposures are spatially non-uniform exposures from discrete radioactive particles with dimensions of less than about 1 mm. Acute ulceration is the end-point to be prevented. To prevent acute transient ulceration, the average dose delivered within a few hours over an area of 1 cm^2, measured between the depths of 100 μm and 150 μm, should be restricted to 1 Sv (Charles, 1990), and to about 5 Sv or 10^{10} beta particles to prevent acute deep ulceration (NCRP, 1989).

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Table 11(4).1: Absolute and relative risk of radiation-induced skin cancer probability (10^{-2} Sv^{-1}) (Working lifetime, 18-64 years of age, both sexes)

(from ICRP 59)

Model	Incidence	Mortality*
Absolute risk model	2.3	0.005
Relative risk model	9.8	0.02

* Based on 0.01% mortality for basal cell carcinomas, and 1% mortality for squamous cell carcinomas.

CHAPTER 12: SECTION 1

HEREDITARY (OR GENETIC) EFFECTS OF RADIATION

12(1).1 INTRODUCTION

Among the adverse biological effects of exposure to radiation are 'hereditary' or 'genetic' effects, namely those associated with gene mutations and chromosomal aberrations induced in the germ cells and transmitted to the progeny. ICRP publication 8, 'The evaluation of risks from radiation' (1966) gave a detailed review of the then existing knowledge of somatic and genetic risks of radiation and made numerical risk estimates; the risk values were revised and updated in ICRP 26 (1977). The material presented in this chapter is based on Annex B of ICRP 60, 'Biological effects of ionizing radiation' and the article by Sankaranarayanan (1991) on 'Genetic effects of ionizing radiation in man' appearing in a report of the Task Group of Committee 1 of ICRP.

Direct human genetic data continue to remain limited (since only Japanese studies provide the data and that only in the form of upper bounds to the estimates of risk). Data from experimental mammals (chiefly the mouse) still constitute the principal basis for the calculations. What they provide are estimates of *mutation rates*; these are converted, using certain assumptions, into estimates of probability of radiation-induced *hereditary disorders* in the human population.

Two kinds of radiation-induced genetic damage are important: gene mutations (alterations in the genes) and gross chromosomal aberrations (alterations in the number or structure of chromosomes). A mutation is *dominant* when its effect is manifest in the first generation progeny (and inheritance of the mutation from one of the two parents will suffice) and *recessive* when its effect is not so manifest. For the effect of recessive mutations to be expressed, the same gene mutation needs to be inherited from both the parents. *X-linked mutations* are those that are in genes located in the X-chromosome and *autosomal mutations* are those in genes located on any other chromosome. Structural chromosomal aberrations include deletions and duplications of parts of chromosomes and exchanges

of segments between different chromosomes (i.e. 'translocations'). Numerical chromosomal aberrations include loss and gain of whole chromosomes. A small proportion of chromosomal aberrations results in congenital abnormalities.

12(1).2 METHODS FOR ESTIMATION OF RADIATION-INDUCED HEREDITARY DISORDERS

Two methods are used, viz. the 'doubling dose method' and the 'direct method', which are comparable respectively to the 'relative risk' and 'absolute risk' methods discussed earlier for cancer risk projections.

The doubling dose is the dose necessary to produce as many mutations as those that occur naturally in a generation, and is obtained by dividing the spontaneous mutation rate by the rate of radiation-induced mutations. If the average spontaneous rate of a set of representative loci is m_1 per locus and the average rate of induction in the same set of loci is m_2 per locus per unit dose, the doubling dose $c = m_1/m_2$. The reciprocal of the doubling dose is the 'relative mutation risk' (RMR) per unit dose.

With the doubling dose method, the probability of excess cases of radiation-induced hereditary disorders is estimated relative to the prevalence of naturally occurring disorders in the population. For a population continuously irradiated at low doses, this probability at equilibrium (see below) per unit dose is equal to the prevalence of naturally occurring disorders divided by the doubling dose.

Risk at equilibrium per unit dose = p x RMR,

where p is the prevalence of spontaneously arising hereditary disorders.

The rationale is that under normal conditions there is an equilibrium in the population between those mutations that arise and those that are eliminated by selection in every generation. With continuous irradiation (and the continuous influx of new mutations that it entails), the population will eventually reach a new equilibrium, and the doubling dose method enables the expected additional probability at the new equilibrium to be estimated. The increased probability of disorders in the first generation progeny is then estimated from that at equilibrium by making certain assumptions.

When the population is exposed to radiation only once, there will be an increase in the proportion of mutant genes, but the number will gradu-

ally (over a number of generations) decay back to the original equilibrium value. Population genetic theory (Crow and Denniston, 1985) predicts that, numerically, the integrated probability of excess genetic damage over all future generations following a single exposure will be the same as that at equilibrium under conditions of continuous irradiation with the same dose in every generation. Thus the estimate of probability of disorders under equilibrium can be taken to represent the total probability following a single exposure.

Implicit in the use of the doubling dose method is the assumption that there is a known proportional relationship between mutation and disease. This is the case for autosomal dominant and X-linked diseases, but not for disorders of complex aetiology (i.e. congenital disorders and multifactorial disorders, see below). The 'mutational component', MC, of multifactorial disorders is the fraction of the incidence that is proportional to the mutation rate (Crow and Denniston, 1985; NAS, 1990). The autosomal dominant disorders have MC of 1 while the multifactorial disorders have MC of less than 1. The equation for the risk at equilibrium per unit dose can therefore be rewritten in a more general form as follows:

Risk at equilibrium per unit dose = p x RMR x MC.

It is also assumed that the spectrum of induced and spontaneous mutations is similar.

An increase in the mutation rate of autosomal recessive genes will not lead to a corresponding increase in the frequency of recessive disorders. This is because (a) when a recessive mutation first arises (or is induced), it is present in the heterozygous condition and its fate depends on the way selection acts, and (b) a recessive gene has to become homozygous, i.e. to have a 'partnership' with a defective allele already established in the population, to manifest the disease; this may take from many to hundreds of generations, depending on a number of factors.

Evidence for the radiation induction of numerical chromosomal anomalies resulting in livebirths either in experimental mammals or humans is insufficient and equivocal (UNSCEAR, 1986). There is definite evidence for the induction of structural anomalies (particularly non-Robertsonian reciprocal translocations).

With the direct method the absolute probability of occurrence of genetic disorders due to radiation-induced mutations in the first generation progeny is estimated from data on rates of induction of dominant (skeletal

and cataract) mutations in male mice (no data are available on female mice); likewise, the absolute risk of congenitally malformed births due to induced chromosomal aberrations is estimated from cytogenetic data on the induction of reciprocal translocations in males of the non-human primate species. These calculations do not rely upon knowledge of the natural prevalence of genetic disorders in the population. However, assumptions are needed to bridge the gap between the experimental animal data on germinal mutation changes and estimates of genetic disorders in the progeny (taking into account species differences in radiosensitivity, germ cell stages, transmission rates, dose and dose rate relationships and relative viability of the aberration types).

The germ cell stages relevant for genetic risk estimations are: (i) stem cell spermatogonia which constitute a permanent germ cell population in the testes and which continue to multiply throughout the reproductive lifespan of the individual, and (ii) the oocytes, primarily the immature ones. (Female mammals are born with a finite number of oocytes already formed during fetal development, but they are arrested at a particular stage until ovulation. The oocytes are not replenished by mitosis during adult life.)

12(1).3 CLASSIFICATION AND PREVALENCE OF NATURALLY OCCURRING GENETIC DISORDERS

Hereditary disorders are classified into 3 groups:

(a) Mendelian (i.e. those due to mutations in single genes and which follow Mendel's law of inheritance; they include autosomal dominant, autosomal recessive and X-linked disorders).

The common forms of autosomal dominants first appear in adult life (e.g. Huntington's disease, polycystic kidney disease, multiple polyposis, cerebellar ataxia, myotonic dystrophy). Other dominants associated with congenital abnormalities appear early in life (e.g. achondroplasia, bilateral aniridia, osteogenesis imperfecta). Most autosomal recessive disorders (e.g. cystic fibrosis, phenylketonuria, adrenal hyperplasia), and X-linked disorders (e.g. Duchenne muscular dystrophy, haemophilia A, fragile-X associated mental retardation, X-linked retinitis pigmentosa) have onset at birth or childhood.

(b) Chromosomal (structural or numerical) abnormalities (e.g. Down syndrome, *cri du chat* syndrome) which have onset at birth or childhood.

(c) 'Multifactorial' or 'irregularly inherited' or 'partially genetic' (resulting from the joint action of multiple genetic and environmental factors). This group includes (i) congenital abnormalities present at birth (e.g. neural tube defects, congenital heart defects, pyloric stenosis, cleft lip with or without cleft palate, undescended testes) and (ii) common disorders of adult life. These common disorders may be of varying severity. Among the serious conditions are schizophrenia, multiple sclerosis, epilepsy, acute myocardial infarction, and systemic lupus erythematosus. Moderately serious conditions include affective psychoses, Graves' disease, diabetes mellitus, gout, glaucoma, essential hypertension, asthma, peptic ulcer and rheumatoid arthritis. The least severe diseases include varicose veins of lower extremities and allergic rhinitis. Multifactorial disorders do not follow any clear-cut pattern of inheritance but they tend to 'cluster' in families.

Current estimates of birth prevalance of genetic disorders are as follows: autosomal dominant - 0.9%; X-linked - 0.1%; autosomal recessive - 0.25%; chromosomal (structural and numerical) aberrations - 0.38%; congenital abnormalities - 6.0%; other multifactorial disorders - 65% (the last refers to the total number of disorders per 100 individuals, i.e. a given individual may have more than one condition) (UNSCEAR, 1988).

One-third to half of all the known naturally occurring disorders may be deemed severe and equal in severity to fatal cancers, either because they occur early in life or because they are as detrimental as lethal diseases in adult life (e.g. Huntington's disease).

12(1).4 ICRP'S CURRENT ESTIMATES

ICRP 60 briefly reviews the UNSCEAR and BEIR estimates. These two estimates have not differed greatly and the major components have changed little over the past 15 years or so. It is pointed out that in the latest reports the bulk of the probabilities for the inducible multifactorial disorders have not been estimated. Consequently, a component of the total genetic detriment has not been included in their estimates.

Table 12(1).1 gives the estimates of these two Committees using the doubling dose method, while Table 12(1).2 gives the estimates by the direct method.

The doubling dose has been estimated as 1 Sv (for low dose, chronic, low LET radiation), based entirely on mouse data for genetically well-defined end points. This value has been accepted and used by ICRP and

UNSCEAR for a decade and a half. This value also approximates the lower 95% confidence limit for the human data from Japan (NAS, 1990). The data are consistent with minimal doubling dose estimates of 1.7-2.2 Sv for acute radiation conditions obtained during the bombings and of 3.4-4.4 Sv for chronic radiation (Neel *et al.*, 1990). The doubling dose value of 1 Sv is considered to be conservative and unlikely to underestimate the risk.

UNSCEAR (1988) arrived at a value of 120 cases of hereditary disorders per Gy of low LET radiation at equilibrium in 10^4 livebirths, i.e. 1.2×10^{-2} Sv^{-1}. For the first two generations the value is 0.3×10^{-2} Sv^{-1}.

The following further assumptions are made. The mean age at reproduction is taken to be 30 years and the average life expectancy 70 years. The genetically significant dose will then be only 30/70 or 40% of the total dose received by the population over a lifetime. (Damage sustained by germ cells of individuals beyond the reproductive period poses no genetic risks.) Thus the probability of genetically significant harm at equilibrium is $1.2 \times 10^{-2} \times 0.40$, or 0.5×10^{-2} Sv^{-1}.

Multifactorial effects are taken into account as follows: A prevalence of 70% is assumed for such disorders, a 'reasonable' value of 5% for the mutational component, and a value of 1 Sv for the doubling dose. Because some of the multifactorial diseases are less detrimental than others, an arbitrary weighting factor of 1/3 is used for the risk estimate. The weighted risk of these disorders at equilibrium (all generations, reproductive populations) is 1.2×10^{-2} Sv^{-1}, and for the total population the estimate is 40% of this value, namely, 0.5×10^{-2} Sv^{-1}.

Combining the risks for Mendelian, chromosomal, and multifactorial disorders, ICRP 60 obtains an overall risk coefficient of 1×10^{-2} Sv^{-1} for serious hereditary effects for the total population.

The risk to the first two generations is one-fourth of the total for all generations in the case of Mendelian and chromosomal diseases and one-tenth the total for each of the first two generations for multifactorial diseases and severe hereditary disorders.

For a working population, the reproductive fraction is (30-18)/(65-18) = 0.25. The probability *per caput* per worker is therefore 0.25 x 70/30 or 60% of that for the total population; this leads to a figure of $0.6 \times 10^{-2} Sv^{-1}$ for the total hereditary harm to the working population.

Table 12(1).3 summarizes the current ICRP risk estimates.

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Table 12(1).1: Genetic effects estimated using the doubling dose method for a doubling dose of 1 Gy and an average genetically significant dose of 0.01 Gy of low LET, low dose rate (chronic) irradiation per generation on a population of one million livebirths
(based on Sankaranarayanan, 1991)

Type of genetic disease	Natural prevalence per million livebirths	Expected increase (per million livebirths) in		
		First generation	Second generation	Equilibrium
		UNSCEAR, 1988		
Autosomal dominant and X-linked	10,000	15	13	100
Autosomal recessive, due to:	2,500			
Homozygous effects		No increase	No increase	11
Partnership effects		Negligible	Negligible	4[a]
Chrosmosomal, due to:				
Structural anamolies	400	2.4	1	4
Sub-total	~ 13,000	18	14	~ 120
Congenital abnormalities	6,000	not estimated		
Other multifactorial	650,000	not estimated		
Chromosmal, numerical	3,400	not estimated		
Heritable tumours	Unknown	not estimated		

(contd ...)

Type of genetic disease	Natural prevalence per million livebirths	Expected increase (per million livebirths) in		
		First generation	Second generation	Equilibrium
		BEIR V, 1990		
Autosomal dominant				
Clinically severe[b]	2,500	5-20[c]	not given	25
Clinically mild[b]	7,500	1-15[c]	not given	75
X-linked	400	<1	not given	5
Autosomal recessive	1,100	<1	not given	very slow
Chromosomal, due to:				
Unbalanced translocations	600	< 5	not given	very little
Trisomies	3,800	< 1	not given	< 1
Congenital abnormalities	20,000 - 30,000	< 10[e]	not given	10-100[d]
Sub-total	35,900 - 45,900	15-40		115-215
Other disorders of complex aetiology[f]				
Heart disease	600,000		not estimated	
Cancer	300,000		not estimated	
Selected others	300,000		not estimated	

Legend for Table 12(1).1

Unless otherwise stated, the equilibrium increase is first estimated as a product of prevalence, relative mutation risk and mutation component. The first generation increase is then estimated from that at equilibrium (15 or 20% of that at equilibrium for autosomal and X-linked and 10% of that at equilibrium for multifactorial conditions). The increase in the second generation is then calculated similarly as a fraction of the equilibrium value minus the first generation increase. Multifactorial disorders have been assumed to have a mutation component of 5% (UNSCEAR) or between 5 and 50% (NAS).

a. From partnership between induced mutations and those already present in the population, assuming 2.5% heterozygous disadvantage, and, on average, one harmful recessive per gamete.
b. Survival and reproduction assumed to be reduced by 20-80% (s = 0.2-0.8; clinically severe) or by 1-20% (s = 0.01-0.20; clinically mild) relative to normal.
c. First generation effect = natural prevalence x (1/100) x s; s values given in footnote (b).
d. A mutational component range of 5-35% has been assumed.
e. Based on 'worst case assumption' that the mutation component results from dominant genes.
f. Rough order of magnitude values.

Table 12(1).2: Estimates of genetic risk by the direct method for low LET, low dose rate (chronic) radiation
(from Sankaranarayanan, 1991, based on UNSCEAR, 1988)

Risk associated with	Expected frequency (per million per 0.01 Gy) of genetically abnormal children in the first generation after irradiation of	
	Males	Females
Induced mutations having dominant effects[a]	~ 10 - ~ 20	0 - ~ 9
Induced recessive mutations	0	0
Unbalanced products of reciprocal translocations[b]	~ 1 - ~ 5	0 - ~ 5

a. Includes risk from the induction of dominant mutations, as well as of deletions and balanced reciprocal translocations with dominant effects; based on data on the induction of dominant skeletal and dominant cataract mutations in male mice; the risk for irradiation of females was derived on the basis of known differences between male and female mice in response to the induction of recessive specific locus mutations for which the data are extensive.

b. Based on cytogenetic data obtained in male primates; the risk for females is derived from that for males. The risk figures pertain to the risk of congenitally malformed births.

Table 12(1).3: ICRP's current estimates of risk coefficients (10^{-2} Sv^{-1}) for serious he editary effects
(from Sankaranarayanan, 1991)

Time span	Dise ase category	For gonadal equivalent dose	
		Reproductive population	Total population
All generations	Me..delian & chromosomal	1.2	0.5
	Multifactorial*	1.2	0.5
		2.4	1.0
First two generations	Mendelian & chromosomal	0.3	0.1
	Multifactorial*	0.23	0.09
		0.53	0.19

* Includes congenital abnormalities and common diseases of adults

CHAPTER 12: SECTION 2

THE RBE FOR HIGH LET RADIATIONS WITH RESPECT TO MUTAGENESIS

12(2).1 INTRODUCTION

ICRP publication 18, 'The RBE for high-LET radiations with respect to mutagenesis' (1972) is an update of the information subsequent to the 1963 RBE Committee Report to ICRP and ICRU (RBE Committee, 1963). Although ICRP 18 is nearly twenty years old, since it has not been superseded, it was thought worthwhile to summarize its contents briefly.

ICRP 18 has attempted a wide-ranging comparative review of work on high LET radiations including information from microorganisms, fungi, higher plants and insects as well as mammals. The scope has been restricted to those studies with a direct bearing on genetic effects, especially from the point of view of their implications in radiation protection. Detailed references to the sources from which the information has been collated may be found in ICRP 18.

12(2).2 PRESENT AND POTENTIAL HUMAN EXPOSURE TO HIGH LET RADIATIONS

The main sources of importance here are internally deposited alpha emitters, cosmic radiation in supersonic and space travel (ICRP Task Group, 1966), and artificial sources from nuclear energy applications (especially power generation and radiation therapy).

The average dose to human gonads from internally deposited alpha emitters (mainly ^{210}Po and ^{222}Rn) is about 0.6 mrad/year (6 μGy/year) (UNSCEAR, 1966) or 60 μSv/year if the RBE is taken as 10. At 20 km altitude the overall dose rate from galactic cosmic radiation is 1 rem/year (10 mSv/year), out of which nearly half comes from neutrons (taking Q = 8) (Shaefer, 1968, 1969). Large solar flares may give at 20 km 1-2 rad (10-20 mGy) in the first hour; 30% of this total is from 0.1-10 MeV neutrons (Q around 8). Average solar radiation dose at this altitude is 0.045 mrad/hour (0.45 μGy/hour) or 0.15 mrem/hour (1.5 μSv/hour) (with a Q of 3.3).

(Author's note: In view of the changes in radiation weighting factors effected by ICRP 60, particularly for neutrons, the figures quoted above for the effective doses would change accordingly).

A small number of people working in reactors and in preparation of neutron sources may get 1-2 rem/year (10-20 mSv/year).

The levels of occupational and population exposure from artificial sources and supersonic travel are generally very low at present, but may increase slightly in the future.

[Author's note: Tables App 12(2).1.1 and 12(2).1.2 of Appendix 12(2).1 to this chapter reproduce data on average doses to the world population from natural and man-made sources of radiation, given in the latest (1988) UNSCEAR Report.]

12(2).3 A NOTE ON LET

The minimum LET_∞ of a singly charged particle (e.g. proton, electron) is 0.2 keV/μm in water. When these particles slow down, the LET increases rapidly near the end of the track, the value around the Bragg peak being some tens of keV/μm, but the track length in the high LET region is generally very small; around 100 μm. Experimental studies on biological effects of a single LET (and hence studies on variation of RBE with LET) are therefore difficult. There is a distribution of LETs in practice. Two types of weighting factors for LET are generally in use, viz. (i) dose average LET, L_D (the weighting factor for the different components of the LET distribution is the fractional dose due to any component); and (ii) track average LET, L_T (the weighting factor is the fraction of the total track length associated with any component). These two averages may be significantly different. For example, for hard X-rays, values of L_D and L_T are around 10 and 1 keV/μm respectively (Bruce *et al.*, 1963). L_D would be a more useful parameter than L_T

12(2).4 RESULTS OF RECENT STUDIES ON MUTAGENESIS USING HIGH LET RADIATIONS

12(2).4.1 Types of Studies

Several types of radiation have been used: thermal, intermediate and fast neutrons; charged particles (alphas, accelerated particles of various types, pions).

Studies have been carried out on various biological systems and for various biological end points. These include:

(i) Microorganisms (e.g. *E coli*, T4 phage, Paramecium);

(ii) Fungi (e.g., haploid and diploid yeast, Neurospora - a heterokaryote);

(iii) Higher plants;

(iv) Insects (e.g. Drosophila, silkworm);

(v) Mammals (mainly mouse). Most of the studies have been with the germ cells most at risk, viz. spermatogonia in males and oocytes arrested at the dictyate stage in females; and

(vi) Humans. These have been mainly on children of Japanese A-bomb survivors and *in vitro* studies in human peripheral lymphocytes.

12(2).4.2 Results

Studies on mammalian cells in tissue culture have given a broad picture of the variation in biological effects of high and low LET radiations, (Rossi, 1971). Briefly, the cell survival curves (at low doses) are sigmoid at low LET and exponential at high LET. The radiosensitivity is influenced significantly by cell type, cell cycle stage, oxygenation, and dose modifiers for low LET but not for high LET radiations.

Table 12(2).1 gives the average RBEs for different classes of high LET radiations with respect to mutagenesis in different organisms.

In all eukaryotic systems the efficiency of production of mutations in both germinal and somatic cells increases with increasing LET, particularly in animals and higher plants. This is also the case for chromosomal structural changes observed at the first post-radiation mitosis. RBE for point mutations seems to be considerably less than for structural deletions in chromosomes. But for single base change mutations in bacteria, the effectiveness steadily falls with increasing LET. The absolute mutation rates in eukaryotic systems are much larger (10-100 times in fungi, and up to 10,000 times in germ cells of insects and mice).

RBE values vary greatly in different groups and at different LETs. Highest values (up to 115) are found in higher plants. Values for insects and mammals are in good agreement with the Quality Factors. RBEs for

gene mutations and for gross structural changes in animals show no marked changes.

It has been clearly demonstrated that for all the experimental systems studied, the RBEs generally show the same pattern of variation with LET, increasing with LET up to about 175 keV/μm but seeming to decrease for higher LETs. 'The reasonable correspondence between RBE values for insects and mice allows one to predict with some confidence that similar values would be found in humans and that, therefore, the present Q values are appropriate.'

We do not have much firm information on RBE of intermediate energy neutrons.

There is a problem with 14 MeV neutrons because the biological damage from them is caused by (a) recoil protons with LET around 20 keV/μm and (b) heavy recoil nuclei and alpha particles with LET around 200 keV/μm. The latter LET is well beyond the peak RBE which occurs around 150 keV/μm. Thus the overall RBE is less than what would be predicted for $\bar{L}_D$ of 92 keV/μm.

RBE values rise under decreasing doses and dose rates (from 6 to 20 for specific locus mutations in mouse spermatogonia). Acute X-irradiation is 3.3 times as effective as chronic irradiation for the induction of specific locus mutations in mice (Russell, 1965). Dose rate effects are more pronounced for mice than for Drosophila. Maximum RBE values will be reached at low levels of exposure. These result from a decreasing effectiveness of low LET irradiation rather than the increased effectiveness of high LET radiations.

Other factors (e.g. sex, germ cell type, oxygen tension) do not seem likely to make RBE values for man different from those in mice.

Experience in humans has not given any direct unequivocal evidence. In Japan there were no overall effects of paternal or maternal exposure on frequency of congenital malformations, stillbirths, neonatal deaths, birth weights, Down's syndrome, or anthropometric measurements in the progeny. (Neel and Schull, 1956; Schull and Neel, 1962). The earlier report of change in sex ratio of offspring has not been confirmed by later analysis (Neel, 1963).

In human peripheral lymphocytes, the general picture is similar to that found with induction of chromosomal aberrations in mouse germ cells.

12(2).5 CONCLUSIONS

ICRP's present recommendations on maximum permissible doses for genetic effects are based on results of fairly high acute exposures. Therefore the RBE results for acute exposures apply and there is no need for any change in recommended Q values. However, if the recommendations were to be based on probable genetic effects at low doses and dose rates, then much higher RBE values would apply and Q values would have to be revised.

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Table 12(2).1: Average RBEs for different classes of high-LET radiations with respect to mutagenes.. in different organisms after acute irradiation

(from ICRP 18)

		Mean relative biological effectiveness														
		Fungi		Higher plants				Insects				Mice				
		Specific-locus forward mutations		Somatic mutations			Germ-line mutations	Specific-locus mutations		Recessive lethals	Translocations	Specific-locus mutations		Translo-cations	Dominant lethals	
				Specific-locus		Chromosome aberration										
LET ∞ (keV/µm)	Quality factor (1963)	Point	Deletion	*Arabidopsis*	*Zea*	*Nigella*	Chlorophyll loci	Silkworm late gonia	*Dahlbominus* oocytes	*Drosophila* pre-meiotic	*Drosophila* post-meiotic	Spermato-gonia	Late oocytes	Spermato-gonia	Spermato-zoa	Late oocytes
<3.5	1															
3.5-7	1-2															
7-23	2-5	2	5.5	9(1.4-15)	49		15	2.5	1		1-2				2	
23-53	5-10			16	63	66	22(3-40)	4		2	4	6	4.5	4	6	3
53-175	10-20			18	90	89	29							6		
175-900	-	5.5	74	11.5												
>900	-			1.5												

APPENDIX 12(2).1

DOSES FROM RADIATION SOURCES, NATURAL AND MAN-MADE

Table Appendix 12(2).1.1 gives the annual effective doses from the various componenets of natural background radiation (UNSCEAR, 1988). Some of the contributions (like that from ingestion of ^{40}K) to the exposure from the natural background are quite constant in space and practically independent of human practices and activities. Others (particularly that from radon and thoron daughter products) strongly depend on human activities, and are therefore widely variable. It may be seen that half the natural background radiation is contributed by lung irradiation of radon and thoron daughters. External exposures typically vary around the mean by a factor of 1.5 and the internal ones by a factor of 2.5. For both types of exposure, the extreme values vary around the mean by a factor of 100.

Table Appendix 12(2).1.2 (UNSCEAR, 1988) summarizes the estimates of effective doses from natural and man-made sources. Per caput doses to the world population, typical doses to the exposed individual from each source or practice, as well as the collective dose commitments are given. The equivalent period of exposure to natural background radiation is given along with the collective dose commitment from each source or practice.

Table APP 12(2).1.1: Annual effective doses from natural sources
(from UNSCEAR, 1988)

Source	Annual effective dose, mSv		
	External	Internal	Total
Cosmic rays			
Directly ionizing component	0.30	-	0.30
Indirectly ionizing component	0.055	-	0.055
Cosmogenic radionuclides	-	0.015	0.015
Primordial radionuclides			
Potassium-40	0.15	0.18	0.33
Rubidium-87	-	0.006	0.006
Uranium-238 series	0.1		1.34
U-238 to U-234	0.005		
Th-230	0.007		
Ra-226	0.007	1.24	
Rn-222 to Po-214	1.1		
Pb-210 to Po-210	0.12		
Thorium-232 series	0.16		0.34
Th-232	0.003		
Ra-228 to Ra-224	0.013	0.18	
Rn-220 to Tl-208	0.16		
Total	0.8	1.6	2.4

Table APP 12(2).1.2: Summary of estimates of effective doses from natural and man-made sources
(from UNSCEAR, 1988)

Source or practice	Present annual individual dose (mSv)		Collective dose commitment	
	Per caput (world population)	Typical (exposed individual)	Million person-Sv	Equivalent years of background
	ANNUAL PER YEAR OF PRACTICE			
Natural background	2.4	1 - 5	11	1
Medical exposures (diagnostic)	0.4 - 1	0.1-10	2 - 5	0.2-0.5
Occupational exposures	0.002	0.5- 5	0.01	0.001
Nuclear power production	0.0002	0.001-0.01	0.001 (0.03)*	0.0001 (0.004)*
	SINGLE PER TOTAL OF PRACTICE			
All test explosions together	0.01	0.01	5 (26)*	0.5 (2.4)*
Nuclear accidents			0.6	

* The additional long-term collective dose commitments from radon and ^{14}C for nuclear power production and from ^{14}C for nuclear test explosions are given in parentheses

CHAPTER 13

EFFECTS OF RADIATION ON THE EMBRYO AND FETUS

13.1 INTRODUCTION

There is abundant information on the effects of prenatal exposure of mammals. A few population-based studies of the effects of prenatal exposure on the developing human embryo and fetus exist. Among these, the size, length of study, variability in dose, and post-fertilization age at exposure make the experiences in Hiroshima and Nagasaki the most important. These populations were exposed at a variety of developmental phases, and, therefore, presumably a variety of sensitivities.

The principal effects of irradiation on the mammalian fetus include:

(a) lethal effects on the embryo; (b) malformations and other growth and structural changes; (c) mental retardation; (d) induction of malignancies including leukaemia; and (e) hereditary effects.

ICRP publication 60 discusses all these effects briefly. ICRP publication 49, 'Developmental effects of irradiation on the brain of the embryo and fetus' (1986) gives a detailed account of the effect of radiation on the developing human brain. ICRP 49 based its estimates of risk in terms of the T65DR dosimetry. After the new DS 86 dosimetry became available the subject has been re-examined by a Task Group of Committee 1 of ICRP and discussed in a recent volume of the *Annals of the ICRP* (Schull, 1991).

13.2 LETHAL EFFECTS ON THE EMBRYO

Lethal effects can be induced in experimental animals by relatively small doses (such as 0.1 Gy) before or immediately after implantation of the embryo into the uterine wall (UNSCEAR, 1986). They may also be induced after higher doses during all stages of intra-uterine development.

Although pregnancy loss is known to occur following exposure to radiation, data on the probability of fetal death at a particular stage in human pregnancy, for different doses, are sparse. It is difficult therefore to develop risk projections.

13.3 MALFORMATIONS

Malformations may be induced which are characteristic of the period of organogenesis at the time of exposure and especially during the most active phase of cell multiplication and differentiation in the structures of concern. Growth disturbances without malformations may also occur at all stages of development, especially in the later phases of pregnancy. Dose-effect relationships for the induction of major teratological malformations in the embryo and fetus of experimental animals are usually curvilinear and become more complex in shape as the development of the relevant structure proceeds. Dose thresholds may well apply to these effects, as have been observed in animals. Malformations can also arise spontaneously (UNSCEAR, 1986).

13.4 EFFECTS ON THE BRAIN

"The brain is one of the most complex organs of the body, with an involved architecture in which different functions are localized in different structures. Differentiation of the various structures takes place at different times and for different durations. This is particularly true of the development of the neocortex, which proceeds over a long time."

"Brain function critically depends on the disposition and interconnections of structures and cells, and normal structure and function hinge on an orderly sequence of events (cell division, programmed cell death, migration, including the positioning and selective aggregation of cells of the same kind, differentiation with the acquisition of new membrane properties, and synaptic interconnection), each of which must progress correctly, in time and space."

"The neurons of the central nervous system are not self-renewing. The capacity of neuronal precursors to divide is exhausted during histogenesis and culminates in differentiated neurons which do not undergo further division." (Report of Task Group quoted by Schull).

13.5 PRENATAL DEVELOPMENT OF THE PRIMATE BRAIN AND CEREBRAL ADNEXA

The material in this section and the next section on developmental disorders of the nervous system is a brief summary of the extensive account given in ICRP 49. For details of the sources from which the material has been collated, reference may be made to ICRP 49.

Embryogenesis is the phase of prenatal development in which the establishment of the characteristic configuration of the embryonic body occurs; it is the period from the appearance of the embryonic disk to the end of the 8th week after fertilization. After this time, the embryo is called a fetus. Most of the architectural complexity of the brain evolves in the fetal period.

Ectodermal tissue destined to give rise to the CNS can be identified in the human embryo at 16 days (O'Rahilly and Müller, 1981). Subsequently, a neural plate forms, followed by a neural groove. Shortly thereafter the closure of the groove begins. The 3 major divisions of the brain (viz., prosencephalon or the forebrain; mesancephalon or the midportion of the developing brain; and the rhombencephalon or the hindbrain which includes the pons and medulla oblongata, is continuous with the spinal cord, and gives rise to the cerebellum) can be identified by 20 days before the actual presence of any portion of the neural tube, which begins to form at 22 days (Müller and O'Rahilly, 1983). The tube rapidly increases in length and growth is completed by the 5th week (O'Rahilly and Gardner, 1977). The primitive forebrain, which until now is similar to the remainder of the neural tube, soon changes as two hemispherical vesicles develop; these evolve eventually into the cerebral hemispheres (O'Rahilly and Gardner, 1977). Two functionally different populations of cells begin to emerge - the neuronal and neurological precursor cells.

There is a period of cell multiplication in the primitive brain from the 8th to the 16th week. By mid-gestation almost all neurons in the cerebral cortex have been generated. Following their last cell division, neurons migrate to their appropriate positions in two waves, the first from the 7th to the 10th week and the second (much larger) one from the 11th to the 15th week (Sidman and Rakic, 1973, 1982). In the human and other primates, no cortical neurons are generated in the cortex itself but migrate over distance of tens of times their cell diameter (Rakic, 1974). Selective neuronal cell death during this period appears to be an important programmed mechanism for the elimination of developmental errors (Cowan *et al.*, 1984).

The human brain is anatomically asymmetrical. Lateralization of cerebral functions begins early in uterine life and is associated with microscopic anatomical differences between regions.

All nonautonomic neurosensory stimuli - auditory, olfactory, tactile and visual - are processed ultimately in the cerebral cortex.

13.6 DEVELOPMENTAL DISORDERS OF THE CENTRAL NERVOUS SYSTEM

Malformations of the CNS are divided into 2 groups, viz. the organogenetic (occurring during major organogenesis) and the histogenetic (occurring during the differentiation and growth of the brain mantle). The sensitive period for histogenetic abnormalities is later and longer (months) instead of days for the organogenetic abnormalities. Among the organogenetic abnormalities are the dysraphic malformations like anencephaly which pertain to incomplete closure of the neural tube (Müller and O'Rahilly, 1984). A common histogenetic abnormality of the cerebellum is hypoplasia with deranged cortical structure. The cerebellum is the most frequent site of genetic abnormalities and can be easily altered by cytostatic agents and radiation (Altman and Anderson, 1972; Caviness and Rakic, 1978).

Abnormalities that involve the brain adnexa could arise from maldevelopment of the end organs (e.g. eyes or ears) and/or of the processing of signals transmitted from these organs to the brain.

13.7 EFFECTS OF RADIATION EXPOSURE ON THE DEVELOPING HUMAN BRAIN

Radiation could interfere with the development of the CNS in a variety of ways. There can be death at mitosis of glial and neuronal precursors, killing of post-mitotic but still immature neurons, alteration of cell surface response, death of glial cells that guide migrating neurons, impaired capacity of the neurons to connect correctly, or somatic mutations in glial or neuronal cells (Brent, 1979, 1980; Hicks and D'Amato, 1966, 1980).

There is abundant information on the biological effects caused by prenatal exposure of mammals to radiation. In general the histological structure of the brain is similar, both in composition and function, from one species to another, and so, too, is the sequence of developmental events in all mammalian species. Some experience in humans is also

available, the most important population being the Hiroshima-Nagasaki group. This will now be discussed.

Absorbed doses to the embryo or fetus are not available, but the dose to the maternal uterus is a good approximation to the former, particularly in the second half of pregnancy. But the uterus dose may slightly overestimate the absorbed dose to the embryo or fetus in the first half when more fluid surrounds the embryo or fetus, and thus the risk in the earlier months of gestation may be underestimated (Hashizume *et al.*, 1973; Kerr, 1979).

The gestational (or developmental) age is the most single important factor in determining the nature of the insult. Four categories, viz. 0-7, 8-15, 16-25 and + 26 weeks post-fertilization, have been distinguished. In the first period, the precursors of the neurons and neuroglia, the two principal types of cells that give rise to the cerebrum, have emerged and are mitotically active. In the second, a rapid increase in the number of neurons occurs; they migrate to their ultimate developmental sites and lose their capacity to divide, becoming perennial cells. In the third, differentiation *in situ* accelerates, synaptogenesis that begins about the eighth week increases, and the definitive cytoarchitecture of the brain unfolds. The fourth period is one of continued architectural and cellular differentiation and synaptogenesis of the cerebrum, with simultaneous accelerated growth and development of the cerebellum. The results of the studies are summarized in the following sub-sections.

13.7.1 Severe Mental Retardation

Severe mental retardation implies an individual unable to perform simple calculations, to make simple conversation, to care for himself, or if he is unmanageable or has been institutionalized (Wood *et al.*, 1965).

Thirty out of the 1544 individuals prenatally exposed at Hiroshima and Nagasaki terminated in a child with severe mental retardation (Otake *et al.*, 1987); 60% of such children had disproportionately small heads (circumference more than 2 standard deviations below the mean) (Miller and Mulvihill, 1976; Tabuchi *et al.*, 1967). The highest risk was seen when exposure occurred during the 8-15 week period (the period with the most rapid production of neuronal elements when nearly all the migration of the immature neurons to the cerebral cortex from the proliferating layers takes place). There was no risk for exposure prior to the 8th or after the 25th week. Whether the absence of effect in the first two

months after conception merely reflects the fact that embryos exposed at this time commonly fail to survive to an age when mental retardation would be recognized is unclear. Within the 8-15 week period, the effect can be approximated by a linear dose-response model; some 43% of fetuses exposed to 1 Gy will be mentally retarded, which is more than 50 times greater than for exposure below 0.01 Gy in the comparison group. It appears that there could be a threshold with a lower bound of 0.12-0.2 Gy (Otake *et al.*, 1990). A period of lesser vulnerability exists in the 16-25 period (10% per Gy); a threshold (0.5 Gy) seems to exist here.

(Three of the severely mentally retarded children had Down's syndrome, a fourth had Japanese B encephalitis in infancy, and a fifth had a retarded sibling. It is conceivable that in these instances the mental retardation was merely a part of the former syndrome or secondary to the infection or inherited, but not radiation-related.)

13.7.2 Small Head Size

About 10% of the individuals with small head sizes were mentally retarded (Wood *et al.*, 1965). (As noted earlier, 60% of the mentally retarded had small head sizes.) It is not clear how independent the development of small head size may be of severe mental retardation.

13.7.3 Intelligence Tests

Intelligence tests differ from one another in the importance given to verbal ability, psychomotor reactions, social comprehension, and so on. Generally, individuals scoring high in one type of test tend to obtain high scores in other tests. The distribution of test results is approximately normal, with 95% of the population falling within 2 standard deviations; individuals whose scores lie below 2 standard deviations are described as mentally retarded. In the Japanese experience, the highest IQ achieved by any of the severely mentally retarded was 64.

The findings are (Schull *et al.*, 1988): There is no effect among individuals exposed before the 7th or after the 26th week. During the 8-15 week period (and to a lesser extent during the 16-25 week period) the mean test scores, but not the variation in scores about the mean, are significantly heterogeneous among exposure categories; the distribution suggests a progressive shift downwards of the IQ curve with increasing exposure. This shift must increase the fraction of mentally retarded individuals with increasing dose and suggests that the fall in IQ and the

increase in severely mentally retarded with dose are interrelated (ICRP 60). During the 8-15 week period, the diminution in intelligence score under the linear model is 21-29 points at 1 Gy.

A mathematical analysis shows that the increase of the fraction of mentally retarded by 0.4 per Gy and an IQ shift of 30 units per Gy are compatible, that the increase in this fraction should not be expected to be linear with dose, and that at low doses this increase per unit dose would be substantially less than 0.4 per Gy. Further, the dose required to cause an IQ shift large enough to make an otherwise normal individual mentally retarded would be high, while the dose that would bring an individual with potentially low IQ over the borderline may be a few tenths of a Gy (ICRP 60).

13.7.4 School Performance (Otake *et al.*, 1988)

For exposure in the 8-15 week period, the performance score was negatively related linearly to the dose. The trend is slightly stronger when the tests were conducted in the earlier years of schooling, suggesting the possibility of some amelioration of the effect with time. There was no effect for 0-7 week or for exposure after 26 weeks. The effect for the 16-25 week exposure was similar to than seen in the 8-15 week group.

13.7.5 Convulsions

Seizures are a frequent sequel of impaired brain development. During the 8-15 week period, seizures were highest among individuals at doses above 0.1 Gy and were linearly related to the dose. This obtained for all seizures without presence of fever or other precipitating causes, and for unprovoked seizures. The risk ratio for unprovoked seizures was 4.4 for doses of 0.1-0.5 Gy and 24.9 for higher doses when the mentally retarded are included, and 4.4 and 14.5 respectively when they were excluded. It is not clear whether seizures can arise by two mechanisms, one of which causes seizures and the other mental retardation in some individuals who are predisposed to develop seizures, or whether both arise from a common brain defect which manifests itself in some instances as mental retardation and in others as seizures. The effect was absent for exposure before the 7th week or at later stages of development (Dunn *et al.*, 1988).

13.7.6 Neuromuscular Performance

Neuromuscular performance was assessed by grip strength test and fine motor coordination test (repetitive action test) (Yoshimaru *et al.*, 1989). Both these are influenced to some extent by sex and body size; these sources of variation have been taken into account in the analysis. More neurons in the motor cortex are involved in the fine motor control test than for activity requiring larger muscle masses in the grip test. Further, probably different regions of the cortex are involved in the two tests. When severe mental retardation is included, an effect on both tests is demonstrable for the 8-15 week exposure. Multiple regression analysis indicates that no effect is seen in the grip test score save that explicable in terms of a reduction in body size. When mental retardation is excluded, no significant effect on either of the test scores was evident for exposure at any gestational age.

13.7.7 Effects on Cerebellum, Mid-brain and Brain Stem

There is no evidence of radiation-related cerebellar damage without concomitant damage to the cerebrum in Japanese survivors. Overt damage to the mid-brain and brain stem has not been reported.

13.7.8 Uncertainties

There are several uncertainties in the risk estimates due to limited nature of data, errors in dosimetry, and other factors like maternal malnutrition, disease and radiation-related haematopoietic damage on the developing child.

The nature of the dose-response relation is not established; both linear and linear-quadratic models fit the data. The presence of a threshold at 16-25 weeks but its uncertainty in the 8-15 week period are not contradictory. During the 16-25 week exposure, the cortical cells are developed and differentiated, and hence less radiosensitive than the immature, undifferentiated ones of the earlier period.

Little is known on the effects of chronic or fractionated exposure. It is reasonable to suppose that they will have some effect; this is supported by animal experiments.

13.7.9 Exposure In Utero: Other Human Data

A Finnish study (Granroth, 1979) showed that there could be an association between CNS abnormalities (anencephaly, microcephaly, hydrocephaly) among newborn infants and *in utero* diagnostic X-ray exposure. However, the majority of these infants were exposed because of the clinical suspicion of either maternal pelvic or fetal anomaly; therefore the exposures were unlikely to have occurred at a time when abnormalities could be induced (Müller and O'Rahilly, 1984).

A recent study of communities near the Hanford site reported an increased frequency of neural tube defects, which the authors ascribe to possible non-radiation related factors (Sever *et al.*, 1988a, 1988b).

Magnetic resonance imaging seems a promising tool to understand mechanisms of damage. Preliminary findings suggest a failure of neurons to migrate from the proliferative zone to the functional sites or abnormal brain architecture.

13.8 CANCER INDUCTION INCLUDING LEUKAEMIA

Irradiated fetuses seem to be susceptible to childhood leukaemias and other childhood cancers which are expressed during the first decade of life. The evidence for this, which comes mainly from the exposure of mothers to diagnostic X-rays, is only marginally at variance with direct observations on the Japanese survivors. Thus at the present time it is considered wise to regard the special susceptibility as real even at very low doses. The risk of fatal childhood cancer due to prenatal exposure has been estimated to be 2.8×10^{-2} per Sv. Constancy of risk throughout pregnancy was assumed (NAS, 1990). A different estimate, based on essentially the same data, gives a much higher risk, by a factor of about 5 (Gilman *et al.*, 1989). The risk in the first trimester appears substantially higher, but this inference has been questioned (Muirhead and Kneale, 1989).

The development of excess cancers later in life following *in utero* irradiation by the A-bombs has evidently not reached completion. Recent results (Yoshimoto *et al.*, 1988) using the revised dosimetry indicate an increased incidence of cancers in later life in those irradiated *in utero*. This incidence is comparable with the values for those irradiated postnatally, but the study of neither group is complete.

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CHAPTER 14

LOW DOSE RADIATION EFFECTS

14.1 INTRODUCTION

In arriving at risk coefficients for situations of interest in radiation protection, viz. low doses and dose rates, there are uncertainties in extrapolating from high dose and high dose rate observations. It would be valuable if quantifiable information was available in human populations directly for low dose exposure. The matter requires some attention because the estimates of cancer risk implied in some of the low dose effect studies are apparently higher (by as much as two orders of magnitude) than the estimates derived from high doses.

Annex B to ICRP 60 on 'Biological effects of ionizing radiations' briefly discusses the subject. A recent review can be found in the article by Modan (1991) appearing as part of the report of a Task Group of Committee 1 of ICRP. This chapter is a summary of the contents of the material contained in the above two references.

14.2 SUMMARY OF EVIDENCE

Numerous studies of low dose exposure exist in the literature. They can be grouped into the following categories:

(i) Populations exposed to nuclear sources such as fallout, weapon tests, or in the vicinity of nuclear reactors;
(ii) Occupational exposure;
(iii) Intra-uterine diagnostic X-rays;
(iv) Medically irradiated persons;
(v) People living in high natural background areas.

14.2.1 Nuclear Sources

This group includes civilians in the Utah-Nevada area believed to have shown higher incidence of leukaemia following fallout from weapon tests, US military participants in tests undertaken in the southwestern US, and UK veterans exposed during weapons testing and subsequently exam-

ined for cancer incidence, and the childhood leukaemia clusters apparently observed around nuclear sites in UK, particularly in the vicinity of Seascale.

Modan (1991) reviews numerous studies made on the Utah-Nevada residents. The results are equivocal. A critical re-examination of the reports of various groups finding higher cancer incidence reveals several methodological deficiencies, and when the data are properly corrected no significant excesses have been substantiated.

A recent comprehensive assessment of cancer, particularly leukaemia, in US populations living near nuclear facilities has not found any excess cancer risk (Jablon *et al.*, 1990).

Darby *et al.* (1988) examined 22,000 British veterans who took part in weapon tests in Australia and the south Pacific, between 1952 and 1967, through the end of 1983, and a similar control group. There was a significant excess mortality from leukaemia, multiple myeloma (relative excess risk for leukaemia-related disorders 1.65), and accidents, but a deficit in certain other cancers. No dose-effect relationship was noted. Increased mortality was not concentrated in the groups selected *a priori* for special examination, being pre-identified as liable to be exposed to excess radiation. The study group did not differ from control in total mortality, and both groups exhibited the 'healthy soldier effect'. No excess risk was noted in a follow-up of Canadian military personnel exposed in the Pacific Ocean testing (Raman *et al.*, 1987).

The reported incidence of excess leukaemia near nuclear installations in the UK has received considerable attention (Roman *et al.*, 1987; Gardner *et al.*, 1987a,b; 1990 a,b; Beral, 1990; Abrahamson, 1990; Dunster, 1990; Cook-Mozaffari *et al.*, 1987; 1989 a,b). Roman *et al.* demonstrated a significantly increased incidence of leukaemia among children below 5 in the immediate vicinity of nuclear installations in 3 specific districts in the UK, compared to neighbouring areas and the rest of England and Wales (relative risk, RR = 1.7). Radiation doses were not specified. Gardner *et al.* presented a follow-up through mid-1986 of 1068 children born to mothers in Seascale parish during 1950-83 and those born elsewhere but attending school there. RR for leukaemia was 9.36 (5 *vs.* 0.55 expected) and for other cancers 3.76 (4 *vs* 1.06). Both total mortality (RR = 0.84) and infant mortality (RR = 0.56) were significantly lower. In comparison there was no excess leukaemia or other cancers among 1546 children attending school at Seascale but born elsewhere. Again

lack of actual dose data precluded a definitive risk assessment. A more recent comparison showed the excess leukaemia in this population to be apparently associated with paternal exposure (RR = 6.42); the fathers had been exposed to over 10 mSv in the 6 months preceding conception and to over 100 mSv in the total. This observation is confounded by maternal age (> 40 years), proximity of residence to the nuclear installations, patient's age at diagnosis, and possibility of residential exposure through contamination of paternal clothing. This correlation of childhood leukaemia with paternal exposure is yet to find a satisfactory explanation.

Cook-Mozaffari *et al.* (1987), in a comprehensive analysis of cancer incidence near nuclear installations in the UK, found that the proportion of statistically significant deviations for individual cancer sites was similar for installation areas and their controls: 7-8% for incidence and 4% for mortality. This somewhat higher incidence rate could reflect local and temporal variations in the efficiency of cancer registration. In their 1989 paper they reported that in districts near nuclear installations there were significantly increased rates of mortality from leukaemia (RR = 1.15), particularly of the lymphoid type (RR = 1.21), and from Hodgkin's disease (RR = 1.24). Surprisingly, there was also a significant increase for these two anatomical sites in locations where nuclear installations were planned but not really established. This finding casts a shadow of doubt on causal significance of the previous apparently positive findings. A possible (yet unproved) explanation (Kinlen, 1988) attributes the phenomenon to a lower herd immunity in populations migrating to the vicinity of nuclear plants; a non-nuclear new community of Glenrothes, Scotland also showed an increase in childhood leukaemia.

14.2.2 Occupational Exposure

Mancuso *et al.* (1977) reported increased cancer mortality among 25,000 employees at the US Hanford atomic plant, from which the doubling dose for cancer was interpreted to be as low as 28 mGy. Their original observations lacked systematic follow-up, adequate dosimetry, and a suitable control population. Re-analysis of the data (Hutchinson *et al.*, 1979; Gilbert and Marks, 1979; Gilbert *et al.*, 1989; Peterson *et al.*, 1990; Tolley *et al.*, 1983) suggests that the observed excess cancer can at best be valid only for multiple myeloma, and is limited to persons who had a cumulative exposure above 0.15 Gy.

Najarian and Colton (1978) reported excess of leukaemia (fivefold) and other cancers (twofold) in workers with cumulative doses under 0.1

Gy. Confounding factors were absence of dose monitoring data, reporting by next of kin, and possible exposure to chemical agents. The effect practically vanished when a more substantial study was undertaken. Rinsky *et al.* (1981), in their analysis of mortality in 7615 US naval shipyard workers who received a mean exposure of 5 mSv and a control group of 16,930 workers showed no excess of leukaemia or other cancers. On the other hand, lower mortality rate for all cancers, including leukaemia, were observed in line with the 'healthy worker effect'.

Beral *et al.* (1985, 1988), in a study of British Atomic Energy employees covering the period 1951-82, showed a significantly increased mortality rate for prostatic cancer, particularly in young employees with single dosimeter readings exceeding 10 mSv (RR = 2.23). Mortality for total neoplasms showed a significant increase of 7.6% per 10 mSv, but stemmed primarily from increased rate of prostatic cancer. It is worth noting that prostatic cancer, being rare in young persons, is rarely looked for in this age group unless the persons are routinely followed up, and the reported incidence is, therefore, highly dependent on the frequency of screening. The healthy worker effect was exhibited, and contamination by tritium and radon daughters could not be ruled out. The inexact dosimetry for exposure to inhaled radionuclides and the better chance of diagnosis among people who are in a regular follow-up framework precluded the derivation of a true risk estimate.

14.2.3 Fetal Exposures

Studies of *in utero* exposures have not yielded consistent results. Some of the data have been criticized for selection bias and faulty methodology.

Till recently, no excess cancer has been reported in Japanese children irradiated *in utero* during the atomic bomb explosions. However, Yoshimoto *et al.* (1988) recently showed that subjects exposed to the atomic bombs *in utero* had a cancer risk in adult life comparable to that observed in survivors exposed during childhood (excess RR of 0.03 per 10 mGy).

Two major surveys, viz. the Oxford childhood survey (Stewart *et al.*, 1956) and the Tri-State study in US (Bross and Natarajan, 1972) indicated an increased risk of childhood cancer from diagnostic *in utero* exposure. Retrospective cohort studies by Monson and MacMahon (1984) also demonstrated an increased cancer risk for leukaemia (RR = 1.52). Two potential sources of bias in both the Oxford and Tri-State studies centre on the chance that mothers of children who died of cancer would have a better recall of their X-ray history and that certain characteristics of either

the mother or the child would result in diagnostic X-ray examinations, and in turn correlate with childhood cancer development.

More recent assessments by Alice Stewart's group (Knox *et al.*, 1987; Gilman *et al.*, 1988) support the contention of a higher susceptibility of fetal tissue to cancer. Mole (1990) who reassessed the *in utero* studies, emphasized the parallel decline of cancer risk with decreasing pelvimetry and increased protection standards. He supports the interpretation of a causal role for fetal radiation exposure. A significant excess of X-ray examinations of the pelvis and other parts of the body among mothers of children diagnosed to have leukaemia below 2 years of age has been reported in UK (Hopton *et al.*, 1985)..

14.2.4 Therapeutically Irradiated Populations

A follow-up through 1986 of 10,834 children below age 15 years irradiated for tinea capitis in Israel between 1949 and 1960 showed a RR of 4.12 for thyroid cancer following an average thyroid dose of 0.09 Gy. An excess of female breast cancer (estimated breast dose 0.016 Gy) also appears to have developed in this cohort after the subjects reached the age at which spontaneous breast cancer becomes prevalent (Modan *et al.*, 1974, 1989).

14.2.5 Background Radiation

An epidemiologicl survey of a high background radiation area of Yangjiang, China, on the basis of one million person-years showed no increased cancer mortality (Wei *et al.*, 1990). The dose was about 2.1 mGy per year in the high background area and 0.77 mGy per year in the control area. There was a tendency for the cancer mortality in the high background area to be lower. The prevalence of hereditary diseases and congenital defects was similar in both areas, but the frequency of Down's syndrome was higher in the high background area (though within the normal range), possibly due to a difference in the maternal age.

With the possible exception of a correlation study in UK (Knox *et al.*, 1988) from which no risk estimate can be derived, no excess cancer risk has been demonstrated in high background areas. On the other hand, many studies have noted an inverse correlation between background radiation levels and cancer mortality.

Several recent studies on radon exposures suggest that a prolonged exposure to low doses of radon in domestic facilities contributes to lung cancer development (e.g. Svensson *et al.*, 1989; Biberman *et al.*, 1990). The Swedish studies by Svensson's group report a 2-fold increase in risk for lung cancer among persons who have resided for a prolonged period in houses where radon levels were above the average. Other studies have not corroborated these findings.

14.3 PROBLEMS IN THE INTERPRETATION OF THE RESULTS

Several problems and sources of bias may confound the interpretation of the results of these studies. They include:

(a) *Small sample size* (*low signal-to-background noise ratio*): Since the level of excess risk at low doses is small compared to baseline rates, exceedingly large populations (not available in practice) are needed. Studies based on small numbers are likely to yield chance associations (Pochin, 1988).

(b) *Lack of adequate controls*: Among the complicating factors are the 'healthy worker effect', referring to a better physical status of industry workers, for whom rates of incidence of diseases in the total population may not be applicable. There may also be bias from non-random selection, e.g. comparison of groups with specific diseases who undergo irradiation for benign conditions with the general population.

(c) *Extraneous effects*: It is virtually impossible to discriminate between a radiation effect and that of chemical carcinogens in the workplace or in the general environment.

(d) *Inadequate dosimetry*: Most of the low dose studies have been of the retrospective category, where dosimetry becomes very uncertain.

(e) *Confounding factors*: Examples are social strata, lifestyle, type of house construction, or consanguinity. (An example of such a confounding factor is the reporting of a higher leukaemia mortality in Aberdeen which has a haematological clinic to which leukaemia patients are referred for treatment.) (Pochin 1888).

(f) *Positive reporting*: It is likely that the literature is weighted by 'positive findings' where an excess risk is reported, while negative results are not reported.

14.4 CONCLUSIONS

A significant proportion of the reported low dose studies yields risk estimates higher, for certain sites, than those derived from high dose studies. A range of doses is rarely available to establish the strength of the association. While the conclusions of many of the studies are spurious because of methodological problems, some remain puzzling nevertheless. Some studies show significant deficits in the response in certain sites relative to the risk estimates derived from high dose data. Some even show negative correlations between cancer risk and dose in the low dose range. ICRP 60 states, in summary, that none of the findings is sufficiently strong to provide a quantitative basis for re-assessing the current risk estimates derived from high dose studies.

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CHAPTER 15

BIOLOGICAL EFFECTS OF INHALED RADIONUCLIDES

15.1 INTRODUCTION

ICRP publication 31, 'Biological effects of inhaled radionuclides' (1979) is a report of a Task Group of Committee 1 of ICRP.

The deposition and retention of radionuclides in the respiratory tract constitute a unique radiation protection problem. Of insoluble particles deposited, a large proportion may remain in the lungs for long periods. Another large portion, with average concentrations several-fold above those in lungs, may accumulate in the regional lymph nodes. In both tissues, radioactive particles may form loci of high radiation doses. High lung concentrations may cause early death by destroying functional tissue, whereas lower concentrations may cause progressive fibrosis. It is, however, more likely that the number of particles inhaled under accidental conditions will be few enough that only a small fraction of the total respiratory functional tissue will be altered. In these cases, the primary concern is the eventual development of neoplasia. Eventually, some material deposited in the respiratory tract is accumulated in other tissues, such as liver, skeleton and thyroid.

Inhalation of radionuclides leads to problems of spatial distribution of dose within tissues. The variables are considerable: specific activity of the radionuclide and its concentration in particles, kind of radiation emitted, concentration of radiation sources (single high activity particles versus many particles of low specific activity), degree of particulate aggregation, cellular interactions and movement of particles within the tissues, solubility, presence of other toxic materials, etc.

ICRP 31 has specially assessed the problem of inhaled plutonium. It has attempted to enumerate the biological responses to radionuclides deposited in the respiratory tract, identify cells and tissues at risk, derive risk coefficients for inhaled radionuclides and determine 'equal effectiveness ratio' of alpha emitters relative to beta-gamma emitters deposited in the respiratory tract.

15.2 DEPOSITION OF INHALED RADIONUCLIDES

The comprehensive Lung Model originally developed by the ICRP Task Group on Lung Dynamics (1966) was slightly modified in ICRP 19 (1972) and further revised in ICRP 30 (1979). The ICRP 30 Lung Model is discussed in Chapter 21. This model itself is under revision by ICRP; a summary of the salient features of the new proposed model is also presented in Chapter 21.

An initial rapid clearance phase occurs within the first few days after deposition in the respiratory tract. This is attributable to rapid absorption of readily soluble material into the blood and to transport of material deposited on the ciliated epithelium of the respiratory tract to the esophagus, which is swallowed and either absorbed from the GI tract or excreted in the feces. There is a slower second phase, and, in some cases, an even slower third clearance phase involving material deposited deep within the pulmonary region. In addition to mucociliary action and dissolution, there is transport of a large portion of the insoluble material in the lungs to the bronchial and tracheobronchial lymph nodes (Thomas, 1972). The oxides of the actinides are considered as class Y compounds (i.e. compounds with clearance half-lives of several months; Y stands for years; see Chapter 21).

15.3 BIOLOGICAL EFFECTS

Apart from human experience on exposure of miners to radon, we have no other human data. A number of biological effects have been observed following inhalation or intrathecal instillation in the lungs of experimental animals. These include bronchiolar and alveolar hyperplasia, metaplasia and neoplasia; lymphocytopenia; leukocytopenia; thoracic sarcomas; osteosarcomas; lymphosclerosis and lymphadenopathy of the thoracic lymph nodes; adenomatosis and various soft tissue tumours.

15.3.1 Alpha Emitters

Alpha emitters deposited in the respiratory tract are either 'insoluble' (e.g., $^{239}PuO_2$) which are not readily translocated to other tissues, or 'relatively soluble' (e.g., $^{239}Pu(NO_3)_4$) which are more readily transported to other tissues. After inhalation of $^{239}PuO_2$, the plutonium is almost entirely distributed in the lungs and the thoracic lymph nodes for a long time. In dogs, even after some years, the distribution is: lungs - 10%; thoracic lymph nodes - 40%; liver - 15%; and skeleton - 5% (Bair, 1976).

$^{238}PuO_2$ seems to be relatively soluble and more easily cleared from the lungs, probably because of its high specific activity which results in the ejection of the plutonium atom from the particle on radioactive decay (Fleischer, 1975). Accumulation in liver and thoracic lymph nodes is about the same but in skeleton it is 10 times more than for ^{239}Pu (Park *et al.*, 1978). Plutonium nitrate is more soluble and translocated into bone and liver more readily than the oxide. Oxides of other alpha emitters like polonium and the transplutonium elements are much more readily translocated than $^{239}PuO_2$.

15.3.1.1 Life-span Shortening

Several animal studies showed that for initial lung burdens of $^{239}PuO_2$below 0.01 μCi/g lung, there was no significant shortening of mean life-span.

(Author's note: In this chapter values are given only in μCi, following the original ICRP 31 report; conversions to SI units, i.e. Bq, have not been made.)

Above this dose, life-span shortening increased with dose. The dose causing a 50% reduction of mean life-span was 0.01-0.1 μCi/g lung For soluble alpha emitters the corresponding dose was 0.1-0.8 μCi/g lung.

15.3.1.2 Pathological and Clinical Response

The shortening of life-span is accompanied by pathological (and clinical) changes, including lymphocytopenia, respiratory insufficiency, pulmonary and lymph node fibrosis, and cellular metaplasia. (Stochastic effects include pulmonary and skeletal neoplasia.)

(a) *Haematological effects*: The most consistent response after inhalation of $^{239}PuO_2$ is a dose-dependent leukocytopenia and depression of myelopoiesis. At low doses (0.003-0.04 μCi/g lung) lymphocytopenia became apparent a year after exposure and persisted throughout the lifespan of the dogs (Park *et al.*, 1972). Severe lymphocytopenia and leukocytopenia were consistently observed at initial lung burdens above 0.16 μCi/g lung (which in dogs would deliver 124 Gy to lungs and 150 Gy to lymph nodes (Yuile *et al.*, 1970). Lymphocytopenia is considered a deterministic effect, and would probably be marginally detectable after a long time following pulmonary deposition of very low amounts (< 0.0005 μCi/g lung) but the magnitude and time of onset are dose-dependent.

(b) *Non-neoplastic pulmonary lesions*: Respiratory insufficiency characterized by increased respiratory rate, decreased arterial partial pressure of oxygen and increased arterial partial pressure of CO_2, and alveolar edema, due to diffuse fibrosis of the lungs, may cause death within a few months after exposure to large amounts of the material (Bair *et al.*, 1973; West and Bair, 1964). Such deaths can also occur after months to years following exposure to smaller amounts, and is the major cause of death in those groups of animals that showed a greatly reduced mean life-span. At low levels of exposure, there may be early hypoxemia (hours to days), followed by compensation for respiratory insufficiency, with an increase in circulating erythrocytes and amount of hemoglobin (Kalmykova, 1972). At high levels (> 6 μCi/g lung) pulmonary edema and alveolar flooding with proteinaceous fluids from lung capillaries can cause death by internal drowning in a week. The threshold for deterministic effects appears to be 0.01 μCi/g lung.

(c) *Tracheobronchial lymph node lesions*: Alveolar deposited particles (particularly insoluble ones) may be translocated to the tracheobronchial and mediastinal lymph nodes resulting in concentrations many times that in lungs. Hepatic lymph nodes may also show concentrations after years. The primary lesions are characterized by lymphadenitis and fibrosis with depletion of germinal centers (Dagle and Park, 1976). The phenomenon is probably deterministic, with a threshold of 0.001 μCi/g lung for insoluble compounds and a higher threshold for soluble ones.

(d) *Neoplastic pulmonary lesions*: Pulmonary neoplasia has been identified in uranium miners due to radon inhalation. In animals neoplasia has been reported with other transuranics and thorium also, after inhalation of both the soluble and insoluble forms.

With ^{210}Po, the induced cancers arose almost exclusively peripherally in contrast to benzo(α)pyrene which produced mainly epidermoid carcinoma of the trachea or major bronchi. There was a synergistic action between benzo(α)pyrene and ^{210}Po when they were administered together (Little and O'Toole, 1974).

For relatively soluble alpha emitters, initial lung burdens above 0.001 μCi/g lung led to an increasing probability of causing lung cancer in rats. The low incidences at high doses reflected the shortened life-spans due to deaths from causes other than neoplasms which prevented the full lung cancer potential from being expressed.

In the case of insoluble compounds, as with relatively soluble compounds, the lung cancer incidence increased markedly above initial lung burdens of 0.001 μCi/g lung.

Human radon data are discussed more fully in Chapter 42. Cancers were induced in animals at exposures from 900-9000 WLM. (For definition of WLM see Chapter 42.) Above 14,000 WLM cancers did not appear since animals died because of pulmonary fibrosis. At 300 WLM cancers appeared in 10% of the rats after 2 years. Half the cancers were bronchiogenic and the other half bronchiolo-alveolar. The tumour type, common in uranium miners (oat cell carcinoma), was not observed in rats. Cigarette smoking did not increase the frequency of radiation-induced cancer in dogs, but did precipitate the onset of other respiratory tract lesions (Chameaud *et al.*, 1974; Stuart *et al.*, 1977).

(e) *Extra-pulmonary neoplasia*: Inhalation of insoluble compounds of the transuranics has not shown an increase in bone neoplasia in animals. But for soluble compounds, deposition of more than 0.007 μCi/g lung of soluble plutonium could cause detectable increase in bone cancers. There can also be extra-pulmonary and extra-skeletal cancers. Although thoracic, and, to a lesser extent, hepatic lymph nodes accumulate plutonium at levels many times that in lungs, no primary neoplasias have been reported in these tissues in animals. (The situation with regard to humans is not clear. Long follow-up of thorotrast cases may provide an answer.)

(f) *Summary*: Fig. 15.1 (ICRP 31, modified from Bair, 1974) is an attempt to summarize the effects of inhaled alpha emitters in animals. The direct effects in lungs include pulmonary fibrosis and pulmonary neo-asms. Irradiation of blood as it passes through lungs may depress mphocyte levels and reduce the immunological competence, increasing usceptibility to neoplasia. Deposition of transuranics in lymph nodes and lymphatics leads to fibrosis with loss of viable lymphocytes.

The lowest dose at which significant increase of pulmonary neoplasms has been observed in animals is 0.001 μCi/g lung. (Since carcinogenesis is considered a stochastic process, there is a probability of cancer being induced at lower levels.) For inhaled soluble alpha emitters the effects include bone neoplasia (above .01 μCi/g lung).

15.3.2 Beta-Gamma Emitters

It is likely that beta-gamma emitters behave similarly to alpha emitters, with the insoluble forms being retained in the lungs and the thoracic lymph nodes while the more soluble forms are transported to other tissues.

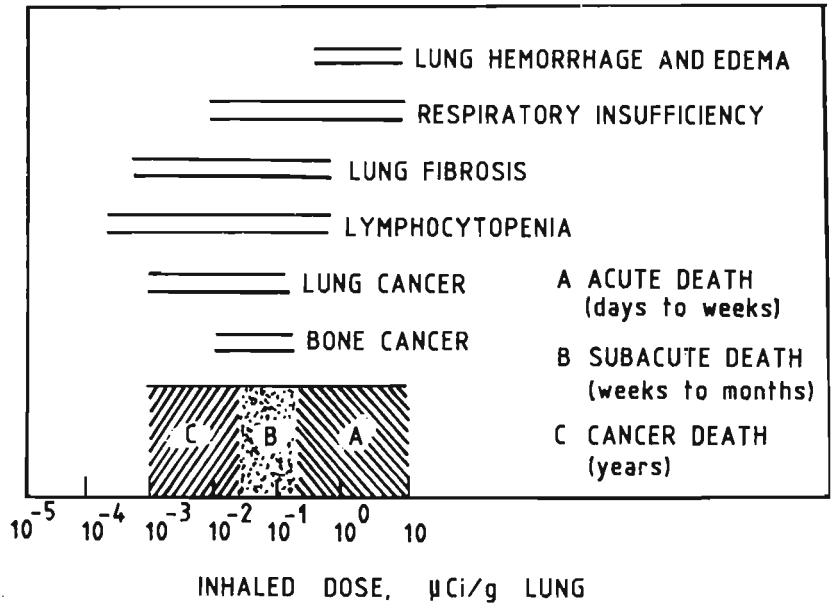

Figure 15.1. Observed biological effects of inhaled plutonium (from ICRP 31, modified from Bair, 1974).

15.3.2.1 Pathological and clinical responses

(a) *Haematological effects*: The effects are heavily influenced by the solubility in body fluids of the form inhaled and the effective half-life in lungs and other organs. In the case of inhalation of high levels of soluble chlorides of ^{90}Sr, ^{91}Y and ^{144}Ce by dogs, substantial cumulative doses were received by the skeleton (6-13 Gy) from the translocated radionuclides. This resulted in early death (12-44 days) as a result of marrow hypoplasia, panleukocytopenia, terminal hemorrhage and bacterial infection (McClellan *et al.*, 1969). Animals exposed to lower levels exhibited less severe dose-related depression in the formed elements of the blood and survived the early post-exposure period.

When the nuclides were inhaled in the insoluble form (in fused aluminosilicate vector particles) a dose-related depression of circulating lymphocytes was seen soon after inhalation. Recovery occurred rapidly

(within 6 weeks) for ^{90}Y, and partially in 200 days for ^{144}Ce, but depression of levels persisted even after 2 years with ^{90}Sr with no indication of recovery (Jones *et al.*, 1976).

(b) *Non-neoplastic pulmonary lesions*: Radiation pneumonitis and pulmonary fibrosis were observed at higher doses, leading to death in a proportion of the exposed animals. Time at death appeared to be related to dose rate. The pulmonary changes were generally reflected in substantial changes in pulmonary function during life.

(c) *Neoplastic pulmonary lesions*: Animal studies with single glass beads (~ 300 μm diameter, with activity levels of 1.1 to 59.3 μCi per bead) containing radioactive material injected into the lungs, or insertion of silicone rubber pegs impregnated with ceramic spheres containing radionuclides, showed that lung cancers (mainly squamous cell carcinomas) were induced. In all these cases there is a highly localized dose to selected portions of the lung. Intrathecal instillations would also lead to more uneven distribution of the radionuclide than after inhalation exposure. In both the above types of studies bronchiogenic carcinomas (mainly squamous cell type) were observed (Cember and Watson, 1958; Divertie *et al.*, 1967; Laskin *et al.*, 1964).

In the case of $^{144}CeCl_3$ inhalation studies, at high doses, life-span shortening and tumour incidence were observed in rats (Skupinski *et al.*, 1976). Severe pneumonitis was noticed in mice that died early from inhalation of $^{144}CeO_2$. Animals that died later showed in addition severe fibrosis and pulmonary hemorrhage. No malignant tumours were observed even at high doses (> 100 Gy) (Lundgren *et al.*, 1974). In dogs pulmonary neoplasms were a predominant cause of death after inhaling any of several radionuclides in fused aluminosilicate particles, a large proportion of these being pulmonary hemangiosarcomas with widely disseminated metastases (Boecker *et al.*, 1976).

Dogs that inhaled $^{239}PuO_2$ had mainly peripheral bronchiolo-alveolar carcinomas, and nearly half of these had also concurrent epithelial or mesenchymal tumours; very few hemangiosarcomas were observed. These differences in tumour types may be due to the differences in LET and range of alpha and beta emitters.

Initial lung burdens above 0.1 μCi/g lung led to increased lung cancers in animals.

(d) *Non-neoplastic extrapulmonary lesions*: Significant translocation from the lungs to the tracheobronchial lymph nodes eventually occurred for inhaled insoluble material, where the concentration exceeded that in lungs after 100 days. Histological changes included atrophy of the germinal centres and lymphocytic follicles, loss of lymphocytes, accumulation of macrophages in the cortical zone and in the medullary cords, and fibrosis of the medullary cords. No tumours associated with tracheobronchial lymph nodes have been observed (Hahn *et al.*, 1977a,b).

(e) *Neoplastic extrapulmonary lesions*: Most of the extrapulmonary neoplasms were in the skeleton. Neoplasms of the nasal cavities have been observed after a long time following inhalation (Benjamin *et al.*, 1976).

Skeletal and liver tumours have been noted at long times after inhalation of insoluble particles. The doses to liver and skeleton at which these tumours occurred have been lower than lung doses.

(f) *Comment*: Limited animal data pertaining to inhaled beta-gamma emitters have shown non-stochastic effects such as pneumonitis, pulmonary fibrosis and respiratory insufficiency, although thresholds could not be established. Neoplasms have also been observed.

15.4 TISSUES AT RISK

15.4.1 General Remarks on the Concept of Critical Tissue and Tissue Components

There are 40 distinctive cell types in lungs (Sorokin,1970). In defining tissue components at risk, several factors have to be considered involving both the radioactive particles (size, specific activity, type of radiation emitted, aggregation, solubility, displacement with time, excretion), and lung dynamics (stem cell characteristics, growth pattern, cell cycle phases and their relative radiosensitivity, cell migration, turnover and death rates of cells). Inhaled material may migrate and cells at risk may undergo passive transport. A microphage containing the particle may appear as a 'hot spot' in an autoradiograph; if, however, this cell migrates in the alveolar spaces, a diffuse type of distribution results. Larger particles kept in motion by the tracheobronchial mucociliary apparatus will act as mobile radiation sources tending to distribute the dose. Mucus that contains particles and accumulates near the tracheal bifurcation could give higher doses delivered to this area than to small bronchi. Then there

are problems of extrapolation from small to large animals because of the unequal number of cells at risk. There is evidence that the neoplastic risk is increased by uniformity of distribution rather than by hot spots.

Most particles deposited in the alveolar region are phagocytized by macrophages in peripheral circulation. They are differentiated cells whose stem cells in bone marrow may receive only a small dose; this will lead to replacement of damaged lung phagocytes by their precursors. Alveolar deposited matter may be partially translocated to other organs.

Cells passing through lung blood vessels, lymphatics and regional lymph nodes will be irradiated. Most blood cells do not re-enter a proliferative state and are radioresistant. Lymphocytes are radiosenisitive and can receive lethal doses. There could be internal irradiation of circulating blood and lymph due to particles in the lung and tracheobronchial lymph nodes, leading to lymphocytopenia with immunological consequences.

15.4.2 Criteria for Defining Tissues at Risk

The important hazards are induction of neoplasia, impairment of cellular defence mechanisms (particularly through lymphocyte damage), structural and functional changes (e.g., fibrosis of lungs) and life-shortening. Animals studies show that for prolonged exposure at low levels of radioactive burdens, pulmonary tumours, pulmonary fibrosis and edema, and fibrosis of tracheobronchial lymph nodes are the main changes. Higher levels of radioactivity produce life-shortening.

15.4.3 Tissues and Cell Types Apt to be Damaged

All tissue components and contents of lymph or blood vessels localized within 50 μm of alpha emitters in unit density material are of interest. The effective density of lung tissue is only 0.2 and particles can reach greater distances than that mentioned above. Almost any tissue constituent of the lungs may receive large doses. Tracheobronchial lymph nodes can get a dose several times the mean lung dose because of their higher concentration of radioactive particles. Even for beta-gamma emitters with long range, the largest part of the energy will be absorbed in the lungs and lymph nodes.

15.4.4 Nasal Passages

In dogs, large radioactive particles (> 50 μm diameter) tend to be deposited in the nasopharynx and excreted in the feces over a few days

and can give large doses to the squamous cell epithelium (Willard and Stuart, 1968). There is less damage to the upper oropharynx, nasopharynx, upper respiratory passage, or GI tract.

15.4.5 Epithelia of the Tracheobronchial and Pulmonary Regions, Pneumocytes, and Alveolar Macrophages

The cleaning function of mucociliary epithelium is more effective for inhaled $^{239}PuO_2$ with a MMD of 0.2 μm and less for 0.4 μm diameter. The half-life for clearance of smaller sized aerosols is 12-36 hours. All the plutonium initially lodged at this site is removed and excreted through the feces within 10 days (Morrow *et al.*, 1967). Non-phagocytized particles in the 1-4 μm range can deliver substantial doses to the epithelium within a few days. Damage to epithelia of the larger bronchi is relevant since they are the predominant sites of lung cancer in man. It is not known whether chronic low level inhalation exposure has the same effect as a single inhalation giving a high dose. Nor is information available on radiation effects on neuroepithelial bodies which may be responsible for the large bronchial cancers found in uranium miners.

Alveolar lining cells, especially alveolar macrophages (except type II pneumocytes) are capable of phagocytosis and hence candidates for receiving high doses. Both type II pneumocytes and bronchial epithelial cells seem to be the origin of adenocarcinomas in rat lung after plutonium inhalation (Sanders, 1972; Sanders *et al.*, 1976).

15.4.6 Blood and Lymph Vessel Constituents of the Lungs, Especially Endothelial Cells

The endothelial cells of pulmonary blood capillaries, which represent half of the cells in alveolar tissue (Weibel, 1963), are with in the range of alpha particles deposited in the alveolar wall. Irradiation may lead to progressive obliteration of the capillaries resulting in pneumosclerosis and concomitant right heart failure. Haemangiosarcomas are rare compared to carcinomas in the case of alpha radiation, but common for inhalation of insoluble beta emitters (Park *et al.*, 1972). Damage to the capillary bed and to alveolar lining cells and formation of a fibrous exudate would lead to alveolar septal fibrosis. Disruption of the basal lamina may lead to local scarring (Vracko, 1974). Arterial and venous endothelial cells as well as connective tissue components within vessel walls are all within range of alpha-emitting particles.

Blood cells passing through the pulmonary vessels, as well as endothelial cells of the pulmonary lymphatics (through which alveolar deposited material is translocated to the tracheobronchial lymph nodes) should also be considered as cells at risk.

15.4.7 Connective Tissue Constituents and Other Cell Types of Lungs

Although fibroblasts are the principal elements of the reparative scar tissue that develops following radiation-induced lesions, they also represent a normal cell population at risk. Alpha emitters entrapped in scar tissue will have reduced effect on epithelial cells and hence alpha-induced (but not the longer range beta-gamma-induced) fibrosis may restrict the number of cells apt to undergo neoplastic transformation. Mesothelial cells of the pleura do not appear to be within the range of alveolar deposited alpha emitting particles (Clarke *et al.*, 1966). No data are available on the effects of inhaled particles on lung nerve fibres.

15.4.8 Regional Lymph Nodes

Tissue constituents of the tracheobronchial and other thoracic lymph nodes concentrate inhaled material several hundred times greater than in the pulmonary regions and receive very high doses. Lymph nodes contain a pool of long-lived recirculating lymphocytes, half of them being mobile radiosensitive T cells and the other half more sessile B cells. Irradiation of the lymph nodes results in damaging both types of cells. Observed lymphocytopenia may be partly due to this type of internal irradiation of recirculating lymphocytes. The immunological hazards of inhaling insoluble radioactive material have not been determined.

Tumourogenesis has been found mainly in tissues that receive high doses. Malignant lymphomas have not been seen in animals; nor have they been seen in humans showing heavy Thorotrast burdens in lymph nodes (Mole, 1978). The role of tracheobronchial pathology as a correlate of lung tumour induction has not been elucidated.

Except for lymphoid cells and plasmoblasts, most other lymph node components are fairly radioresistant. Long-term effects on small blood vessels and connective tissue elements are well known. As long as the supportive network of reticulum cells, blood vessels and lymph sinuses of a lymph node is preserved, despite considerable accumulations of radioactive material, the lymphatic parenchyma in it can continuously be replaced by blood lymphocytes. However, excessive scarring may restrict

entry of lymphocytes. Such findings have been made in human lymph nodes with a heavy burden of Thorotrast.

15.4.9 Relationship of Cellular Kinetics to Damage Caused by Inhaled Radionuclides

Our knowledge of cytokinetics of the more than 40 cell lines in the lungs and tracheobronchial lymph nodes is insufficient for a complete evaluation of the dynamic processes following inhalation of radionuclides. It appears that the regenerative activity and the risk of carcinogenesis are to some extent related. The time parameters for cell renewal vary from cell line to cell line, and, for epithelia, from site to site.

More is known about the cellular kinetics of lymph nodes than of the lungs. Lymphocytes are produced in the thymus, in peripheral lymphoid tissues, and, at least from birth, in bone marrow. From these sites they migrate to other sites. Generation times for these cells in mammals are around 6-11 hours. Certain compartments like the thymic cortex, bone marrow, and lymph node germinal centres exhibit high renewal rates; others such as primary follicles and the recirculating lymphocyte pool contain large numbers of long-lived lymphocytes. The lymphoid system's long recovery time following short-term external irradiation results from the longevity of many lymphocytes (Cottier *et al.*, 1964). Little is known about the kinetics of connective tissue cells, nervous tissues of the lungs, and of blood and lymph vessel constituents.

15.4.10 Interaction of Radioactive Particles with Cells

Information on the subject is limited. Particles have been found over prolonged periods in type 1 cells. In rats it appears that over 50% of alveolar deposited material may be phagocytized by alveolar macrophages within a week (Sanders, 1972). After inhalation of large amounts of plutonium, microphage death was noticed even at 1 hour. Most cells, however, did not show such early effects. Microphage death is concomitant with release of enzymes that may elicit some inflammatory response which may contribute to pulmonary fibrosis.

Endothelial cells of pulmonary capillaries do not seem to phagocytize inhaled particles but may be damaged by radioactive material deposited in their vicinity. In the course of these events, structures and functions of pneumocytes might undergo changes. Lymphocytes may be damaged while

passing through lung capillaries. There may be damage to bone marrow and liver through translocation of material to these organs.

15.4.11 Tissues and Cells at Risk Based on Type of Neoplasia Produced

Uranium miners had undifferentiated small cell carcinoma after lower dose exposures and epidermoid carcinomas in the larger proximal bronchi after higher doses (Archer *et al.*, 1974; Horacek *et al.*, 1977). After inhalation of radionuclides the following types of neoplasia are predominant in animals: peripheral adenocarcinoma, epidermoid or squamous cell carcinoma, and haemangioma.

15.5 ADDITIONAL FACTORS TO BE CONSIDERED

In the evaluation of hazards from inhaled radioactive materials, it must be remembered that the respiratory system is often subject to a number of other potentially exogenous agents, whose combined or possibly synergistic influence has not yet been clearly delineated, such as smoking, inhalation of dusts in the industrial environment, urban air pollutants, and organic chemicals including home-use preparations like hair sprays.

15.6 DOSE-EFFECT RELATIONSHIPS

Animal data show that for inhalation of both soluble and insoluble alpha emitters, the peak lung tumour incidence occurs at doses of around 10 Gy. At higher doses the incidence starts decreasing because death occurs before the cancer is expressed. For beta-gamma emitters the corresponding dose for maximum lung tumour incidence could be 10-50 times greater. (While alpha emitter studies were from inhalation experiments, the beta-gamma studies were based on intrathecal intubation studies.)

ICRP 31 notes that there has been no uniformity in the experimental protocols of different investigators, nor has the dosimetry been completely satisfactory. ICRP 31 has made a detailed critical analysis of the available data.

Sigmoid curves have been traditionally used to model toxicological data. ICRP 31 has compared the results of the analysis based on the logarithmic probit model with the linear model usually used to reflect conservatism in risk estimation. Most of the dose-response models fit the data in a similar way in the region of 10-90% response but differ markedly for predictions beyond these points, especially further out in the 'tails'.

Probit models fitted to the soluble and insoluble alpha emitters resulted in different, but not statistically significant, descriptions of the incidence data. At similar doses a greater incidence of lung cancer would be projected for insoluble alpha emitters. The linear model for the beta-gamma incidence data gives unrealistically high projected incidences at low doses.

ICRP 31 calculates that to obtain incidence of 1 in a million lung cancer, doses of 0.52, 0.14, 0.40 and 11.9 mGy respectively from soluble alpha, insoluble alpha, all alpha, and beta-gamma emitting radionuclides would be necessary. Equal Effectiveness Ratios of 6 and 40 were obtained from the probit model for the alpha and beta-gamma emitters at the 10% and 40% incidence values respectively. Comparing doses that coincide with maximum incidence, Equal Effectiveness Ratios between 10 and 100 were obtained when plutonium data were compared with the beta-gamma data. The results strongly support a quality factor (radiation weighting factor in current terminology) of at least the value of 20 recommended by ICRP 26 for alpha radiation.

A summary of the extensive tables given in ICRP 31 leads to the following risk coefficients (lung cancer cases per million per Gy):

Rodents and Dogs

Soluble alpha-emitting transuranics	2,000 - 80,000
Insoluble alpha-emitting transuranics	6,000 - 160,000

The lower values are based on the probit model while the higher ones correspond to the linear model.

Humans (Linear Model) (Mays, 1976; MRC, 1975; UNSCEAR, 1972, 1977; ICRP, 1977).

External irradiation (X-, gamma rays, and neutrons)	20,000 - 50,000
Internal irradiation (Thorotrast, radon daughters, radium)	20,000 - 100,000

There are no deaths known to have been caused by lung cancer attributable to inhalation of transuranics among the many hundreds of persons who have been exposed. ICRP 31 believes that risk estimates calculated from available data are supportive of the ICRP 26 risk factor for lung cancer of 2×10^{-3} Sv^{-1}.

15.7 HOT PARTICLES

Knowledge about the behaviour of inhaled alpha emitting particles in the lungs and the interaction of alpha radiation with cells is inadequate either to support or deny the hot particle theory concerning the induction of lung cancer. Animal experiments indicate that the lung cancer risk associated with insoluble inhaled plutonium in quantities that could be distributed in hot spots may be slightly greater than for more soluble and therefore more diffusely distributed alpha emitters. Other experiments with plutonium microspheres clearly showed that diffuse radiation sources in the lungs were more likely to cause both benign and malignant tumours than highly localized sources. ICRP 31 concludes that the risk of lung cancer from inhaled radioactive particles will be greatly over-estimated if based on the hot particle concepts.

15.8 SUMMARY AND CONCLUSIONS

Besides uranium miners and other miners who were exposed to high concentrations of radon and radon decay products, there are no human populations that have shown health effects attributable to inhalation of radionuclides. Radon and its daughter products have short half-lives and short retention times in the respiratory tract. Therefore, data from uranium miners may not be totally applicable to the assessment of the biological effects of radionuclides that may have long retention times in the lungs or be translocated to other organs and tissues which are then irradiated. Thus, animal experiments provide virtually all the information of relevance.

In its review ICRP 31 notes that most abundant are the animal data describing biological responses. The greatest uncertainties are in the definition of cells at risk and in quantitative dose-response relationships. A further uncertainty arises from the extrapolation of animal data to humans.

15.8.1 Biological Response to Inhaled Radionuclides

High lung burdens of inhaled radionuclides result in profound structural and functional changes commonly referred to as progressive pulmonary fibrosis. This cause of radionuclide-induced life-shortening affects many of the more than forty distinctive cell types of the respiratory tract. However, the most prominent cells are pulmonary capillary endothelial cells, which represent nearly half of all cells in the lungs. Radiation-induced damage to other cells, particularly alveolar macrophages and pneumocytes, may also contribute to the development of pulmonary fibrosis.

With low doses of inhaled radioactive particles, carcinogenesis is the most important hazard. The risk of neoplasia is connected with the proliferative potential of cells. Cells at risk are all precursors of renewal systems located in the lungs, and, if translocation to other organs occurs, in certain extrapulmonary tissues such as bone.

15.8.2 Tissues and Cells at Risk

At particular risk are:

1. Certain cells and tissues within or in association with the respiratory system; these include:

(a) Epithelial precursor cells in bronchioli, especially at the bronchioalveolar junction; possibly also type II pneumocytes (granular pneumocytes), and Clara cells. These groups are of major concern if in human beings most of the radiation dose is delivered by particles deposited in alveolar regions as in experimental animals.

(b) Basal cell layers of bronchial epithelia; these elements are at risk especially in the case of chronic inhalation of radioactive aerosols where a constant and/or repeated radiation dose is delivered by particles of various size transported by the mucociliary apparatus.

(c) Cellular constituents of bronchial and tracheobronchial lymph nodes, which, by progressive translocation of particles from the lung parenchyma, may accumulate large doses of radiation. Cells in this location having proliferative potential are: T and B lymphocytes, germinal center cells, plasma cell precursors, endothelial cells, and, possibly, reticulum cells, histocytes, fibroblasts, and other mesenchymal elements. However, radiation-induced lymph node neoplasia have been very rare in animals.

2. Certain extrapulmonary cells and tissues after translocation of radionuclides. These include: bone tissue, particularly on endosteal surfaces, and, to a much lesser extent, haematopoeitic marrow; liver tissue; spleen tissue, and other tissues.

The types of neoplastic disorders observed in animals may not adequately reflect the types of cells at risk in man. The predominant type of cancer found in miners was the small cell anaplastic carcinoma of large bronchi. Animals subjected to a single short-term inhalation develop mainly peripheral lung carcinomas of the adenocarcinoma, bronchiolar adenocarcinoma and epidermoid carcinoma type.

3. *Lymphocytes*: As a population of cells at risk, lymphocytes deserve special interest because blood lymphocytopenia is among the earliest and most sensitive changes caused by inhaled radioactive particles. The mechanism involved is probably irradiation of the circulating blood and lymph by particles deposited in alveolar and/or regional lymph nodes.

4. *Germ Cells*: Genetic damage to germ cells after inhalation of radionuclides is not excluded.

15.8.3 Risk Calculation

Risk coefficients were calculated from animal data and compared with values for human beings calculated largely from external radiation but included data from uranium miners. Linear and non-linear models used to evaluate the animal data would project risk coefficients between 2,000 and 160,000 cases per million animals per Gy. These span the range of values published for human beings. When described by probit models, the lowest risk coefficients were obtained. ICRP 31 believes that the results from animal experiments are generally supportive of the ICRP 26 lung cancer risk coefficient of 2×10^{-3} Sv^{-1}.

Animal data give risk coefficients 2 or 3 times greater for insoluble alpha emitters than for soluble alpha emitters. While this tends to support the concept of a slightly increased risk when alpha-emitting radionuclides are in insoluble particulate form in quantities sufficient to be distributed in hot spots, experiments designed specifically to detect hot particle effects have been negative.

15.8.4 Equal Effectiveness Ratio for Alpha Radiation

ICRP 31's analysis using a refined model yields a low risk estimate of 0.84×10^{-4} Gy^{-1} for beta-gamma emitters. Comparison of this value with the risk estimate obtained by similar analysis of all the alpha-emitting data, 25×10^{-4} Gy^{-1}, gives an Equal Effectiveness Ratio of 30 for inhaled alpha-emitting radionuclides. Equal Effectiveness Ratios of 6 and 40 are obtained when the alpha and beta-gamma dose-effect models are compared at 10 and 40% cancer incidence values respectively. Thus the experimental animal data tend to support the ICRP 26 recommendation of a quality factor (radiation weighting factor) of 20 for alpha radiation.

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Part III

Internal and External Dosimetry

CHAPTER 16

QUANTITIES USED IN RADIOLOGICAL PROTECTION

16.1 INTRODUCTION

The material contained in this chapter is taken from Chapter 2 of ICRP publication 60 (1991), entitled 'Quantities used in radiological protection', Annex A to ICRP 60 with the same title as Chapter 2, ICRP publication 42, 'A compilation of the major quantities used by ICRP' (1984), and Appendix A of ICRP publication 51, 'Data for use in protection against external radiation' (1987). Some of the quantities have already been briefly discussed in Chapter 2.

Historically, the quantities used to measure the 'amount' of ionizing radiation have been based on the gross number of ionizing events in a defined situation or on the gross amount of energy deposited in a defined mass of material. These approaches ignore consideration of the discontinuous nature of the process of ionization, but are justified by the observation that these quantities correlate fairly well with the resulting biological effects.

ICRP continues to recommend the use of macroscopic quantities, known as *dosimetric quantities*, although future developments may show that it would be better to use quantities based on the statistical distribution of events in a small volume of material corresponding to the biological cell or its nucleus (*microdosimetric quantities*).

For expressing the *primary* or *basic limits* in radiation protection, the main *radiation protection quantities* (ICRU, 1980) are: equivalent dose to an organ or tissue and effective dose (both the above for external radiation), and committed tissue or organ equivalent dose and committed effective dose (both for internal radiation). *Secondary limits* include the dose equivalent index (for external radiation) and the annual limit on intake (for internal radiation). *Derived limits* are normally expressed in measurable quantities. They are related to the basic limits by a defined model of the situation and are intended to reflect the basic limits.

The derived limits are called *radiometric quantities* (ICRU, 1980) if they relate to the radiation itself; examples are particle flux, particle fluence, and energy fluence. When radiometric quantities are combined with quantities associated with the interaction of radiation and matter, *dosimetric quantities* (ICRU, 1980) result: these include absorbed dose, kerma, ambient, directional, and individual dose equivalents.

We can classify the quantities into 5 groups, viz. (i) radiometric quantities; (ii) dosimetric quantities (which include all quantities used for expressing quantitatively the radiation exposure of individuals, whether identified or hypothetical persons); (iii) ICRU (International Commission on Radiation Units and Measurements) quantities (ICRU Reports 30 (1979), 33 (1980) and 39 (1985)); (iv) quantities used to express the radiation exposure of populations from defined sources of exposure; and (v) other quantities which ICRP uses. The International System of Units (SI) is used by ICRP.

16.2 GROUP I: RADIOMETRIC QUANTITIES

16.2.1. Activity

The activity, A, of an amount of radionuclide is dN/dt, where dN is the expectation value of the number of spontaneous nuclear transformations in the time interval dt.

The unit of activity is the reciprocal second, s^{-1}, with the special name *becquerel* (Bq). (The earlier unit of activity was the *curie* (Ci); 1 Ci = 3.7×10^{10} Bq)

16.2.2. Particle Flux

The particle flux, $\dot{N}$, is the quotient of dN by dt, where dN is the increment of particle number in time dt.

$\dot{N} = dN/dt$ (unit: s^{-1})

16.2.3. Particle Fluence

The particle fluence, Φ, is the quotient of dN by da, where dN is the number of particles incident on a sphere of cross-sectional area da.

$\Phi = dN/da$ (unit: m^{-2})

16.2.4. Energy Fluence

The energy fluence, ψ, is the quotient of dR by da, where dR is the radiant energy incident on a sphere of cross-sectional area da.

$$\psi = dR/da \quad \text{(unit: J m}^{-2}\text{)}$$

16.3 GROUP II: DOSIMETRIC QUANTITIES

16.3.1 Mass Energy Transfer Coefficient

The mass energy transfer coefficient, μ_{tr}/ρ, of a material for uncharged ionizing particles, is the quotient of dE_{tr}/EN by $\rho\, dl$, where E is the energy of each particle (excluding rest energy), N is the number of particles, and dE_{tr}/EN is the fraction of incident particle energy that is transferred to kinetic energy of charged particles by interactions in traversing a distance dl in the material of density ρ.

$$\mu_{tr}/\rho = \frac{1}{\rho EN}\frac{dE_{tr}}{dl} \qquad \text{(unit: m}^2\text{ kg}^{-1}\text{)}$$

16.3.2 Mass Energy Absorption Coefficient

The mass energy absorption coefficient, μ_{en}/ρ, of a material for uncharged ionizing particles is the product of the mass energy transfer coefficient, μ_{tr}/ρ, and $(1-g)$, where g is the fraction of the energy of the secondary charged particles that is lost to bremsstrahlung in the material.

$$\mu_{en}/\rho = \frac{\mu_{tr}}{\rho}(1-g) \qquad \text{(unit: m}^2\text{ kg}^{-1}\text{)}$$

16.3.3 Total Mass Stopping Power

The total mass stopping power, S/ρ, of a material for charged particles is the quotient of dE by ρdl, where dE is the energy lost by a charged particle in traversing a distance dl in the material of density ρ. S is the total linear stopping power.

$$S/\rho = \frac{1}{\rho}\frac{dE}{dl} \qquad \text{(unit: J m}^2\text{ kg}^{-1}\text{)}$$

E may be expressed in eV and hence S/ρ may be expressed in eV m^2kg^{-1}.

16.3.4 Linear Energy Transfer or Restricted Linear Collision Stopping Power

The linear energy transfer or restricted linear collision stopping power, L_{Δ}, of a material for charged particles is the quotient of dE by dl, where dE is the energy lost by a charged particle in traversing a distance dl due to those collisions with electrons in which the energy loss is less than Δ,.

$$L = \left(\frac{dE}{dl}\right)_{\Delta} \quad \text{(unit: J m}^{-1}\text{)}$$

E may be expressed in eV and hence L_{Δ} may be expressed in eV m^{-1}, or some convenient sub-multiple or multiple, such as keV μm^{-1}.

Notes:

(a) Although the definition specifies an energy cut-off and not a range cut-off, the energy losses are sometimes referred to as 'energy locally transferred'.

(b) In order to simplify notation, Δ may be expressed in eV. Then L_{100} is understood to be the linear energy transfer for an energy cut-off of 100 eV.

(c) $L_{\infty} = S_{col}$, the linear collision stopping power; it is generally denoted by L and is also called the unrestricted linear energy transfer.

It may not be out of place to define certain microdosimetric quantities connected with LET (Kellerer, 1985).

The *frequency distribution* of LET is defined in terms of the total track length of the charged particles, or, equivalently, in terms of the particle fluence. The *distribution function* (or *sum distribution*) $F(L)$ is the fraction of the total track length, i.e. the fraction of fluence, that is associated with linear energy transfer not larger than L:

$$F(L) = \phi_L/\phi$$

where ϕ is the total fluence and ϕ_L is the fluence of particles with LET not exceeding L.

The density of LET in track length or fluence is denoted by $f(L) = dF(L)/dL$. The *track* (or *frequency) average* is the mean value that corresponds to the distribution:

$$\bar{L}_F = \int Lf(L)dL = \int [1 - F(L)]\, dL$$

The *dose distribution* (or *weighted distribution*) of LET is defined in terms of the absorbed dose delivered by particles of specified LET. The distribution function $D(L)$ is the fraction of absorbed dose due to particles with linear energy transfer not larger than L:

$$D(L) = D_L/D$$

where D is the total absorbed dose (see definition in Sec 16.3.7), and D_L is the absorbed dose due to particles with LET not exceeding L.

The corresponding density of LET in dose is denoted by

$$d(L) = dD(L)/dL.$$

The *dose average* (or *weighted average*) is

$$\bar{L}_D = \int Ld(L)\,dL = \int [1 - D(L)]\,dL$$

The dose distribution of LET is related to the frequency distribution as:

$$d(L) = Lf(L)/\bar{L}_F$$

16.3.5 Mean Energy Expended in a Gas per Ion Pair Formed

The mean energy expended in a gas per ion pair formed, W, is the quotient of E by $\bar{N}$, where $\bar{N}$ is the mean number of ion pairs formed when the initial kinetic energy E of a charged particle is completely dissipated in the gas.

$$W = E/\bar{N} \quad \text{(unit: J)}$$

W may be expressed in eV.

16.3.6 Energy Imparted

The energy imparted, ε, by ionizing radiation to the matter in a volume is:

$$\varepsilon = R_{in} - R_{out} + \sum Q \text{ (unit : J)}$$

where R_{in} is the radiant energy incident on the volume, i.e. the sum of energies (excluding rest energies) of all those charged and uncharged ionizing particles which enter the volume; R_{out} is the radiant energy emerging from the volume, i.e. the sum of the energies (excluding rest energies)

of all those charged and uncharged ionizing particles which leave the volume; and ΣQ is the sum of all changes (decreases: positive sign; increases: negative sign) of the rest mass energy of nuclei and elementary particles in any nuclear transformation which occur in the volume.

16.3.7 Absorbed Dose

The fundamental dosimetric quantity in radiological protection is the absorbed dose, D. It is the energy deposited per unit mass, and its unit, the *gray* (Gy) is one joule per kilogram (J kg^{-1}). It is defined by the relation:

$$D = d\bar{\varepsilon}/dm,$$

where $d\bar{\varepsilon}$ is the mean energy imparted by ionizing radiation to the matter in a volume element of mass dm. (The previous unit of absorbed dose was the *rad*, corresponding to an energy absorption of 100 ergs per gram; 1 Gy = 100 rad.)

The time derivative of absorbed dose is the absorbed dose rate, $\dot{D}$:

$$\dot{D} = dD/dt.$$

16.3.8 Organ Dose

For radiation protection purposes, it is useful to define a tissue or organ average absorbed dose, D_T:

$$D_T = \varepsilon_T/m_T$$

where ε_T is the total energy imparted in a tissue or organ, and m_T is the mass of that tissue or organ.

16.3.9 Specific Energy

If the energy imparted is determined in a small mass, random fluctuations of energy deposition can be important. The specific energy (imparted), defined by ICRU, is given by:

$$z = \varepsilon/m$$

where ε is the energy imparted to a matter of mass m. The specific energy z is a stochastic quantity. The absorbed dose D (a macroscopic

quantity) is the limit of the mean specific energy imparted as m tends to zero.

16.3.10 Exposure

The quantity *exposure* (whose unit was the *roentgen*, R, and whose SI counterpart is coulomb per kilogram) was used widely in the early days of radiation measurement of X- and gamma rays but is now out of use, except as a quantity for reference standards.

The exposure, X, is the quotient of dQ/dm where dQ is the absolute value of the total charge of the ions of one sign produced in air when all the electrons (negatrons and positrons) liberated by photons in air of mass dm are completely stopped in air.

$$X = dQ/dm \quad \text{(unit: C kg}^{-1}\text{)}$$

$$1\,R = 2.58 \times 10^{-4}\ \text{C kg}^{-1}\ \text{(exactly)}.$$

16.3.11 Air Kerma

The quantity *exposure* is being replaced by the *air kerma*. Kerma (kinetic energy released in material), K, is defined by the relation:

$$K = dE_{tr}/dm \quad \text{(unit J kg}^{-1}\text{)}$$

where dE_{tr} is the sum of the initial kinetic energies of all the charged ionizing particles liberated by uncharged ionizing particles in a medium of mass dm. The special name of the unit for kerma is gray (Gy).

16.3.12 Lineal Energy

The lineal energy, y, is defined as

$$y = \varepsilon/\bar{l} \quad \text{(unit: keV } \mu\text{m}^{-1}\text{)}$$

where ε is the energy imparted to the matter in a volume of interest by an energy deposition event and $\bar{l}$ is the mean chord length in that volume. (For a spherical site of diameter d, we have $\bar{l} = 2d/3$.)

The definition of y is restricted to energy imparted in one event; y is the stochastic analogue of the macroscopic quantity LET. The single-

event distributions of lineal energy are equivalent to the single-event distributions of specific energy. We can define the frequency mean lineal energy $\bar{y}_F$ (which is analogous to $\bar{L}_F$, the track or frequency average LET), and the weighted distribution (or dose distribution) of lineal energy $\bar{y}_D$ (which is analogous to $\bar{L}_D$, the weighted distribution of LET).

Since the mean lineal energy represents discrete energy deposition, in principle, it is more meaningful than LET as the physical quantity to be used in the specification of radiation quality. Although y is directly measurable, L has been used in most of the existing radiation protection calculations. Therefore, the *quality factor*, Q, is given by ICRP as a function of L.

16.3.13 Quality Factor

Since the probability of stochastic effects depends on the quality of the radiation, a weighting factor has been traditionally introduced to modify the absorbed dose, and to define the equivalent dose. This dimensionless factor, called the *quality factor*, Q, is given as a function of the unrestricted linear energy transfer, which is a measure of the density of ionization along the track of an ionizing particle.

Q-L Relationship: ICRP has modified its recommendations on the formal relationship between the quality factor, $Q(L)$, and unrestricted linear energy transfer, L, to reflect our lack of precise information and an appreciation of the practical aspects of radiation protection. For example, ICRP does not believe it is helpful to adopt different quality factor values for different photon energies. ICRP also recognizes the reduced effectiveness of heavy ions with L greater than 110 keV/μm. The formulation given in Table 16.1 is adopted.

16.3.14 Radiation Weighting Factor

ICRP now believes that the detail and precision inherent in using a formal Q-L relationship to modify absorbed dose to reflect the higher probability of detriment resulting from exposure to high LET radiations is not justified because of the uncertainties in the radiobiological information. In place of Q, or more precisely $\bar{Q}$, ICRP now selects radiation weighting factors, W_R, based on a review of the biological information, a variety of exposure circumstances and inspection of the results of traditional calculations of the ambient dose equivalent.

ICRP now specifies modifying factors which apply to the tissue or

organ absorbed dose and are based on the type and quality of the external radiation field or on the type and quality of the radiation emitted by an internally deposited radionuclide. The specified values of W_R have already been given in Table 2.1 (Chapter 2).

To assist in providing consistency in calculations, a smooth fit to the W_R values for neutrons as a function of neutron energy is given in Fig. 16.1. The mathematical relationship is:

$$W_R = 5 + 17 \exp. \{-\ln(2E)^2/6\}$$

where E is the neutron energy in MeV. There is no intention to imply any biological meaning to this relationship; it is simply a calculational tool.

Auger electrons emitted from nuclei bound to DNA present a special problem because it is not realistic to average the absorbed dose over the whole mass of DNA as would be required by the present definition of equivalent dose. The effects of Auger electrons have to be assessed by the techniques of microdosimetry.

For radiation types and energies which are not included in Table 16.1, an approximation can be obtained by calculation of $\bar{Q}$ at a 10 mm depth in the ICRU sphere:

$$\bar{Q} = (1/D) \int_0^\infty Q(L)\, D(L)\, dL$$

where $D(L)\, dL$ is the absorbed dose at 10 mm between linear energy transfer L and $L + dL$, and $Q(L)$ is the quality factor of L at 10 mm. The Q-L relationships have already been given in Table 16.1. Figure 16.2 demonstrates the application of this formulation to photons and can be seen to give values consistent with the recommended values of W_R.

16.3.15 Equivalent Dose in an Organ or Tissue

The equivalent dose, $H_{T,R}$, in tissue or organ T due to radiation R, is given by:

$$H_{T,R} = W_R \cdot D_{T,R}$$

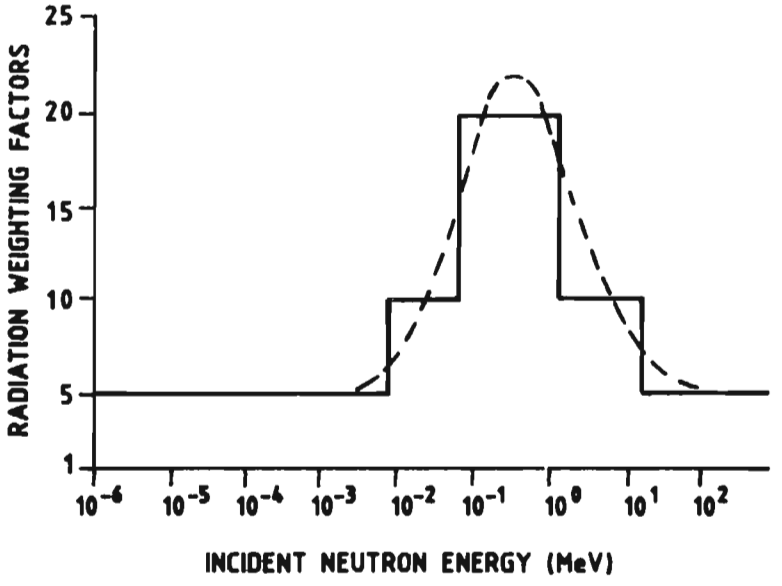

Figure 16.1. **Radiation weighting factors for neutrons (from ICRP 60).**

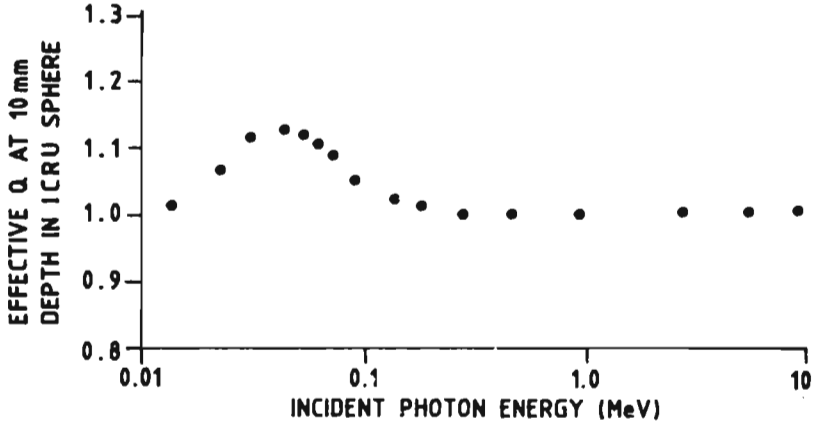

Figure 16.2. **Effective Q ($\overline{Q}$) as a function of photon energy (from ICRP 60).**

where $D_{T,R}$ is the average dose from radiation R in the tissue or organ T and W_R is the radiation weighting factor. (The earlier quantity corresponding to the equivalent dose was the dose equivalent at a point, H.) Since W_R is dimensionless, the SI unit of equivalent dose is the same as for absorbed dose, namely $J\ kg^{-1}$, and its special name is *sievert* (Sv). (The earlier unit of equivalent dose was the *rem*; 1 Sv = 100 rem.) The time derivative of the equivalent dose is the equivalent dose rate, $\dot{H}_{T,R}$.

When the radiation field is composed of types and energies with different values of W_R, the absorbed dose must be subdivided into blocks, each block multiplied by its own value of W_R, and summed to determine the total equivalent dose, i.e.

$$H_T = \sum_R W_R \cdot D_{T,R}$$

where $D_{T,R}$ is the average absorbed dose from radiation R in tissue T. Alternatively, the absorbed dose resulting from increments of energy between E and $E + dE$ can be multiplied by the W_R values obtained from Table 2.1 (of Chapter 2), or as an approximation from the continuous function given above illustrated in Fig. 16.1, and integrated over the energy spectrum to determine the total equivalent dose.

16.3.16 Tissue Weighting Factors and Effective Dose

The relationship between the probability of stochastic effects and equivalent dose also varies with the organ or tissue irradiated. To indicate the combination of different doses to several tissues which will correlate well with the total of stochastic effects, we define the *tissue weighting factor*, W_T, by which the equivalent dose in tissue or organ T is weighted. This factor represents the relative contribution of that organ or tissue to the total detriment due to these effects resulting from uniform irradiation of the whole body. The values of W_T are chosen so that a uniform equivalent dose over the whole body gives an effective dose numerically equal to that uniform equivalent dose. The sum of the tissue weighting factors is then unity. This weighted equivalent dose (a doubly weighted absorbed dose) is the *effective dose*, E (previously called the *effective dose equivalent*). The unit of effective dose is $J\ kg^{-1}$, with the special name sievert (Sv).

$$E = \sum_T W_T H_T$$

where H_T is the equivalent dose in tissue or organ T and W_T is the weighting factor for tissue T.

Evidently:

$$E = \sum_R W_R \sum_T W_T \cdot D_{T,R} = \sum_T W_T \sum_R W_R \cdot D_{T,R}$$

where $D_{T,R}$ is the mean absorbed dose in tissue or organ T delivered by radiation R. The recommended values for tissue weighting factors have already been given in Table 2.2 (Chapter 2).

The radiation weighting factor is taken to depend only on the type and energy of radiation and independent of the tissue or organ. Similarly, the tissue weighting factor is taken to be independent of the type and energy of the radiation. These are simplifications and may be only approximations to the true biological situation.

The biological effects depend, in addition to the above factors, on the distribution of the dose in time (dose rate and protraction of exposures). (The previous use of an unspecified weighting factor N to take into account effects like the above, which, however, had always been given the value of unity, is now discontinued by ICRP.) In case it is considered desirable to modify the values of W_T or W_R in the light of newer radiobiological findings, this can be done in the present formalism and the effective dose recalculated.

Equivalent dose and effective dose are quantities intended for use in radiological protection, including general risk assessments. They provide a basis for estimating the probability of stochastic effects only for absorbed doses well below the threshold for deterministic effects.

16.3.17 Committed Tissue or Organ Equivalent Dose and Committed Effective Dose

Tissue irradiation from internally incorporated radionuclides is spread out in time, energy deposition occurring as the radionuclide decays. To take account of this time distribution, ICRP recommends the use of committed equivalent dose which is the time integral over time τ of the equivalent dose rate in a particular tissue that will be received by an individual following an intake of radioactive material. When the period of integration is not given, a period of 50 years is implied for adults or a period of 70 years for children.

The committed equivalent dose is defined by

$$H_T(\tau) = \int_{t_0}^{t_0+\tau} \dot{H}_T(t)\, dt$$

for a single intake of activity at time t_0, where $H_T(t)$ is the relevant equivalent dose rate in an organ or tissue at time t, and τ is the time period over which the integration is performed.

Figure 16.3 gives the equivalent dose rate in a tissue as a function of time after an intake of radionuclides with short and long effective half-lives. This shows the relationship between the equivalent dose rate in the tissue and the committed equivalent dose which is the total area.

The committed effective dose, $E(\tau)$ is defined as:

$$E(\tau) = \sum W_T . H_T(\tau)$$

where τ is in years.

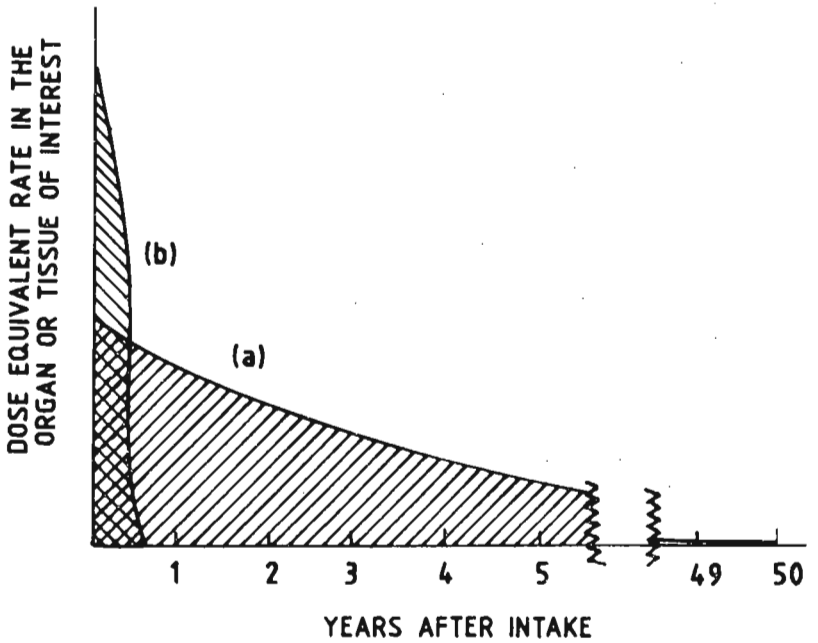

Figure 16.3. Equivalent dose rate in a given organ or tissue following intake of a radionuclide with (a) long and (b) short effective half-lives (from ICRP 42).

16.4 GROUP III: ICRU QUANTITIES FOR ENVIRONMENTAL AND INDIVIDUAL MONITORING (ICRU, 1985)

These quantities are helpful approximations to the effective dose and the equivalent dose to the skin when these quantities are calculated using the *Q-L* relationship given in Table 16.1. Their determination can be done separately for environmental (including area) and individual monitoring. (ICRP will be examining these quantities in detail as part of a general revision of ICRP 51 (1987) which will incorporate the new radiation weighting factors.) They are based on the concept of the dose equivalent at a point in the ICRU sphere. This is a 30 cm diameter, tissue-equivalent sphere with a density of 1 g cm^{-3} and a mass composition of 76.2% oxygen, 11.1% carbon, 10.1% hydrogen and 2.6% nitrogen. Trace elements are not generally considered important for these dosimetric purposes and have been ignored.

In defining the quantities associated with these 'concepts', it is useful to stipulate certain radiation fields that are derived from the actual radiation field. The terms *expanded* and *aligned* are given by ICRU (1985) to characterize these derived radiation fields. In the expanded field, the fluence and its angular and energy distribution have the same values throughout the volume of interest as the actual field at the point of reference. In the aligned and expanded field the fluence and its energy distribution are the same as in the expanded field but the fluence is unidirectional.

16.4.1 Environmental Monitoring

Two concepts linking the external radiation field to the effective dose, and to the equivalent dose in the skin, are introduced for purposes of environmental and area monitoring.

The ambient dose equivalent, $H^*(d)$, is appropriate for strongly penetrating radiation, while the directional dose equivalent, $H'(d)$, is suitable for weakly penetrating radiation. (If the equivalent dose received by any small area of the sensitive layer of the skin is more than 10 times the effective dose for a given orientation of the body in a uniform and unidirectional radiation field, the radiation is said to be *weakly penetrating*; in other cases, it is said to be *strongly penetrating*.)

16.4.2 Ambient Dose Equivalent

The ambient dose equivalent, $H^*(d)$, at a point in a radiation field, is the dose equivalent that would be produced by the corresponding aligned and expanded field in the ICRU sphere at a depth, d, on the radius opposite the direction of the aligned field. The recommended depth, d, for monitoring in terms of $H^*(d)$ is 10 mm and $H^*(d)$ may be written in this case as $H^*(10)$.

16.4.3 Directional Dose Equivalent

The directional dose equivalent, $H'(d)$, at a point in a radiation field, is the dose equivalent that would be produced by the corresponding expanded field in the ICRU sphere at a depth, d, on a radius in a specified direction. The depth, d, recommended for monitoring in terms of $H'(d)$ is 0.07 mm, and $H'(d)$ may in this case be written as $H'(0.07)$.

16.4.4 Absorbed Dose Index

The absorbed dose index, D_I, at a point is the maximum absorbed dose within the ICRU sphere. Its unit is the gray (Gy).

Notes:

(a) As the outer 70 μm thick layer of skin is composed of dead cells, the magnitude of the absorbed dose in the outer 70 μm thick layer of the sphere is ignored in determining the absorbed dose index.

(b) The maximum absorbed dose due to different components of a complex radiation field may occur at different locations in the sphere. Hence the absorbed dose index for the radiation field as a whole is generally less, and never more, than the sum of the values for its components.

16.4.5 Dose Equivalent Index

The dose equivalent index, H_I, at a point is the maximum dose equivalent within the ICRU sphere. Its unit is the sievert (Sv).

Notes:

(a) As with the absorbed dose index, the magnitude of the dose equivalent in the outer 70 μm thick layer of the sphere is ignored in determining the dose equivalent index.

(b) As the dose equivalent in the sphere usually decreases with depth in the sphere, and as higher dose equivalent limits are usually given for organs near the surface of the body, it is convenient to divide the sphere into two outer shells and a core. The outermost shell has a thickness of 70 μm and values of dose equivalent in this shell are ignored. The second shell extends from a depth of 70 μm to 1 cm. The maximum dose equivalent in this shell is termed the *shallow dose equivalent index*, $H_{I,s}$. The maximum dose equivalent in the core is termed the *deep dose equivalent index*, $H_{I,d}$. Collectively, these two indices are termed the *restricted dose-equivalent indices*.

(c) In general, the maximum values of the quality factor, (Q_{max}), and the absorbed dose (D) occur at different locations in the sphere, and, therefore, the location of maximum H can be different from that of the maximum D. However, $Q_{max}.D_I$ will not underestimate H_I.

16.4.6 Individual Monitoring

Two concepts are introduced for individual monitoring. The individual dose equivalent, penetrating, $H_p(d)$, is appropriate for organs and tissues deeply situated in the body which will be irradiated by strongly penetrating radiation. The individual dose equivalent, superficial, $H_s(d)$, is suitable for superficial organs and tissues which will be irradiated by both weakly and strongly penetrating radiation.

16.4.7 Individual Dose Equivalent, Penetrating

The individual dose equivalent, penetrating, $H_p(d)$, is the dose equivalent in soft tissue (defined as in the ICRU sphere) below a specified point on the body at a depth, d, that is appropriate for strongly penetrating radiation. The recommended depth, (d), for monitoring in terms of $H_p(d)$ is 10 mm, and in this case $H_p(d)$ may be written as $H_p(10)$.

16.4.8 Individual Dose Equivalent, Superficial

The individual dose equivalent, superficial, $H_s(d)$, is the dose equivalent in soft tissue (defined as in the ICRU sphere) below a specified point on the body at a depth, d, that is appropriate for weakly penetrating radiation. The recommended depth, d, for monitoring in terms of $H_s(d)$ is 0.07 mm, and in this case $H_s(d)$ may be written as $H_s(0.07)$.

16.5 GROUP IV: QUANTITIES CONNECTED WITH POPULATIONS

16.5.1 Collective Equivalent Dose

The collective equivalent dose, S_T, in tissue T (which expresses the total exposure of a specific tissue or organ, T, in a group of individuals), is given by

$$S_T = \int_0^\infty H_T \cdot (dN/dH_T) \cdot dH_T$$

where $(dN/dH_T).dH_T$ is the number of individuals receiving an equivalent dose between H_T and $H_T + dH_T$, or by

$$S_T = \sum \bar{H}_{T,i} N_i$$

where N_i is the number of individuals in the population subgroup I receiving mean organ equivalent dose $\bar{H}_{T,i}$.

16.5.2 Collective Effective Dose

The collective effective dose, S (which is a measure of the exposure in a population), is given by

$$S = \int_0^\infty E(dN/dE) \cdot dE, \quad \text{or} \quad \bar{E}_i \cdot N_i$$

where $\bar{E}_i$ is the mean effective dose to population subgroup i. (The time period and population over which S is summed or integrated should be specified.)

16.5.3 Per Caput Equivalent Dose

If a population is uniformly irradiated and the population size increases, then the collective equivalent dose will increase proportionately. It is sometimes useful to express results in terms of equivalent doses to a hypothetical average individual. This is the *per caput equivalent dose*, *H(t)*. Although it appears to be referring to an individual, it is included under population-oriented quantities, because it only coincidentally represents the equivalent dose to an actual individual.

The per caput equivalent dose may be obtained by dividing the collective equivalent dose over a given time in a specified population by the number of individuals in the population at the time, or, more directly, by calculating the average absorbed dose rate, or intake of radionuclides, from the source.

16.5.4 Critical Groups

Dose limits for members of the public are intended to be applied to the average effective dose to the members of the *critical group.* This group should be representative of those individuals in the population expected to receive the highest equivalent doses. It should be small enough to be relatively homogeneous with respect to age, diet and those aspects of situation and behaviour that affect the doses received. The size of most critical groups will be a few tens of persons.

In arriving at the critical group, the pathways of exposure from the radionuclides have to be investigated. The pathway leading to the exposure of the critical group is the *critical pathway* and the radionuclide that gives the highest dose the *critical nuclide.*

16.5.5 Dose Commitment (for Populations)

The dose commitment ($H_{c,T}$ or E_c) is a calculational tool, which can be assessed for a critical group or for the whole population. It is the infinite time integral of the per caput dose rate ($\dot{H}_T$ or $\dot{E}$) due to a specific event, such as a unit of practice (e.g. a year of practice).

$$H_{c,T} = \int_0^\infty \dot{H}_T(t)\,dt\,; \quad E_c = \int_0^\infty \dot{E}(t)\,dt$$

In the case of an indefinite practice at a constant rate, the maximum annual per caput dose rate ($\dot{H}_T$ or $\dot{E}$) in the future for the specified population will be equal to the dose commitment of one year of practice, irrespective of changes in the size of the population. If the practice is continued only over a time period, τ, the maximum future annual per caput dose will be equal to the corresponding *truncated dose* where the upper limit of integration in the above equations is τ years.

16.6 GROUP V: OTHER QUANTITIES

16.6.1 Secondary Limits

16.6.1.1 Annual Limit on Intake

In order to simplify the comparison of the committed effective doses from intakes with the equivalent dose limits, it is convenient to define the secondary dose limit called the annual limit on intake, ALI. It normally

corresponds to a committed effective dose from an intake of a given radionuclide equal to the appropriate equivalent dose limit for workers. Restriction of intake in each year to less than the ALI therefore ensures that the maximum annual equivalent dose from that radionuclide will always be less than the equivalent dose even if intake occurred every year for 50 years.

For some specific radionuclides ALI values may be dictated by deterministic rather than stochastic dose limits.

16.6.1.2 Derived Air Concentration (DAC)

This is obtained by dividing the ALI by the value of air inhaled by the Reference Man in a working year of 2000 hours at a breathing rate of 1.2 $m^3\ h^{-1}$.

(For short-lived radionuclides there may be a substantial additional contribution from external radiation which should be assessed as part of the external radiation. DACs are also derived, where appropriate, for submersion of Reference Man in a cloud of radioactive inert gases or tritium.)

In deriving ALI or DAC values attention will have to be given to variations in anatomical and metabolic chracteristics from the adult Reference Man in calculation of the doses in the case of children who are part of members of the public.

16.6.2 Derived Limits

In many practical situations it is difficult to relate measured quantities directly to the dose limits. It is therefore useful to provide limits asociated with quantities actually measured (e.g. contamination levels in environmental materials). When they are related to the basic limits by a defined model, they are called *derived limits*. When they are related to the source upper bound they are called *derived upper bounds*.

A derived limit may be calculated for a specific and well characterized situation (in which case the derived limit will correspond closely to the dose limit), or the model may be generalized, in which case the derived limit will be more conservative to take account of uncertainties.

16.6.3 Authorized Limits

It is normal for practical working limits on dose rate, release rate of radionuclides, or concentrations in environmental materials to be set by national authorities or managements. These are the *authorized limits*. They will, in general, be below the derived limits or derived upper bounds.

Authorized limits for the public are generally applied to control the doses to the members of the critical group.

16.7 OTHER QUANTITIES USED BY ICRP IN A GENERAL SENSE

In relating the probability of stochastic effects to dosimetric quantities, it is convenient to use a probability coefficient. The *fatality probability coefficient* is the quotient of the probability that an increment of dose will cause death and the magnitude of that increment of dose.

ICRP uses generic terms that can apply to any of the dosimetric quantities. *Dose* is one such term used in phrases such as *dose limit*, which may be a limit applied to equivalent or effective dose. *Exposure* is also used in a generic sense to mean the process of being exposed to radiation or radioactive material. The significance of an exposure in this sense is determined by the resulting doses.

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Table 16.1: Specific Q-L relationships
(from ICRP 60)

Unrestricted linear energy transfer in water (keV/μm)	$Q(L)$*
< 10	1
10 - 100	0.32L - 2.2
> 100	$300/L^{0.5}$

* L expressed in keV/ μm.

CHAPTER 17: SECTION 1

REFERENCE MAN: INTRODUCTION

17(1).1 CONCEPT OF THE REFERENCE MAN

ICRP publication 23, 'Reference man: anatomical, physiological and metabolic characteristics' (1975) is a report prepared by a Task Group of Committee 2 of ICRP.

Estimation of radiation doses to the human body requires a certain amount of data about the exposed individual. In the case of external sources, fairly simple specifications of mass, dimensions, and elemental composition of the organs and tissues are largely sufficient. However, for dosimetry of radionuclides entering the body, it is necessary to have much more biological information. Data are required on daily intakes of air and water or of elements which serve as carriers and, to some extent, influence the uptake of other elements, the breathing rate, excretion, and parameters governing the elimination rates of material from various tissues or from the body. Since the uptake and retention in man of many radionuclides cannot be determined accurately, data on the corresponding stable elements are often used for constructing a model. The age dependence of such factors is also necessary to be considered.

Although individuals vary considerably in such respects, it is important to have a well-defined reference individual for routine dosimetric purposes when the levels are sufficiently low that individual differences may be ignored. Such a reference individual will enable health physicists to compare and check their results.

17(1).2 HISTORICAL DEVELOPMENT

In the early post-world war II years it became necessary to make recommendations concerning many new radioactive materials. At the 1949 Chalk River Conference on Permissible Dose (attended by representatives from UK, Canada and USA), the first 'Standard Man' data were formalized, although certain values had general acceptance before that time. The Standard Man values were modified at subsequent meetings of

the International Congress of Radiology and other bodies. The gastrointestinal (GI) tract model was developed. ICRP publication 2, 'Report of Committee II on Permissible Dose for Internal Radiation' (1960) contained data on 46 naturally occurring elements as compared to 15 in the previous 1954 ICRP report. Also a report listing the concentration values of 44 naturally occurring elements in 36 tissues was included.

[Author's note: ICRP publication 30 has superseded ICRP publication 2.]

In 1963 a Task Group of Committee 2 was formed for revision and extension of the Standard Man concept. The name was changed from 'Standard' Man to 'Reference' Man. The Reference Man was to be defined, in the first instance, as a typical occupational individual; variability about the values for the Reference Man was to be indicated, and differences due to age, sex or habits were also to be indicated with emphasis on fetuses, children and adults.

17(1).3 DEFINITION, IMPORTANCE AND LIMITATIONS OF REFERENCE MAN.

Considering the easier availability to the Task Group of data on the European and American populations, the Reference Man was defined as being 20-30 years of age, weighing 70 kg, of height 170 cm, and living in a climate with a temperature range of 10-20°C. He is a Caucasian and a Western European or North American in habitat and custom.

The importance of the Reference Man concept is that his characteristics are defined rather precisely, and thus, if adjustments for individual differences are to be made, there is a known basis for making them (by the national authorities concerned with radiation protection) suited to the population at risk. The values selected by the Task Group are often average or median values, and are generally rounded to two digits. ICRP 23 gives extensive references to the sources from which the data have been derived (a total of over 1660 references).

ICRP has drawn attention to the limitations of some of the data. In many cases the data available in the literature were based on samples of only a few individuals. The analytical procedures in some cases leave much to be desired.

17(1).4 SCOPE OF ICRP 23

ICRP 23 report is organized as follows:

Introduction: Historical development of the concept and generalities applying to all sections;

Chapter 1: Data relating to weights, dimensions, physical characteristics, and composition of biological tissues (e.g., blood, fat, etc.);

Chapter 2: Data relating to elemental composition of tissues and whole body;

Chapter 3: Data relating to intake and excretion.

The reports of the Task Groups on Lung Dynamics and the GI Tract, both published in *Health Physics*, Vol. 12 (February 1966) supplement ICRP 23 in many respects.

A partial summary of Reference Man data is also presented in ICRP 23. Specific absorbed fractions of photon energies for Reference Man are presented as an appendix.

CHAPTER 17: SECTION 2

ANATOMICAL VALUES FOR REFERENCE MAN

17(2).1 INTRODUCTION

Chapter 1 of ICRP 23, 'Anatomical values for reference man' has 13 subsections giving data for the whole body and 11 other systems, as well as data for pregnancy. There is a description of the physiological function of each system and its anatomy: data are given on weight, dimensions and shape, and composition, as a function of prenatal gestational age, as well as various postnatal ages from birth to 80 years, for both males and females, depending upon the availability of the data based on an extensive literature survey. In the interests of conserving space, it became necessary to restrict the presentation of data in this Handbook mainly to representative adult values. Summary anatomical data for Reference Adult Male and Adult Female are given in Tables 17(2).1A and 17(2).1B. Some further discussions follow.

17(2).2 TOTAL BODY

17(2).2.1 Weight (W), Length (L), and Surface Area (SA) of the Total Body and Regions of the Body

Many anthropometric measurements are approximately Gaussian (Garn, 1960); however, the distribution of total body weight is skewed to the right, approximating a log-normal distribution (Grasbeck and Fellman, 1968).

Figures are given in ICRP 23 for the variation of weight (W) and length (L) of the total body as a function of gestational age, as well as a function of age from birth to 55 years for males and females. Total body weight tends to increase throughout life, but the increase is much less after 20 years of age for males and 16 for females.

A widely used formula relating W, L, and SA (DuBois and DuBois, 1916) is:

$$SA = (71.84)\,(W)^{0.425}\,(L)^{0.725}$$

where SA is in cm^2, W is in kg, and L is in cm.

The 'rule of nines' (Gardner *et al.*, 1963; Long *et al.*, 1961) may be used for estimating the SA of specific regions of the body: Head and neck - 9%; Upper limbs - 2 x 9%; Lower limbs - 2 x 18%; Front of trunk - 18%; Back of trunk - 18%; Perineum - 1%; Outstretched palm and fingers - 1%.

17(2).2.2 Growth Patterns for Regions of the Total Body during Post-natal Life

Four different general growth patterns (general, lymphoid, neural, and genital) can generally be defined, as shown in Fig.17(2).1.

17(2).2.3 Composition of the Total Body

Table 17(2).2 gives the composition of the total adult body.

17(2).2.4 Body Cell Mass

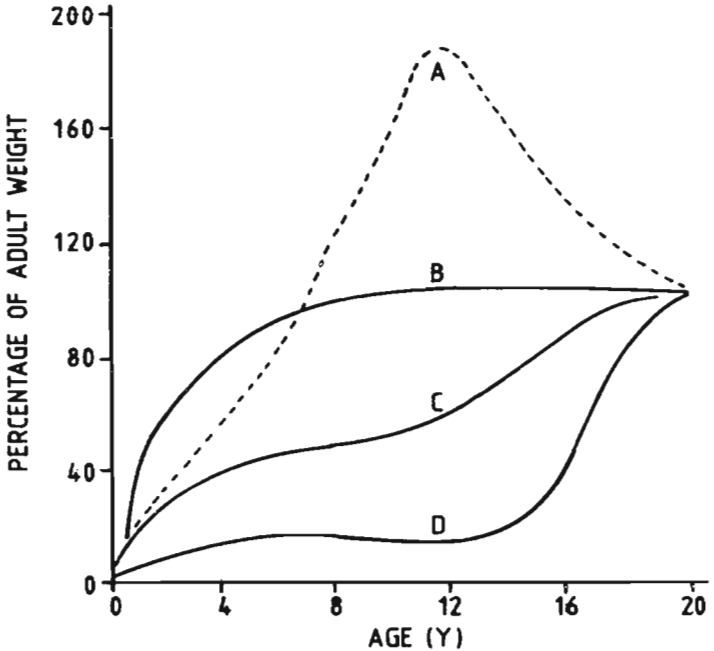
The body cell mass for adult males is around 41.9 kg and for females 27.4 kg (Burmeister and Bingert, 1967).

17(2).2.5 Total Number of Cells in Body (Cheek, 1968)

Fetus, 7 week	1.3×10^9
Fetus, 21 week	2.2×10^{11}
Newborn	2×10^{12}
Adult male	6×10^{13}
Average diameter of body cell	100 μm

17(2).2.6 Total Body Water (TBW), Extracellular Water (ECW), and Intracellular Water (ICW)

Total body water (TBW) may be divided into two major components, ECW and ICW, which are separated by the cell membranes. The ICW is the site of metabolic processes, and the ECW provides a constant external environment for the cells (Ruch and Patton, 1965). Table 17(2).3 gives the distribution of TBW between different compartments.



A LYMPHOID TYPE : THYMUS, LYMPH NODES, INTESTINAL LYMPHOID MASSES

B NEURAL TYPE : BRAIN AND ITS PARTS, DURA, SPINAL CORD, OPTIC APPARATUS, MANY HEAD DIMENSIONS

C GENERAL TYPE : BODY AS A WHOLE, EXTERNAL DIMENSIONS (WITH EXCEPTION OF HEAD AND NECK) RESPIRATORY AND DIGESTIVE ORGANS, KIDNEYS, AORTA AND PULMONARY TRUNKS, SPLEEN, MUSCULATURE AS A WHOLE, SKELETON AS A WHOLE, BLOOD VOLUME

D GENITAL TYPE : TESTIS, OVARY, EPIDIDYMIS, UTERINE TUBE, PROSTATE, PROSTATIC URETHRA, SEMINAL VESICLES

Figure 17(2).1. Four general postnatal growth patterns according to age (from ICRP 23, reproduced with permission from the University of Minnesota Press, Minneapolis).

17(2).2.7 Total Blood, Erythrocytes, Leukocytes, Platelets and Plasma

The various types of circulating blood cells (mainly erythrocytes) constitute 46% by volume of whole blood, and plasma about 54% (Best and Taylor, 1966). Table 17(2).4 gives the composition and other characteristics of blood in adults.

17(2).2.8 Body Fat, Adipose Tissue and Lean Body Mass

Body fat consists of two histological entities, 'essential' and 'non-essential' fat. Essential fat is composed of lipid constituents of cells and constitutes 2-5% of LBM. It is present even during starvation. Non-essential fat (storage fat or excess fat) is contained in adipose tissue which occurs principally in subcutaneous tissue, yellow bone marrow and the abdominal cavity; it accumulates or is utilized in response to alterations in caloric balance (Behnke, 1964). Adipose tissue is a poorly cellulated type of connective tissue. In obesity, an unusually large amount of fat is laid down as adipose tissue.

In females, total body fat continues to increase throughout life, from about 12% at birth to 35% at age 60 years. For males, it increases from 12% at birth to 17% at age 10, then falls to 12% at age 15, and thereafter increases with age to 26% at 60 years. Beyond the age of 10, the values are always smaller for males than for females (Anderson, 1963; Cheek, 1968; Cresta and Passarello, 1965; Forbes, 1964; Moore *et al.*, 1963; Novak, 1963; Owen *et al.*, 1966; Young *et al.*, 1963).

Lean body mass differs from fat-free body mass since it includes essential fat, which is present even during starvation.

17(2).2.9 Reticuloendothelial System

Reticuloendothetical system (RES) refers to the network of reticulum cells in certain tissues such as spleen, bone marrow and lymphoid tissues, together with littoral cells of the sinuses in these tissues and in the liver (Loutit, 1960). A common feature of RES cells is their phagocytic function. The weight of RES is 1800 g (60% in liver, 5% in spleen and 35% in lymph nodes and other tissues) (Snell, 1960; Wintrobe and Lee, 1970).

17(2).2.10 Connective Tissue (Bloom and Fawcett, 1968)

Connective tissue consists of cells and extracellular fibres embedded in an amorphous substance containing tissue fluid. It can be loose or dense. Connective tissues bind together or support various tissues of the body; they include cartilage (which contains no blood or nerves), tendons (which help to attach muscle to bone), and the fascia. The superficial fascia is a layer immediately under the skin which covers the entire body.

17(2).3 INTEGUMENTARY SYSTEM

17(2).3.1 Skin and Hypodermis

The anatomy of the skin has been discussed in Chapter 11, Section 4. The water content in fresh whole skin is around 80% during fetal life, decreasing to about 68% in the newborn and 62% in adolescents and adults (Meyer, in Rothman, 1954).

The skin contains the following glands: (i) eccrine sweat glands (2-5 x 10^6 in the adult), distributed over most of the body surface, being most numerous in the palms and soles (around 420/cm^2); (ii) apocrine sweat glands (located in the axilla, external auditory meatus, eyelids, and circumanal region) and which are attached to the hair follicles; and (iii) sebaceous glands, mostly associated with hair, especially on the scalp, forehead, cheeks, and chin (density 400-900/cm^2) (Montagna, 1956; Wray, 1964).

17(2).3.2 Hair and Nails

The number of hairs per cm^2 varies from 40 to 880, depending on the region of the body (Gardner *et al.*, 1963). The nails grow in length and thickness, chiefly through cellular activity in the proximal region of the nail bed bone. The length varies from 10 to 13 mm and the breadth from 9 to 13.5 mm, depending upon the finger.

17(2).4 SKELETON, CARTILAGE, NONSKELETAL DENSE CONNECTIVE TISSUE (TENDONS, FASCIA, PERIARTICULAR TISSUE), AND TEETH

The anatomy and biodynamics of bone as well as bone dosimetry are discussed in Chapter 11, Section 3 and Chapter 23. Some further aspects are summarized below.

17(2).4.1 The Skeleton

The skeleton is an anatomical structure which includes bone, skeletal cartilage, certain periarticular tissues adhering to joints, and red and yellow marrow. When cartilage, periarticular tissue, and red and yellow marrow are removed, the remainder of the skeleton may be classified as bone.

The relative dry weights for important bones (as percentages of the weight of the total adult skeleton) are given below (ICRP 23; Ingalls, 1931):

Skull	13.1
Clavicle, scapula (2 sets)	4.2
Humerus, radius, ulna (2 sets)	12.1
Hands (2)	2.8
Ribs (24)	6.6
Spine	11.6
Pelvis	11.3
Femur, tibia, fibula (2 sets)	31.5
Feet (2)	6.0

From about 40 to 80 years of age, about 25% of the absolute skeletal volume is lost (Frost, 1963). There is a loss in cortical thickness of 20% in males and 30% in females, with the largest percentage occurring after the fifth decade (Garn *et al.*, 1966).

17(2).5 HAEMATOPOIETIC SYSTEM, LYMPHATIC SYSTEM, SPLEEN, AND THYMUS

17(2).5.1 Haematopoietic System

In addition to the major function of producing blood cells, the tissues where haematopoiesis occurs also destroy blood cells, produce antibodies, carry out phagocytosis and other functions of the reticuloendothelial tissues and play an important role in the immunological system. During prenatal life the major phases of haematopoiesis occur in the yolk sac, liver, bone marrow, and thymus. The major postnatal haematopoietic organs are red bone marrow, lymph nodes, spleen, and thymus (Weiss, 1966).

17(2).5.2 Bone Marrow

Bone marrow is encased within all bones of the body and enclosed by the endosteum which is the lining of the bone cavities. Bone marrow consists of a variety of blood cells and their precursors, fat cells, a framework of reticulum (the stroma) which is connected with the endosteum and in close contact with blood vessels, sinusoids, and an intercellular substance containing mucopolysaccharides.

There are two kinds of marrow: red and yellow. The red marrow is haematopoietically active, while the yellow marrow has no haematopoietic function and consists principally of fat tissue. The proportion of these two types differs normally in different portions of the skeleton and with age; variations of this ratio and the relative distribution of red and yellow marrow are also an indication of certain diseases.

At birth, nearly all of the bone marrow is red. Until about 18 years of age, the yellow marrow tends to replace the red marrow progressively in the distal portions of the skeleton as the person ages. At ages greater than 18 years, red marrow is found predominantly in the vertebrae, sternum, ribs, skull, pelvis, and the proximal ends of the femurs and the humerii.

All erythrocytes (red blood cells), granulocytes and monocytes (two types of white blood cells), platelets (subcellular structures that are important for the coagulation of the blood), and a significant portion of lymphocytes (another type of white blood cell) are produced in the bone marrow.

Table 17(2).5 gives the distribution of total bone marrow space.

The percentage masses of different cell series present in the adult marrow are:

Erythrocyte series	3.3
Leukocyte series	
Granulocytic	30.0
Lymphocytic	3.3
Other haemic	6.6
Fat, blood, connective tissue	56.8

Only 4% of the total cells of the erythrocytic series are in the marrow, the rest being in circulating blood. Around 32% of the leukocytic series are in the marrow, the rest being outside both the blood and blood-forming organs (Hudson, 1965; Osgood, 1954, 1955).

In response to stimuli such as an increased blood destruction, blood loss, and anoxemia, the daily rate of RBC production may increase by a factor of 10 or more (Wintrobe, 1967). Extramedullary haematopoiesis, mainly in spleen and lymph nodes, may supplement red marrow haematopoiesis.

The turnover times of the erythrocytic series are 11-40 hours, and of the granulocytic series 20-140 hours, depending on the type of cell (Cronkite, 1960), and of the thrombocytic (platelet-forming) series 10-25 days (Schumacher and Erslev, 1965).

17(2).5.3 Lymphatic System

The lymphatic system is composed of lymphatic vessels, patches of lymphatic tissue, lymphatic organs containing lymph, and isolated lymphocytes. The lymphatic capillaries unite to form larger vessels (thoracic duct and right lymph duct) which empty into the veins. Lymphatic tissue is contained in the red marrow and in the lymphatic organs, namely, lymph nodes, spleen, thymus, mucous membranes, tonsils, adenoids, Peyer's patches, and in the vermiform appendix (Bloom and Fawcett, 1968; Gardner *et al.*, 1963; Yoffey, 1959).

The small lymphocyte is 6-8 μm in diameter and forms 20-25% of the total lymphocytes in the blood (Bloom and Fawcett, 1968). There are two categories of lymphocytes - one with a mean survival time of a few days, and another with a mean survival time of months to a year or more (Elves, 1972; Wintrobe, 1967). Lymphocytes with chromosomal defects induced by irradiation have a half-time of around 3 years.

A substantial fraction of the lymphocytes in the lymphatic structure is 'fixed', not reappearing in circulation. The remaining fraction recirculates between the blood, the tissue spaces, and the lymphatic system, with a cycle time of hours; the circulating lymphocytes cannot, however, re-enter the thymus or bone marrow.

17(2).5.4 Spleen

The spleen size varies greatly in different persons, and in the same individual under different conditions. It is oblong and flattened, lies posterior to the stomach, and is in contact with the left kidney. In the adult its length is 10-14 cm, width 6-10 cm, and thickness 3-4 cm (Anson,

1966; Goss, 1959; Martin and Saller, 1962; Pryce and Ross, 1963; Saphir, 1958; Stitt *et al.*, 1948).

17(2).5.5 Thymus

The thymus is a relatively large organ at birth and reaches a maximum size during adolescence. Soon after puberty, it undergoes retrogression or involution, so that it is scarcely recognizable in adults. Before undergoing retrogression, it plays a fundamental role in the immunological processes of the body (Anson, 1966; Gardner *et al.*, 1963; Goss, 1959).

The thymus can assume different shapes. It is, in general, bilobed, and each lobe is somewhat pyramidal, with the apex extending towards the lower neck and partly in the thorax, extending from about the fourth rib to the lower border of the thyroid (Anson, 1966; Goss, 1959).

Lymphocyte migration pathways involve the thymus, bone marrow and peripheral lymphoid organs.

17(2).6 SKELETAL MUSCLE SYSTEM

There are three varieties of muscle: (i) skeletal, voluntary, or striated; (ii) smooth, involuntary, non-striated, or visceral; and (iii) cardiac or heart. Here we shall deal only with skeletal muscle.

Muscle mass is about 22% of body weight for newborn infants (Anson, 1966). In the adult, 25-30% of skeletal muscle mass is in the head and trunk, 20% in upper extremities, and around 55% in lower extremities (Scammon, 1923).

Adult growth proceeds by enlargement of fibres and hypertrophy. The male exhibits a 14-fold and the female a 10-fold increase in the number of cells in the muscle during postnatal life; the cell size also shows an increase from infancy through adolescence and beyond (Cheek, 1968).

17(2).7 CARDIOVASCULAR SYSTEM

The cardiovascular system consists of the heart, arteries, veins, and capillaries.

17(2).7.1 Heart

The heart is a hollow muscular organ shaped like a blunt cone and contains four chambers - the right and left atrium, and the right and left ventricle. The large veins (superior and inferior vena cava) bring the venous blood from the body to the right atrium from which it passes to the right ventricle. From the right ventricle the blood is pumped through the pulmonary artery to the lungs where it is oxygenated. The blood is returned from the lungs through the pulmonary vein to the left atrium from where it passes to the left ventricle which pumps it into the aorta for distribution throughout the body.

The heart muscle is histologically different from smooth and skeletal muscle. The striated muscular fibres of the cardiac muscle are separate cellular units. The rhythmical contraction of the cardiac fibres is involuntary.

17(2).7.2 The Blood Pool

The cerebral blood pool comprises about 3% of the total blood volume, the thoracic blood pool 52%, and splanchnic blood pool (liver, spleen and mesenteric vessels) about 20% (Grollman *et al.*, 1959).

17(2).8 DIGESTIVE SYSTEM

The digestive system includes the mouth, tongue, salivary glands, pharynx, tonsillar ring, and gastrointestinal (GI) tract, as well as the liver, gall bladder, and pancreas. For purposes of dosimetry, the GI tract is further divided into the esophagus, stomach, small intestine, upper large intestine (cecum, ascending colon, and transverse colon), and the lower large intestine (descending colon, sigmoid colon, and rectum).

17(2).8.1 Mouth

In the mouth food is prepared for digestion, but the residence time is short. The cavity of the mouth is divided into an anterior vestibule or labial cavity (between the lips, cheeks, and teeth) and a posterior cavity or buccal cavity which lies within the dental arches and communicates with the oral pharynx (Hamilton, 1957).

17(2).8.2 Salivary Glands

For the most part, salivary secretion is provided by 3 pairs of glands: the parotid, the submaxillary, and sublingual. The parotid glands lie anterior and downward from the pinna of the ear, while the others are embedded in the tissues forming the lower jaws and the floor of the mouth.

17(2).8.3 Pharynx

The pharynx is a musculofibrous tubular passage somewhat conical in form with its base upward. It extends from the base of the skull to the esophagus at the level of the cartilage of the larynx anteriorly and to the level of the sixth cervical vertebra posteriorly. It is divided into 3 regions: (i) nasal pharynx, which is exclusively respiratory in function; (ii) oral pharynx, which is both respiratory and alimentary; and (iii) laryngeal pharynx, which is also respiratory and alimentary (Anson, 1966; Goss, 1959).

17(2).8.4 Tonsillar Ring

It consists of the two palatine or faucial tonsils (usually referred to as the tonsils) which are located on either side of the oral pharynx, the pharyngeal tonsil (adenoid) located in the posterior wall of the nasal pharynx, and the lingual tonsil located at the back of the tongue.

17(2).8.5 Esophagus

The esophagus is a muscular tube extending from the pharynx to the stomach, with a length of about 25 cm in the adult (Goss, 1959; Lerche,1950).

The lumina of the cervical and abdominal portions of the esophagus are closed except during the passage of food, but the thoracic part is more or less open, containing some air (Anson, 1966). The diameter at the level of the dilated segments in the adult is 16-22 mm (Brash and Jamieson, 1943; Lerche, 1950).

17(2).8.6 Stomach

The shape, size, and position of the stomach are highly variable, depending on the state of filling, degree of musculature contraction, pres-

ence or absence of peristaltic waves, respiratory cycle, position of body, etc. In general, it is J-shaped and lies obliquely in the upper left quadrant of the abdominal cavity and is directed caudally, or anteriorly, and to the right (Anson, 1966; Brash and Jamieson, 1943; Gardner *et al.*, 1963; Goss, 1959). Its capacity in the adult is around 1300-4800 ml (Eve, 1966; Martin and Saller, 1962; Vierordt, 1906).

17(2).8.7 Intestinal Tract

The small intestine is subdivided into the duodenum, jejunum, and ileum. The duodenum is well limited anatomically as being located between the stomach and jejunum. It is difficult to separate the jejunum and ileum anatomically.

The large intestine is subdivided into the upper large intestine, consisting of the cecum, ascending colon, and transverse colon, and the lower large intestine, consisting of descending colon, sigmoid colon, and rectum. The large intestine surrounds the small intestine, making a circuit around the abdominal cavity from right to left (Anson, 1966).

The surface area of the small intestine depends upon the method of computing it. The smooth surface area (SA) of the wall is around 3.3×10^3 cm^2 in the adult. The presence of villi, which are finger-like processes, results in a 10-fold increase in the surface over the value for the simple cylinder. The microvilli increase the SA by another factor of 20 over the SA based on the villi (Wilson, 1962).

The villus is 700 μm long and 130 μm in diameter. There are 9×10^7 villi in the entire intestine. The epithelial cells have dimensions of 5 x 5 x 40 μm and their total number is 7.2×10^{11} in the intestine. The total number of cells produced and lost per day is 2.4×10^{11} (Crosby, 1961).

The mucosa of the large intestine has a rather smooth surface except in the rectum where folds in the mucous membrane are found. The surface areas of the large intestine in the adult male and female are around 3460 and 3000 cm^2 respectively (Bloom and Fawcett, 1968; Martin and Saller, 1962).

The surface of the GI tract is formed by the mucosa which secretes mucus, a viscous fluid layer which overlays the surface of the tract. Thus a layer of nonviable material offers some shielding for radiation emitted by radionuclides present in the gut contents in insoluble form.

Epithelial cells are renewed rapidly in the intestinal tract. After the mitotic cycle has been completed, the cells may enter a number of different paths: (i) some cells undergo differentiation and maturation to various types; (ii) some cells may migrate in the course of a few days to the luminal surface of the mucosa to be sloughed; (iii) others remain in the crypts and pass through the proliferation or regeneration cycle or mitosis (Lipkin, 1965).

17(2).8.8 Liver

The liver is a large organ which lies immediately below the diaphragm and occupies the upper portion of the abdominal cavity, mainly on the right side. It is an irregular, 4-sided pyramid laid on one side (Brash and Jamieson, 1943).

The greatest length, width and height of the adult liver are 20-30 cm, 10-21 cm, and 7-15 cm respectively. (Hilding, 1961; Shennan, 1935; Stitt *et al.*, 1948; Testut, 1899, 1901).

17(2).8.9 Gallbladder

The gallbladder is a conical or pear-shaped sack-like organ of length around 10 cm and width 4-5 cm which normally stores and concentrates the bile which it receives by way of the hepatic and cystic ducts (Bloom and Fawcett, 1968; Goss, 1959; Martin and Saller, 1962; Oser, 1965; Testut, 1899, 1901). Bile is secreted by the liver and has several metabolic functions. Bile passes from the liver to the gallbladder where it is stored temporarily prior to discharge by contractions of the gallbladder into the duodenum. The gallbladder stores, concentrates, and alters the composition of bile (Oser, 1965).

17(2).8.10 Pancreas

The pancreas is an elongated gland extending transversely across the dorsal abdominal wall, dorsal to the stomach from the duodenum to the spleen (Anson, 1966). In the adult its length, width and thickness are 14-18 cm, 3-9 cm, and 2-3 cm, respectively (Martin and Saller, 1962). Pancreatic tissue includes acinous cells, duct cells, and two types (A and B) of islet cells. The A cells secrete glucagon and the B cells insulin; in the adult, the ratio of the number of A to B cells is about 4 (Bloom and Fawcett, 1968; Martin and Saller, 1962).

17(2).9 RESPIRATORY SYSTEM

Anatomically, the respiratory system includes the nose, larynx, trachea, and lungs. The ICRP lung model is described in Chapter 21.

17(2).9.1 Nose

The nose is a hollow structure composed of bone, cartilage, and muscle. The interior openings are lined with mucosa. The openings (nares or nostrils) are followed by an unciliated portion (vestibule) which leads to the inner nasal cavities (nasal fossae) which are ciliated and contain the turbinates. Because of the intrusion of the turbinates, the air passages of the nasal cavities consist of ribbon-like openings having complexly folded surfaces with a high surface-to-volume ratio. These passages are separated by the nasal septum.

In the adult, the openings of the naris or nostril are about 20 mm in length and 7-8 mm in width, but they vary considerably among individuals. The approximate dimensions of the nasal cavity in the adult are: length - 70 mm; greatest anteroposterior diameter 40 mm; floor width along passage 1-2 mm (Anson, 1966; Proctor, 1964; Testut and Jacob, 1905). The nasal septum is the partition separating the two nasal cavities in the midplane.

17(2).9.2 Larynx

The larynx is a tubular organ, the framework of which consists of cartilage and elastic membranes from which the vocal cords are formed. The inner surface is covered with mucous membrane continuous with that of the pharynx and trachea (Anson, 1966).

In the adult male, the distance from the tip of the epiglottis, which is a lid-like structure covering the larynx, to the trachea is 70 mm, and in the adult female 48 mm. The diameter of the adult larynx is 2.5 to 4.4 cm, being smaller for the female.

The mean length of the vocal cords in the relaxed and stretched states are 15 mm and 20 mm respectively for the adult male, and 11 mm and 15 mm respectively for the adult female (Anson, 1966; Martin and Saller, 1962).

The glottis is the sound-producing box apparatus which consists of the vocal folds (also called the true vocal cords) and the intervening space;

its diameter is around 23-28 mm and 17-20 mm in the adult male and female respectively (Anson, 1966; Testut, 1899, 1901).

17(2).9.3 Trachea

The trachea is an elastic tube whose walls are held apart by cartilaginous supports and which forms the continuation of the larynx and extends to the point of division into two bronchi (von Hayek, 1960).

The tracheal diameter changes during the respiratory cycle. Its dimensions in the adult are: length - 12 cm; anteroposterior diameter - 1.7 cm; transverse diameter - 1.5 cm (Scammon, 1923). The thickness of the tracheal wall is extremely variable; it is about 2 mm in the adult (Vierordt; 1906). It has around 20 cartilaginous rings which are thicker in males.

17(2).9.4 Lungs

On the basis of function, the lungs may be divided into two principal parts: the conductive part for carrying the air to and from the alveoli, and the respiratory part where oxygen is exchanged with carbon dioxide in the blood. The conducting part consists of the multibranching bronchial tree, while the respiratory part consists of respiratory bronchioles, alveolar ducts, alveolar sacs, atria, and alveoli. 'Parenchyma' is lung tissue with capillary blood, bronchial tree, and associated lymph nodes. The specific gravity of the adult lung containing air is 0.26 (Fowler and Young, 1959).

The right lung is shorter and broader than the left lung due to the position of the liver. The dimensions are given in Table 17(2).6.

Anatomical Volume of the Lungs

The term 'lung volume' refers to the total anatomical volume of the lung, which includes air, tissue, and blood. In the adult, the collapsed volume of lungs is around 995 ml, average total volume 3915 ml, and average maximal volume 6900-8000 ml (Fowler and Young, 1959; Weibel, 1963).

The total blood volume in both lungs is about 10% of the blood volume in the total body (Donato *et al.*, 1964; Fishman, 1963, 1966; Giuntini *et al.*, 1963; Sevelius and Creekmore, 1965).

17(2).9.5 Bronchial Tree

The Weibel (1963) symmetrical model of the bronchial tree involves a regular pattern of bifurcation. A summarized version of the Weibel model is given in Table 17(2).7. While the Weibel model may be reasonably adequate for deposition studies, Horsfield and Cumming (1968) have suggested that the bifurcation is not so regular, and that the number of bronchioles is less (by a factor of about 2) at the higher branchings. The total number of generations in their model is 27.

As per the Weibel model, the total surface area of the TB region is 6260 cm^2, mean wall thickness 81 μm, and tissue weight 51 g; the corresponding values in the Horsfield-Cumming model are: 4010 cm^2, 100 μm, and 40 g.

The wall thicknesses are around 2250 μm for the trachea, 1530 μm for the main bronchus, 530 μm at the end of the lobar bronchus, and 24 μm at the 16th generation in the terminal bronchus. In the adult, the average length and diameter of the right bronchus are around 22 mm and 16 mm respectively; the corresponding values for the left bronchus are 52 mm and 14 mm respectively (Jesseph and Merendino, 1957). The thickness of the epithelial lining of the bronchial tree is more in smokers (Chang, 1957).

17(2).9.6 Airways of the Pulmonary Region

The pulmonary region consists of the respiratory bronchioles, alveolar ducts, alveolar sacs, atria, and alveoli.

The respiratory bronchioles are a part of the respiratory part of the lungs. They are short tubes whose greatest diameter is about 0.51 mm not including the width of the alveoli, and the diameter decreases with each successive branching to 0.5 mm including the alveoli in the walls. They connect with the terminal bronchioles of the conductive system of the lungs. In this region, the lining is called columnar epithelium, but is devoid of goblet cells. Proceeding only a short distance, the epithelium becomes cuboidal and unciliated. A few alveoli bud from the respiratory bronchioles which accounts for the term 'respiratory bronchioles'. In length, the respiratory bronchioles range from 1 mm to 0.5 mm. Most of the bronchioles branch into alveolar ducts, in which the entire wall is occupied by alveoli. The alveolar sacs essentially are identical in structure with alveolar ducts, except that they are closed off by the terminal alveoli and, therefore, do not branch further. The diameter of the alveo-

lar ducts and sacs varies between 200 and 600 μm in adults. The length of the alveolar ducts and sacs is roughly 0.7 to 1 mm. The atria are spaces between the ends of the alveolar ducts and alveolar sacs (Bloom and Fawcett, 1968; Pump, 1964).

The alveoli are thin-walled sacs, one side of which is always lacking. The alveoli are small, thin-walled outpouchings of the alveolar ducts, alveolar sacs and respiratory bronchioles, and often have been compared to the cells of the honeycomb. The alveolar walls have a dense network of capillaries. The interchange of gases takes place in the alveoli (Bloom and Fawcett, 1968; Weibel, 1963). The diameter of the alveoli in the adult is 200-300 μm (von Hayek, 1960; Weibel, 1963). The alveoli are lined by a secretion that covers the epithelium with a layer about 0.1-0.3 μm in thickness and which has the property of lowering the surface tension. A phagocytic process tends to clear alveolar secretions and whatever particulate material that may be carried in it (Casarett and Milley, 1964; Divertie and Brown, 1964a, b; Gross, 1964; Pattle, 1961).

The surface area of the lungs is about 30 m^2 in expiration and about 3 times more during inspiration (von Hayek, 1960).

17(2).9.7 Pleura

The serous membranes surrounding the lungs and lining the thoracic cavity are called the pleura and consist of two sheets of fibroelastic tissue which are joined in the root of the lung. Some further quantitative values pertaining to the lung are given in Table 17(2).8.

17(2).10 URO-GENITAL SYSTEM

The uro-genital system includes both urinary organs and the reproductive organs.

17(2).10.1 Kidneys

Each kidney is somewhat bean-shaped; the right one is usually slightly lower than the left. The longitudinal axis is tilted ventrally, while the anteroposterior axis is directed downwards. A fibrous tunic, called capsule, forms a firm smooth covering of the kidney. The medulla consists of a series (variable from 8 to 18) of striated conical masses called renal pyramids. The bases of these pyramids are directed towards the circumference of the kidney. The cortex is composed of two portions: (i) a

peripheral layer immediately beneath the capsule and external to the base of the pyramids, and (ii) the renal columns which are located between the pyramids. The collecting system is composed of: (i) the nephron, which is concerned with the formation of urine, and extends from the outer cortex to the collecting tubules, and (ii) the collecting tubules, which serve as the excretory ducts conveying the urine to the renal pelvis (Anson, 1966; Bloom and Fawcett, 1968; Goss, 1959).

In the adult, the kidney length is 10-12 cm, transverse diameter 5-6 cm, anteroposterior diameter 3-4 cm, cortex width 0.4-1.3 cm, medulla width 1.6-1.9 cm, and capsule thickness 0.01-0.02 cm (Anson, 1966; Blau *et al.*, 1975; Vierordt, 1906).

17(2).10.2 Ureters

The ureters are two slightly flattened tubes through which the urine passes from the kidneys to the bladder. Their length is around 30 cm in the adult male, and about 29 cm in the adult female (White House Conference, 1933). Peristaltic waves moving at the rate of 3-4 cm/s propagate the urine in the ureter from the renal pelvis to the bladder. The urine enters the bladder in spurts 10-30 s apart (Anson, 1966; Houssay *et al.*, 1955).

17(2).10.3 Urinary Bladder

The bladder is a musculomembranous sac which acts as a reservoir for the urine (Goss, 1959). The dimensions of the bladder in the adult are (Vierordt, 1906):

Parameter	Contracted (cm)	Distended (cm)
Length	5-6	12-14
Transverse diameter	4-5	8-10
Anteroposterior diameter	2-2.5	8-10

The physiological capacity of the bladder is the amount of urine in the bladder when a sensation to void is noticed, without undue discomfort, and the capacity is the amount causing undue distress. The habit of urination has a direct bearing on the size of the bladder. If the individual urinates frequently, he may be uncomfortable retaining the average amount

of urine. There is probably no inherent difference between the male and female bladder (Anson, 1966; Best and Taylor, 1966).

When the adult bladder is empty, it is tetrahedron-shaped and lies almost within the pelvis. As the bladder fills, it gradually rises to the abdomen, becomes almost spherical, and may reach to the level of the umbilicus. It is situated slightly lower in the female than in the male (Anson, 1966; Gardner *et al.*, 1963).

17(2).10.4 Urethra

The urethra is a membranous canal conveying urine from the bladder to the surface. Its length is 14-21 cm in the adult male, and 2.5-4 cm in the adult female; the width of the urethral orifice is about 0.7 cm in both adult male and female (Anson, 1966; Bloom and Fawcett, 1968; Brock, 1954).

17(2).10.5 Testes

The testes are two ovoid organs in which the male gametes, the sperm, are formed. They are enclosed in the scrotum which is external to the main mass of the body. The dimensions of the adult testis are (Goss, 1959; Saphir, 1958; Testut, 1899, 1901) given below:

Vertical diameter, cm	4-5
Anteroposterior diameter, cm	2.5-3.5
Transverse diameter, cm	2-2.7

The duration of the whole spermatogenesis in man is about 64 days. The duration of one cycle of the germinal epithelium is about 16 days, and there are probably 4 cycles. The spermatozoa survival time in terms of fertility is 1.5-72 h, and in terms of mobility, 48-72 h. The total length of the spermatozoa is about 55 μm (head - 5.0 μm; midpiece - 4.5 μm; tail - 45.0 μm). The concentration of the sperm in semen is 50-150 (average 100) million/ml. The volume of ejaculate is 2-6 ml (average 3.5 ml). Of this volume, less than 10% is sperm and the remainder seminal plasma. The transit time of the sperm from the vagina to ovarian end of fallopian tube is about 3 h, and from the cervix to ovarian end of fallopian tube about 30 minutes (Altman and Dittmer, 1962; Bloom and Fawcett, 1968; Hamilton, 1957; Heller and Clermont, 1963; Heller *et al.*, 1964).

In the adult testis, the connective tissue is about 25% of the total volume of the testis. Each testis is divided into about 250 conical and

intercommunicating compartments of varying size; these are the lobules of the testis, and each lobe contains 1-3 convoluted seminiferous tubules in which the sperm are formed, and which are distributed throughout the testis except in an outer layer. The combined length of the seminiferous tubules in all lobules of one testis is 250-400 m (Bloom and Fawcett, 1968; Hamilton, 1957; Weiss, 1966).

17(2).10.6 Epididymes

The epididymes are oblong bodies located at the upper posterior region of each testis, where the sperm are stored. They consist of the surrounding tunic, entwining connective tissue, and a highly coiled tube (Bloom and Fawcett, 1968).

17(2).10.7 Prostate Gland

The prostate gland is an accessory of the male reproductive system (Anson, 1966; Gardner *et al.*, 1963; Goss, 1959). It is composed of both glandular and muscular tissue. It is a flattened conical structure (of maximum diameter around 3.8 cm) located in the pelvis and is anterior to the rectum. It surrounds the neck of the bladder and the urethra. After the age of 40 years, it frequently increases in weight. The greatest vertical, transverse (or base), and anteroposterior diameters of the prostate in the adult are 3.2 cm, 3.8 cm and 2.7 cm respectively (Teem, 1936).

17(2).10.8 Seminal Vesicles

The seminal vesicles are two sacculated pouches or lobulated organs symmetrically placed on either side of the midline on the caudal portion of the posterior surface of the bladder. Each vesicle is about 5 cm in length and has a capacity of 1.5-3 ml. They produce a large part of the seminal fluid (Gardner *et al.*, 1963; Hamilton, 1957).

17(2).10.9 Ovaries

The ovaries are paired organs located on each side of the pelvic cavity. They become displaced during the first pregnancy (Anson, 1966; Goss, 1959).

The ovarian cells may be classified as somatic or germinal; the latter are subclassified as normal or atretic (degenerating). At birth there are

about 2 million germ cells in both ovaries, of which 50% are atretic. At 6 months, only one-third of the germ cells are present and by the seventh year about 15%. Since most degenerate within the ovary, only about 400 are ovulated during the reproductive lifetime (Baker, 1963; Barnes, 1968; Best and Taylor, 1966; Krohn, 1967).

In contrast to the male germ cells, the female germ cells do not divide any more after birth, except for the last two meiotic divisions leading to the primary and secondary oocytes.

The diameter of the ovum is about 0.09 mm, and it is viable for about 24 hours. The time of transport from the Fallopian tube to uterus is 3 days, and the time of ovulation about 14 days prior to the next menstrual period (Altman and Dittmer, 1962).

17(2).10.10 Fallopian Tubes

The Fallopian tubes or uterine tubes or oviducts convey the ova from the ovary to the uterus. In the adult, its length is about 10.7 cm, and the diameter of the lumen 2.5 mm (Anson, 1966; Boyd, 1941; Scammon, 1930; Stitt *et al.*, 1948).

17(2).10.11 Uterus

The uterus is the organ in which the fertilized ovum normally becomes embedded and in which the developing organism grows and is nourished until its birth. Its length in the nulliparous adult is 6-7.5 cm, and the transverse diameters at the fundus and isthmus are, respectively, 4-5.5 cm and 1.5-3 cm; the anteroposterior thickness varies from 1.5-2.5 cm at the isthmus to 2.2-3 cm at the fundus. (The fundus is the base or region farthest from the mouth of the uterus, and the isthmus is about midway between the base and the mouth.) The dimensions are 1 cm or more greater in the multiparous adult (Martin and Saller, 1962; Pryce and Ross, 1963).

In the adult, the uterus is pear-shaped. Its position is influenced by the position of the bladder and rectum. When both are empty, the body of the uterus is nearly horizontal when the individual is standing; as the bladder fills, the uterus is bent backward toward the sacral vertebrae. During menstruation, it is enlarged and more vascular. In old age, it becomes atrophied (Anson, 1966; Gardner *et al.*, 1963; Goss, 1959).

The thickness of the endometrium, which is the mucous membrane lining the uterus is 0.3-1 mm, and increases to 5-7 mm at the beginning of menstruation (Anson, 1966; Vierordt, 1906).

The mean age at menopause is 47-50 years.

17(2).10.12 Vagina

The vagina is a muscular, highly dilatable canal in the female which extends from the uterus to the external genitalia where it opens to the exterior. Its length is 5.5-7.5 cm at the anterior wall, and 7-9 cm at the posterior wall, width around 3 cm, wall thickness 2 mm, and mucosal thickness 0.15-0.2 mm (Goss, 1959; Vierordt, 1906).

17(2).10.13 Breast

The diameter of the breast before lactation is 10-13 cm. The breast protrudes 5-6 cm from the chest wall. During lactation the weight may increase to 560-1080 g (Anson, 1966; Goss, 1959; Hytten and Leitch, 1964; Stitt *et al.*, 1948; Testut, 1899, 1901; Vierordt, 1906).

17(2).11 ENDOCRINE SYSTEM

17(2).11.1 Thyroid Gland

The thyroid is a bilobed gland connected by an isthmus. From the front it is roughly H-shaped. The two lateral lobes are located on either side of the upper trachea and lower larynx. In some cases the lobes may be of different lengths. The isthmus lies ventral to the upper trachea and varies in size in different individuals. In some it is only a thin band, while in others it is a broad and relatively thick band (Anson, 1966; Gardner *et al.*, 1963).

The weight of the thyroid gland is highly variable, depending upon age, sex, diet, geography, climate, external and internal stimuli, but perhaps the most important factor is iodine intake. Both thyroidal weight and thyroidal iodine uptake appear to be correlated inversely with daily iodine intake (Bell *et al.*, 1968; Dolphin, 1971; Martin and Saller, 1962). ICRP data are mainly based on US populations and iodine intake in USA is generally high. In prenatal life, the follicles (which are the fundamental units of the thyroid gland) appear at about 40 days of gestation. Soon

they develop lumina and by 70 days the colloid appears. Colloid is gelatinous and is the stored product of secretory activity of the follicle. Colloid contains thyroxine (T_4) and triiodothyronine (T_3), which, when released into the blood, constitute the thyroid hormones. By 100 days the production of new follicles stops, and an increase in the size of the gland results from the growth of the follicles already present (DeSmet, 1960).

The weight of the thyroid is around 5-20 mg at gestational age of 84 days, increasing to 150-240 mg at 140 days, 540 mg at 180 days, and 1 g at birth (Evans *et al.*, 1967; Mochizuki *et al.*, 1963).

If a deficiency of iodine in the diet exists, it may lead to difficulties in the synthesis of thyroid hormones, which is manifested by an enlargement of the gland. In geographical areas where such a deficiency exists (endemic goitre areas), a large part of the population may be goitrous (Bell *et al.*, 1968). The adult thyroid in endemic areas may weigh 30-80 g (DeSmet, 1960; Martin and Saller, 1962).

Dimensions of the adult thyroid gland are as follows (Anson, 1966; DeSmet, 1960; Means *et al.*, 1963; Pryce and Ross, 1963; Testut, 1899, 1901; USAF Institute of Pathology, 1960):

	Transverse diameter (cm)	Vertical diameter (cm)	Anteroposterior diameter (cm)
Each lobe	2-4	5-8	1-2.5
Isthmus	2	2	0.2-0.6

17(2).11.1.1 Follicles of the Thyroid Gland

Each lobule consists of 20-40 follicles which are the structural and secretory units of the thyroid. Thyroid hormone is synthesized and stored in the follicular cells. The epithelial lining is one cell deep, the cells being cuboidal in shape and about 15 μm in height. Upon stimulation, the cells become columnar, but return to the cuboidal shape in the resting gland. The average follicular diameter in the adult is about 300 μm (DeSmet, 1960). The thyroid has an abundant blood supply. The thickness of tissue overlying the thyroid in the adult is 1-2 cm (van Dilla and Fulwyler, 1963).

17(2).11.2 Parathyroid Gland

The average number of parathyroid glands is four, but the range is from 1-12 (Anson, 1966). The parathyroid is a somewhat flattened ovoid body with the following diameters in the adult: vertical - 4 to 15 mm; transverse - 3 to 4 mm; anteroposterior - 1.5 to 2 mm (Anson, 1966; Pryce and Ross, 1963; Stitt *et al.*, 1948; Testut, 1899, 1901).

17(2).11.3 Adrenal Glands

The adrenal (or suprarenal) glands are two in number and are usually located above the kidney. Each consists of two divisions, with different functions - the cortex and the medulla (Gardner *et al.*, 1963). Their shape differs from one individual to another as well as from the left one to the right one; the right is more triangular and flattened, while the left is nearly semi-lunar (Anson, 1966). In the adult, the transverse, vertical and anteroposterior diameters of the adrenal are 3-7 cm, 2-3.5 cm, and 0.3-0.8 cm respectively (Anson, 1966; Martin and Saller, 1962; Pryce and Ross, 1963; Saphir, 1958; Stitt *et al.*, 1948; Testut, 1899, 1901; Vierordt, 1906).

17(2).11.4 Pineal Gland (Epiphysis)

The pineal gland is an unpaired ovoid or cone-shaped gland situated in the midbrain above the cerebellum but on the caudal surface of the cerebral hemisphere. In the adult its anteroposterior, vertical, and transverse diameters are 0.5-0.9 cm, 0.3-0.6 cm, and 0.2-0.4 cm respectively (Anson, 1966).

17(2).11.5 Pituitary Gland (Hypophysis)

The pituitary gland has an oval or flattened spheroidal shape and is situated anteriorly to the spinal cord where it joins on to the brain (Anson, 1966). In the adult, its sagittal, vertical and transverse diameters are 5-15 mm, 5-9.7 mm, and 10.5-17 mm respectively (Martin and Saller, 1962). The pituitary is divided into two major subdivisions on the basis of both morphology and function: (i) adenohypophysis or glandular lobe, and (ii) neurohypophysis.

17(2).12 CENTRAL NERVOUS SYSTEM

The central nervous system (CNS) is a continuous structure which is divided into two parts - the brain (or encephalon) and the spinal cord (or medulla spinalis). The CNS is surrounded by 3 membranes or meninges - the dura mater, the arachnoid, and the pia mater. The CNS is composed of many millions of nerve cells called neurons which form the conduction pathways and which are held together by a framework of specialized non-conducting cells known as neuroglia. The lifespan of the neuron is coextensive with normal function and replacement is confined to neuroglia. Neurons are dying continually and being lost in the adult, but cell renewal does not occur (Altman and Dittmer, 1964; Brody, 1955). No lymphatic vessels are found in the CNS (Mayerson, 1962).

Two types of tissue - the white and gray matter - are distinguished in the CNS. In the brain, the gray matter is on the surface, and referred to as the cortex, while the white is in the interior. In the spinal cord, the positions of the gray and white matter are reversed. The gray matter consists largely of bodies of nerve cells, whereas the white matter consists largely of the processes or fibres of the nerve cells.

17(2).12.1 Brain

The brain is that part of the CNS which is enclosed within the skull. It includes the cerebrum, cerebellum, and brain stem with the meninges. The cerebrum is the large oblong structure occupying most of the cranial cavity. It is divided into the right and left cerebral hemispheres by a deep median sagittal groove.The cerebellum lies between the brain stem and the cerebrum; it is posterior and under the cerebrum. The brain stem consists of the diencephalon or interbrain, midbrain, pons, and medulla oblongata (Gardner *et al.*, 1963). The weight of the adult meninges is about 65 g (Anderson, 1966).

In the adult, the cerebrum occupies 85-88% of the weight of the whole brain, the cerebellum 10-12%, and the brain stem 1.9-2.3% (Anderson, 1966; Anson, 1966; Brock, 1954; Dunn, 1921; Hesdorffer and Scammon, 1936; Larroche, 1966; Scammon, 1923; Scammon and Dunn, 1924; White House Conference, 1933).

The adult brain without the brain stem is approximately half an ellipsoid. In the adult male its vertical, transverse, and anteroposterior diameters are 13 cm, 14 cm, and 16.5 cm respectively (Anson, 1966; Saphir, 1958; Stitt *et al.*, 1948; Testut, 1899, 1901; USAF Institute of Pathology,

1960). The dimensions for the female are about 1 cm less for each of the diameters. The blood in equilibrium with the adult brain is 504 g (Cowles *et al.*, 1971). The relative weight of gray matter in the adult cerebrum is 48.8% (Hoedt-Rasmussen and Skinhøj, 1966).

17(2).12.2 Spinal Cord

The spinal cord is an elongated, nearly cylindrical structure of the CNS located within the vertebral canal. It connects with the medulla oblongata which is part of the brain stem. The volume of the adult spinal cord is 28.1 ml (white matter 23.1 ml; gray mater 5.0 ml) (Lassek and Rasmussen, 1938). Its mean length in the adult male is 45 cm and about 2 cm less in the adult female (Goss, 1959). The cerebrospinal fluid (CSF) is renewed 6-8 times a day (Oser, 1965). The weight of the adult meninges is about 65 g (Anderson, 1966).

17(2).13 SPECIAL SENSE ORGANS

17(2).13.1 Eye

The eyeball is spheroidal in shape with a diameter in the adult of about 24 mm (Kronfeld, 1962; Scammon and Armstrong, 1925). The volume of blood present in the eye at any one time is around 211 mm^3 (Adler, 1959).

17(2).13.1.1 Conjunctiva

The conjunctiva is the mucosal membrane that lines the inner surface of the eyelids and covers the eyeball in front. Its thickness is 0.05-0.1 mm. For one eye, the total area of the adult conjunctiva is 16 cm^2. (Anson, 1966; Ehlers, 1965; Goss, 1959).

17(2).13.1.2 Cornea

The cornea is the transparent structure forming the anterior part of the external tunic of the eye which permits rays of light to enter. It is shaped like a segment of a sphere. In the adult, its weight is about 180 mg, and its diameter 11.6 mm. Its mean radius of curvature in the adult is 7.9 mm, peripheral thickness around 0.7 mm, and central thickness 0.5 mm. It is divided into 5 layers (Best and Taylor, 1966; Bloom and Fawcett,

1968; Martola and Baum, 1968; Maurice, 1962; Seefelder, 1938; Stenstrom, 1946).

17(2).13.1.3 Iris

The iris is the circular pigmented membrane behind the cornea which is perforated by the pupil. In the adult its diameter is 12 mm and thickness 0.5 mm (Anson, 1966).

17(2).13.1.4 Aqueous Humour

The aqueous humour is a clear, watery fluid occupying the anterior and posterior chambers of the eye. Its volume is 0.15-0.35 ml and its half-life 45 minutes (Altman and Dittmer, 1961; Best and Taylor, 1966; Davson, 1962; Long *et al.*, 1961).

17(2).13.1.5 Anterior and Posterior Chambers of the Eye

'The anterior chamber of the eye is bounded by the posterior surface of the cornea, tiny portions of the inner surface of the sclera, a variable portion of the anterior surface of the ciliary body, the entire anterior surface of the iris, and the intrapupillary portion of the anterior surface of the crystalline lens.' (Kronfeld, 1962). In the adult its volume is about 0.2 ml and its axial depth about 3.5 mm (Adler, 1959; Kronfeld, 1962; Rosengren, 1950; Stenstrom, 1946). The posterior chamber of the eye contains 14-20% of the total volume of aqueous humour (Davson, 1962).

17(2).13.1.6 Vitreous Humour

The vitreous humour is a transparent jelly filling the space between the lens and the retina. Its volume is about 3.9 ml (Altman and Dittmer, 1961; Anson, 1966; Long *et al.*, 1961).

17(2).13.1.7 Lens

The lens is a transparent, biconvex, semi-solid body in the eye and is devoid of blood supply. The transparency of the lens depends on the physicochemical state of the proteins of the lens. The anterior surface is termed the anterior pole, while the posterior surface is called the posterior pole. The junction of the two poles defines the equator of the lens. The

lens consists of a mass of transparent cells and lens fibres, which are enclosed in an elastic membrane - the lens capsule. The central portion of the lens, called the nucleus, gradually becomes denser and harder than the peripheral portions (Adler, 1959; Anson, 1966; Bloom and Fawcett, 1962; Heyningen, 1962; Kronfeld, 1962).

The volume of the lens increases with increasing age throughout life; in the adult, it increases from 163 ml at age 25 years to 240 ml at age 85 years (Scammon and Hesdorffer, 1937). The thickness of the front lens capsule is around 0.015 mm and that of the back lens capsule 0.006 mm. The cortex of the lens increases from 0.4 mm at 40 years to 0.8 mm at 80 years of age. The transverse diameter of the adult lens is 9-10 mm, and the anteroposterior diameter 3.7-5 mm. The nucleus of the lens is the central region and has a mean thickness of 2.8 mm. (Anson, 1966; Best and Taylor, 1966; Bloom and Fawcett, 1968; Hamilton, 1957; Huggert, 1946; Seefelder, 1938; Vierordt, 1906).

17(2).13.1.8 Cataract

Any partial or complete opacity of the lens is called a cataract. The essential change in the cataractous lens is a progressive coagulation of the lens proteins. Two stages are recognized in the coagulation process: (i) denaturation of the lens proteins which consists presumably of a molecular rearrangement, and (ii) aggregation of the protein particles into a flocculent mass. The germinal area of the lens epithelium with its relatively high mitotic index seems to be a principal area of involvement in the production of cataract by ionizing radiation. It is located 3-4 mm below the surface of the eye in the adult. It appears that the upper eyelid provides some additional shielding to the upper portion of the germinal layer (Cogan, 1958; Ham, 1953, 1969; Lerman, 1962).

The lens does not have any blood supply and is located at least 2 mm from any blood supply. It relies upon the aqueous and vitreous humours, especially upon the aqueous humour, for its nutrients and removal of waste products (Heyningen, 1962).

17(2).13.1.9 Sclera

The sclera is one part of the external protective covering of the eye. The front part of the sclera is commonly called 'the white of the eye'. Its weight in the adult is 1.0 to 1.3 g. The wall thickness is around 1 mm at the posterior pole, 0.3-0.4 mm at the equator, and about 0.6 mm at the

edge of the cornea (Bloom and Fawcett, 1968; Gardner *et al.*, 1963; Testut, 1899, 1901).

17(2).13.2 Ear

The external acoustic meatus is the narrow passage of the external ear which leads to the tympanic membrane (eardrum). In the adult, its length is 25-31 mm. (Anson, 1966; Gardner *et al.*, 1963).

17(2).13.2.1 Tympanic Membrane (Eardrum)

Its shape is elliptic. In the adult, the long axis is 9-11 mm long, and the short axis 8-9 mm. Its thickness is 0.015 to 0.1 mm, and surface area 63 mm^2 (Anson, 1966; Beksey, 1949; Brock, 1954; Gardner *et al.*, 1963).

17(2).14 PREGNANCY

17(2).14.1 Duration of Pregnancy or Time of Gestation

A typical duration of pregnancy or time of gestation is considered as 280 days, or 40 weeks, or 10 lunar months. The time of gestation is estimated by (i) size of uterus; (ii) the first appearance of heart sounds (about 20 weeks), or (iii) the time since the last menstrual period.

17(2).14.2 Components in Gained Weight during Pregnancy

The average total weight gain for primagravidae (first pregnancy) is 12.5 kg. This is made up as follows: Fetus - 26.4%; placenta - 5.2%; amniotic fluid - 6.4%; breasts - 3.2%; uterus - 7.2%; blood - 10.0%; extracellular water - 9.6%; not accounted for - 32.0%. Total body water increases by 7000 ml, out of which 74% is extracellular water and 26% intracellular water. The mean weight increase for multigravidae (3 or more pregnancies) is probably 900 g less than for primagravidae. (Hytten and Leitch, 1964).

During the first half of pregnancy, the uterine wall thickness averages 8-9 mm, but during the second half, due to the increase in the size of the uterus, it becomes thinner, so that at term the average thickness is under 6 mm. The rate of blood flow in the uterus at 10 weeks of pregnancy is

52 ml/min, at 28 weeks 185 ml/min, and at term 500-700 ml/min. The amount of blood in the vessels of the gravid uterus is about 10% of its weight (Hytten and Leitch, 1964).

17(2).14.3 Placenta

The placenta is the highly specialized organ by means of which the fetus makes its functional contact with the uterine wall. After the birth of the child, the placenta and membranes are expelled from the uterus. The average weight of the full term placenta without the membranes and the cords is 420 g. Its thickness is 1.6 mm and diameter 15-20 cm. Its blood content is about 250 ml at term. The mean minimum thickness of the maternal-fetal barrier is about 3.5 μm (Abramson, 1962; Aherne and Dunnill, 1966; Goss, 1959; Younoszai and Haworth, 1969a,b).

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Table 17(2).1A: Reference adult male and female: summary of anatomical values - weights of organs and tissues
(from ICRP 23)

Organ/tissue	Weight	
	Male (g)	Female (g)
Adipose tissue	15,000	19,000
Subcutaneous	7,000	13,000
Separable	5,000	4,000
Yellow marrow	1,500	1,300
Interstitial	1,000	700
Adrenal glands (2)	14	14
Blood (total)	5,500	4,100
Red blood cells	2,400	1,500
Plasma	3,100	2,600
Body (total)	70,000	58,000
Bone marrow (total)	3,000	2,600
Red bone marrow	1,500	1,300
Yellow bone marrow	1,500	1,300
Brain	1,500	1,200
Breasts (2)	26	360
Bronchial tree	30	25*
Cartilage (skeletal)	1,100	900*
Connective tissue	5,050	4,100
Cartilage	2,500	2,000
Tendons and fascia	850	700
Other	1,700	1,400
Esophagus	40	34
Eyes (2)	15	15
Fat (total)	13,500	16,000
Nonessential	12,000	15,000
Essential	1,500	1,000
Fingernails and toenails	3	3
Gallbladder	10	8*
GI tract (without contents)	1,200	1,100
Esophagus	40	34
Stomach	150	140
Small intestine	640	600
Duodenum	60	60
Jejeunum	280	250
Ileum	300	290
Large intestine	370	360
Upper	210	200
Ascending	90	90

(contd ...)

Organ/tissue	Weight	
	Male (g)	Female (g)
Transverse	120	110
Lower	160	160
Descending	90	90
Sigmoid colon and rectum	70	70
GI tract contents	1,005	-
Stomach	250	-
Small intestine	400	-
Upper large intestine	220	-
Lower large intestine	135	-
Hair	20	300
Heart (without blood)	330	240
Heart (at end of systole)	570	470*
Heart (at end of diastole)	425	350*
Hypodermis	7,500	13,000
Kidneys (2)	310	275
Larynx	28	19
Lenses (2)	0.40	0.40
Liver	1,800	1,400
Lung tissue (plus arterial and venous blood)	1,000	800
Pulmonary blood	530	430
Arterial	200	160
Venous	230	190
Capillary	100	80
Lung tissue or parenchyma without capillary blood and bronchial tree but with associated lymph nodes	440	360
Lymphocytes	1,500	1,200
Lymphatic tissue ('fixed')	700	580*
Muscle (skeletal)	28,000	17,000
Palatine tonsils (2)	4	4
Pancreas	100	85
Parathyroid glands (4)	0.12	0.14
Periarticular tissue (total)	1,500	1,200*
Pineal gland	0.18	0.15
Pituitary gland	0.60	0.70
Prostate gland	16	-
Pulmonary blood	530	-
Salivary glands (3 prs.)	85	70*
Parotid (2)	50	-

(contd ...)

Organ/tissue	Weight Male (g)	Female (g)
Submaxillary (2)	25	-
Sublingual (2)	10	-
Skeleton (wet weight)	10,000	6,800
Wet weight, fat-free	8,000	5,800
Bone (dry weight)	5,000	3,400
Bone, cortical	4,000	-
Bone, trabecular	1,000	-
Marrow	3,000	-
Other tissue	2,000	-
Skin (total)	2,600	1,790
Epidermis	30	28
Dermis	2,500	1,700
Spinal cord	30	28
Spleen	180	150
Stomach	150	140
Superficial fascia	500	400*
Teeth	46	41
Tendons and deep fascia	850	700*
Testes (2)	35	-
Thymus	20	20
Thyroid	20	17*
Tongue	4	4
Tonsils (2 palatine)	10	8*
Trachea	10	8*
Ureters (2)	16	15
Urethra	10	3
Urinary bladder	45	45

* Estimated on the basis of ratio of total body weights

Table 17(2).1B: Reference adult male and female: summary of other anatomical values
(from ICRP 23)

Anatomical value	Male	Female
Alveoli, surface area, m^2	75	66*
Blood (distribution of in total body)		
Arterial system, ml	1,000	750
Venous system, ml	3,200	2,400
Pulmonary system, ml	500	400
Heart cavity (av. value), ml	500	350
Blood volume (total), ml	5,200	3,900
Volume of red blood cells, ml	2,200	1,350
Volume of plasma, ml	3,000	2,500
Body length, cm	170	160
Body water (total), ml/kg W	600	500
Extracellular water, ml/kg W	260	200
Intracellular water, ml/kg W	340	300
Bronchial tree,		
Mean wall thickness, μm	75	70*
Bronchial tree, surface area, cm^2	3,950	3,500*
Cerebrospinal fluid volume, ml	120	100
Hypodermis thickness, μm	3,750	6,600
Intestinal tract length, cm	660	-
Small intestine, cm	500	-
Duodenum, cm	25	-
Jejeunum, cm	190	-
Ileum, cm	285	-
Large intestine, cm	160	-
Upper large intestine, cm	75	-
Cecum, cm	7	-
Ascending colon, cm	18	-
Transverse colon, cm	50	-
Lower large intestine, cm	85	-
Descending colon, cm	30	-
Sigmoid colon, cm	40	-
Rectum, cm	15	-
Lens (depth and size)		
Anterior aspect of lens to anterior pole of cornea, cm	0.3-0.4	0.3-0.4
Anterior aspect of lens to anterior aspect of closed lid, cm	0.8	0.8
Equator of lens to anterior of corneal border, cm	0.3	0.8
Equatorial diameter of lens, cm	0.9	0.9
Axial thickness of lens, cm	0.4	0.4

(contd ...)

Anatomical Value	Male	Female
Nose (specifications)		
Surface area of both vestibules, cm^2	21	18
Surface area of turbinates and nasal passages, cm^2	160	135
Thickness of mucus, mm	0.5	0.5
Thickness of epithelium, mm	0.1	0.1
Thickness of entire mucosa, mm	2	2
Weight of mucosa, g	32	27
Skin, total thickness, μm	1,300	1,300
Epidermis, μm	50	50
Dermis, μm	1,250	1,250
Specific gravity of total body	1.07	1.04
Surface area of body, m^2	1.8	1.6
Urinary bladder capacity		
Capacity (distress), ml	500	500
Physiological capacity, ml	200	200
Contents of urinary bladder (moderately distended), ml	100	-

* Estimated on the basis of ratio of total body weights

Table 17(2).2: Composition of total adult body

[from ICRP 23, based on Brožek *et al.*(1963); Forbes *et al.* (1953, 1956); Mitchell *et al.* (1945); Vierordt (1906); Widdowson *et al.* (1951).]

Component	Percent of body mass
Water	60
Protein	15-20
Fat	19
Carbohydrate	0.6
Mineral	5.8
Ash	4.8-5.8
Blood	7.9

Table 17(2).3: Distribution of total body water
[from ICRP 23, based on Edelman and Leibman (1959); Epstein and Maibach (1965); Ruch and Patton (1965).]

Compartment	% of TBW
A. *Extracellular Water*	
Plasma	7
Interstitial lymph	20
Dense connective tissue and cartilage	7
Inaccessible bone water	7
Transcellular	2.5
Total ECW	43
B. *Intracellular Water*	
Muscle cell	38
Bone cell	4
Connective cell	4
Erythrocytes	5
Other tissues	6
Total ICW	57

Table 17(2).4: Composition and other characteristics of blood (adults)
[from ICRP 23, based on Altman and Dittmer (1961, 1964); Bishop and Surgenor (1964); Leeson and Leeson (1966); Shock (1966); Smith (1959); Tipton (1966); Wintrobe (1967).]

Parameter	Value	
pH of whole blood	7.39	
RBCs, million/mm^3	5.4	(male)
	4.8	(female)
RBCs, mean survival time, days	126	
RBC's, rate of production, million per kg W per day	3500	(male)
	2500	(female)
Surface area of RBCs, m^2	3500	
Haemoglobin, g/100 ml blood	16.5	(male)
	14.5	(female)
Leukocytes (white blood cells) total, per mm^3	7000	(5000-10,000)
Differential leucocyte count, %		
Myelocytes	0	
Juvenile neutrophils	7.9	
Segmented neutrophils	47	
Eosinophils	3	
Basophils	0.6	
Lymphocytes	35	
Monocytes	6.5	
Platelets, per mm^3	250,000	
Survival time of platelets, days	8-11	
Composition of plasma		
Water, %	94	
Proteins, g/100 ml	6.5-7.2	
Albumin, g/100 ml	4.5	
Glucose, mg/100 ml	90	
Total lipids, mg %	735	
Ash, % wet weight	0.95	

Table 17(2).5: Distribution of total bone marrow space in adult bone
[Condensed from ICRP 23, based on Mechanik (1926) and Woodard and Holodny (1960).]

Bone	% of total marrow space
Femura	17.1
Pelvia	16.3
Tibiae	10.9
Ankles and feet	8.4
Dorsal vertebrae	7.3
Ribs	7.3
Cranium	6.3
Humerii	6.0
Lumbar vertebrae	5.6
Others	14.8

Table 17(2).6: Dimensions of the lung
[from ICRP23; based on Miller (1950); Vierordt, (1906).]

	Adult male		Adult female	
	Right	Left	Right	Left
Sagittal diameter or length, cm	27	30	22	23
Transverse diameter at the base, cm	14	13	12	11
Anteroposterior diameter, cm	20	18	18	16

Table 17(2).7: Summary of Weibel model of the tracheobronchial region
[adapted from ICRP 23; based on Weibel, (1963).]

Anatomical structure	Generation (down)	No. in final generation	Mean diameter in final generation (cm)	Mean length in final generation (cm)
Trachea	0	1	1.8	12.0
Main bronchus	1	2	1.22	4.76
Lobar bronchus	2-3	8	0.56	0.76
Seg. bronchus	5-10	1,024	0.130	0.46
Terminal bronchus	11-16	65,536	0.060	0.165

Table 17(2).8: Some further quantitative values for the adult lung
[adapted from ICRP 23; based on Dunnill (1962).]

Lung volume, ml	5500
Parenchymal volume, ml	4950
Alveolar air volume, ml	2945
Parenchymal tissue volume, ml	535
Capillary volume, ml	35
No. of alveoli, x 10^6	296
No. of respiratory airways, x 10^6	14
Generation of airways	23
Air-tissue interface, m^2	75

CHAPTER 17: SECTION 3

GROSS AND ELEMENTAL CONTENT OF REFERENCE MAN

Tables are presented in ICRP 23, Chapter 2, of the values for the physical properties (weight, specific gravity), gross content (water, ash, fat, protein), blood content and elemental content of a total of 114 constituents of the human body including total body, various organs (such as kidney, liver and skeleton), tissues (such as adipose tissue and blood), and components (such as body water and body fat) of the Reference Man. In the tables, asterisked tissues and organs are considered to make up the totality of the Reference Man and to contain within them the components; the sum of the asterisked weights is 70 kg. Values are generally for 'normal' (i.e. not diseased) tissue. Values for the more common trace elements are based on analysis of 150 adult victims of accidental death. Data on physical properties are mainly drawn from ICRP 23, Chapter 1. The weight in grams of any constituent has been calculated by multiplying its concentration in mg/g of wet tissue by the weight in grams of the organ or tissue. The single values for the parameters have been chosen/calculated from the median values of the range of values discussed in ICRP 23, Chapter 1, and, where available, the 80% range (10th and 90th quartiles) of values is also included. The values have generally been rounded to two significant figures.

A detailed table in ICRP 23, Chapter 2 (not reproduced in this Handbook) gives values for the weight, total quantity of blood (almost completely based on animal data), 'residual blood' (i.e. the quantity which remains in a tissue or organ after it has been allowed to bleed freely; equivalent to 'capillary blood'), the quantity of water, ash (mineral content), fat, protein, and specific gravity of each of the 114 organs/tissues/components mentioned earlier.

The conventions and meanings adopted by ICRP 23 in arriving at values for the different parameters are briefly explained in the following. The weight of each organ includes the blood vessels, lymphoid, adipose and connective tissue, and blood. Adipose tissue is a kind of connective tissue which includes hypodermis or subcutaneous adipose, the adipose that surrounds organs like the kidney and intestines which may be readily

separated from the organ at dissection, the adipose (interstitial) which occurs interspersed among the cells of an organ so intimately that it would be included with the organ at dissection, and yellow marrow. Body fat is a component, not a tissue or an organ. It is considered to be the ether-soluble extract of tissues. Essential fat includes the lipid constituent of cells, while non-essential fat represents the fat contained in adipose tissue. Body water is also a component, not a tissue. Extracellular water is associated with plasma, lymph, cartilage, connective tissue, bone and secretory cells; intracellular water is within the cells of the body. Cartilage is considered as part of the skeletal system. Connective tissue includes tendons and fascia, periarticular tissue (closely associated with bones, especially at their joints), and some other types. The weight of the lung includes all of the pulmonary tissue below the bifurcation of the trachea plus the pulmonary blood. 'Lung tissue' includes the bronchial tree, pulmonary lymph nodes, and capillary blood as well as the lung parenchyma; this is essentially the tissue that makes up lungs removed at autopsy, since the venous and arterial blood would be lost at dissection.

Table 17(3).1 (Bronner, 1964; Fruton and Simmons, 1959; Hawk *et al.*, 1947) gives the elemental composition (carbon, hydrogen, oxygen, nitrogen) of the body constituents (water, protein, fat, and carbohydrates); also included are the percentage of carbon and oxygen in bone mineral. Another table in ICRP 23, Chapter 2 (not reproduced in this Handbook) contains the values for the elemental content in grams of 51 elements in the 114 constituents. Median values, as well as, where available, 80% range of values are given.

Table 17(3).2 gives the total body content for 36 elements in Reference Man; it includes only those elements for which the concentration was known in at least 50% of the body, including the skeleton.

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Table 17(3).1: Elemental composition of body constituents
[from ICRP 23; based on Bronner (1964); Fruton and Simmons (1959); Hawk *et al.*, (1947)]

Component	Carbon (%)	Hydrogen (%)	Nitrogen (%)	Oxygen (%)
Water	-	11	-	89
Fat	77	12	-	11
Protein	52	7	16	23
Carbohydrates	42	6	-	52
Bone ash	-	-	-	40

Table 17(3).2: Reference man: total body content for some elements
(from ICRP 23)

Element	Amount (g)	Percent of total body weight
Oxygen	43,000	61
Carbon	16,000	23
Hydrogen	7000	10
Nitrogen	1800	2.6
Calcium	1000	1.4
Phosphorus	780	1.1
Sulphur	140	0.20
Potassium	140	0.20
Sodium	100	0.14
Chlorine	95	0.12
Magnesium	19	0.027
Silicon	18	0.026
Iron	4.2	0.006
Fluorine	2.6	0.0037
Zinc	2.3	0.0033
Rubidium	0.32	0.00046
Strontium	0.32	0.00046
Bromine	0.20	0.00029
Lead	0.12	0.00017
Copper	0.072	0.00010
Aluminium	0.061	0.00009
Cadmium	0.050	0.00007
Boron	< 0.048	0.00007
Barium	0.022	0.00003
Tin	< 0.017	0.00002
Manganese	0.012	0.00002
Iodine	0.013	0.00002
Nickel	0.010	0.00001
Gold	< 0.010	0.00001
Molybdenum	< 0.0093	0.00001
Chromium	< 0.0018	0.000003
Cesium	0.0015	0.000002
Cobalt	0.0015	0.000002
Uranium	0.00009	0.0000001
Beryllium	0.000036	-
Radium	3.1×10^{-11}	-

CHAPTER 17: SECTION 4

PHYSIOLOGICAL DATA FOR REFERENCE MAN

17(4).1 INTRODUCTION

This Section is a summary of ICRP 23, Chapter 3. It reviews those characteristics of man that relate directly or indirectly to intake, metabolism, and excretion of stable elements in man's environment. This background information is required for evaluation of intake and metabolism of radionuclides, since significant differences of metabolism are not expected for most isotopes of physiological importance (with the exception of hydrogen isotopes) when they reach the body in reasonable quantities and as food components. Values are also included for the overall intake of food, water, and air for the metabolic levels of Reference Man. ICRP 23, Chapter 3 is divided into two parts, the first part providing data for a physiological model of Reference Man (or Woman or Child), and the second relating to the metabolic balance of individual stable elements.

The following convention has been adopted by ICRP 23 regarding age: infants - up to 1 year; younger children - 1 to 8 years; children - 8 to 12 years; older children - 12 to 20 years; adults - > 20 years; Reference Man - 20 to 30 years. Subjects older than 60 years are rarely included.

Intake estimates have been derived from dietary surveys of relatively large groups, or by controlled balance studies of relatively few individuals. The two methods often provide widely different estimates of intake.

Table 17(4).1 is a summary of the extensive physiological data from the tables of ICRP 23. The parameters are arranged in alphabetical order. Values for Reference Man (adult), Reference Woman (adult) and Reference Child (10 years) have been tabulated. Table 17(4).2 gives some very limited data that were available for infants (1 year old) and newborns (arranged alphabetically).

Some additional information as well as explanation of some terms are provided in the following paragraphs. The order of presentation follows that of ICRP 23, Chapter 3.

17(4).2 ENERGY EXPENDITURE

Energy expenditure increases with body size up to about 20 years and then decreases by 3% for each decade up to 45 years, by 7.5% for each decade from 45 to 65 years, and by 10% from 65 to 75 years (Durnin and Passmore, 1967; FAO, 1957).

The gross energy expenditure (kcal/min) graded in relation to occupational activities for men are: light work - 2.2 to 5.3; moderate work - 5.4 to 8.0; heavy work - 8.1 to 11 (Durnin and Passmore, 1967).

Energy expenditure also depends on environmental temperature. From a mean standard temperature of 10°C, energy expenditure is decreased by 5% for every increase of 10°C, and increased by 3% for every decrease of 10°C (FAO, 1957).

17(4).3 RESPIRATORY STANDARDS

17(4).3.1 Definitions (Comroe et al., 1962)

Total lung capacity (TLC) is the amount of gas contained in the lungs at the end of a maximal inspiration. Vital capacity (VC) is the maximal volume of gas that can be expelled from the lungs by forceful effort after a maximum inspiration. Inspiratory capacity (IC) is the maximal volume of gas that can be inspired from the resting expiratory level. Functional residual capacity (FRC) is the volume of gas remaining in the lungs at the resting expiratory level. Tidal volume (TV) or depth of breathing is the volume of gas inspired or expired during each respiratory cycle. Inspiratory reserve volume (IRV) and Expiratory reserve volume (ERV) are the maximal volume of gas that can be inspired/expired from the end-inspiratory and end-respiratory positions respectively. Residual volume (RV) is the volume of gas remaining in the lungs at the end of maximum expiration. The anatomical dead space (VD) is the region where no respiratory exchange takes place; it includes the nose, nasopharynx, trachea, and tracheobronchial tree down to the respiratory bronchioles. The respiratory volume, or ventilatory flow (V), or minute volume (V), is a product of the tidal volume and frequency of breathing.

17(4).3.2 Changes in Respiratory Parameters under Different Conditions

Lung volumes increase in relation to increase in body weight and are correlated with body surface area. In the adult, TLC and VC decline with age, FRC does not change much with age, while VD increases slightly with age (Dejours, 1966). Respiratory rate increases 5-6 per minute for each degree C rise in body temperature (Comroe, 1944). Both tidal volume and frequency of breathing increase during exercise. Crying in infants increases minute volume by a factor of 3 (Deming and Washburn, 1935).

17(4).4 DAILY DIETARY INTAKE AND PRINCIPAL NUTRIENT CONTENT OF DIET

Estimates of per capita dietary intake can be arrived at either by national household surveys or on the basis of national supplies and size of the population.

The daily dietary intake as well as the types of food groups which constitute the intake are highly variable between geographical regions, increasing from about 900 g/day in the Far East to about 2200 g/day in North America (Hollingsworth and Hobson, 1966). For North America, the contributors to the diet are as follows (percentage by weight): milk - 39%; vegetables and fruits - 23%; meat, eggs and fish - 15%; cereals - 8%; starchy roots - 6%; sugar - 5%; fats and oils - 2.5%; pulses and nuts - 0.9%.

Carbohydrates can be considered as starch $(C_6H_{10}O_5)_x$, fat as glycerol tripalmitate $(C_{15}H_{31}COO)_3C_3H_5$, and protein as 50% carbon, 7% hydrogen and 20% oxygen.

17(4).5 MODEL FOR WATER BALANCE

A model for water balance is difficult to derive except on theoretical grounds because of the scarcity of adequate data for normal conditions of environment or activity, More information is available about extreme conditions. In addition, considerable variation is found among individuals or within the same individual on different occasions. Over long periods, homeostasis of body fluids and tissues will ensure that water gains exactly balance water losses.

The sources of water intake are: milk, tap water, other water-based fluids, fluid content of food, and oxidation of food. Water is lost from the body by a number of routes - in urine, in feces, and through the skin as sweat or by diffusion and transpiration.

17(4).5.1 Water Intake

At low temperatures fluid intake and water losses are scarcely affected by ambient temperature or activity; but at temperatures greater than 25° C, there is a sharp rise in water intake, largely to meet the demands of an increased sweat rate. (Greenleaf *et al.*, 1966).

17(4).5.2. Water Loss

17(4).5.2.1 Urine

Within an age group, daily amount of urine probably increases with body size, but is dependent on a number of other factors also. Circadian changes occur, with the greatest rate of flow between 3 and 6 p.m., and the smallest rate between 3 and 6 a.m. (Diem, 1962).

17(4).5.2.2 Sweat

Sweat loss is exceedingly variable, depending on surface area, body weight, environmental temperature, activity, and wind speed. Sweat production per unit body surface area is slightly greater in men than in women. Sweat production per unit body area in children is half that in adults (Dobson, 1967). An adult engaged in light indoor activity at 29° C produces 2 to 3 l/day; if temperature or activity is increased, rates can go up to 15 l/day (Schwartz, 1960).

At low rates of secretion, the concentration of sodium and chlorine is greater in sweat than in blood; but at high rates the concentrations in sweat approach those in blood.

17(4).5.2.3 Insensible Water Loss

Insensible water loss is related to body surface area, body weight, body temperature, and metabolic rate.

17(4).5.2.4 Saliva

Although up to 2 l of saliva may be secreted daily, there is usually little loss, as the bulk is swallowed and reabsorbed in the GI tract. In babies who lack an efficient swallowing reflex, the sodium balance may be affected in critical situations.

17(4).5.2.5 Nasal Secretion

The flow ranges from 100 to 1000 ml/day (Proctor, 1964). The bulk of the secretion will find its way to the GI tract.

17(4).6 EFFECT OF PREGNANCY AND LACTATION

During the total period of pregnancy, the total additional energy requirement is 80,000 kcal/266 days (9% for fetus; 9% for placenta and maternal tissues; 50% for 4-5 kg of additional fat; and 31% for increased metabolism) (Durnin and Passmore, 1967).

Milk secretion during lactation is 500 to 1000 ml per day (average 850 ml) (Young, 1964). For average milk production, the additional requirement is 1000 kcal/day. Table 17(4).3 gives the composition of human milk.

17(4).7 SUMMARY OF MODEL VALUES FOR DAILY BALANCE OF ELEMENTS

Table 17(4).4 gives the summary of model values for daily balance of elements in Reference Man, arranged alphabetically. Data for intake from food and fluids as well as from airborne intake are tabulated. The losses are mainly through urine, feces and sweat; other losses, such as through hair, are minor.

There is a fairly detailed discussion of the metabolism of 51 elements in ICRP 23, Chapter 3. This part is not included in this Handbook. However, in Chapter 25 dealing with dosimetric data for some important radionuclides, Table 25.5 summarizes the metabolic data for the concerned elements.

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Table 17(4).1: Reference man, woman and child: summary of physiological data

(based on ICRP 23)

Physiological data	Adult man	Adult woman	Child (10 y)
Carbon dioxide exhaled, g/day	1000	700	660
Dietary intake (nutrients), g/day			
Proteins	95	66	63
Carbohydrates	390	270	260
Fat	120	85	81
Dietary intake (major constituents), g/day			
Carbon	300	210	200
Hydrogen	350	245	230
Nitrogen	16	13	10
Oxygen	2600	1800	1700
Sulphur	1	0.7	0.7
Energy expenditure, kcal/day	3000	2100	2000
Feces, weight of, g/day	135	110	85
Feces, composition of, g/day			
Water	105	90	70
Solids	30	20	19
Ash	17	15	6
Fats	5	4.5	4
Nitrogen	1.5	1.3	1
Other substances	6.5	5	8
Feces, major elements in, g/day			
Carbon	7	6	4.2
Hydrogen	13	11	8.6
Nitrogen	1.5	1.3	1.0
Oxygen	100	90	62
Intake of milk, ml/day	300		
Lung capacities, l			
Total capacity	5.6	4.4	-
Functional residual capacity	2.2	1.8	-
Vital capacity	4.3	3.3	-
Dead space	0.16	0.13	-
Lung volume and respiration			
Minute volume, resting, l/min	7.5	6.0	4.8
Minute volume, light activity, l/min	20.0	19.0	13.0

(contd ...)

Physiological data	Adult man	Adult woman	Child 10 y
Lung volume and respiration			
Air breathed, 8 h light work activity, l/min	9600	9100	6240
Air breathed, 8 h non-occupational activity, l/min	9600	9100	6240
Air breathed, 8 h resting, l/min	3600	2900	2340
Metabolic rate, cal/min-kg W	17	16	25
Oxygen inhaled, g/day	920	640	600
Urine values			
Volume, ml/day	1400	1000	1000
Specific gravity	1.02		
pH	6.2		
Solids, g/day	60	50	47
Urea, g/day	22		
"Sugars", g/day	1		
Bicarbonates, g/day	0.14	0.12	-
Creatinine, g/day	1.7	1.0	-
Urinary loss of major elements, g/day			
Nitrogen	15	13	11
Hydrogen	160	130	110
Oxygen	1300	1100	970
Carbon	5	4	3
Water balance (gains), ml/day			
Total fluid intake	1950	1300	1400
Milk	300	200	450
Tap water	150	100	200
Other	1500	1100	750
In food	700	450	400
By oxidation of food	350	250	200
Total	3000	2100	2000
Water balance (losses), ml/day			
In urine	1400	1000	1000
In feces	100	90	70
Insensible loss	850	600	550
In sweat	650	420	350
Total	3000	2100	2000

Table 17(4).2 : Reference infant and newborn: some physiological data
(based on ICRP 23)

Physiological data	Infant (1yr)	Newborn
Feces, weight of, g/day	85	24
Feces, components of, g/day		
Water	70	19
Solids	19	5
Ash	6	1
Fats	4	3
Nitrogen	1	0.3
Other substances	8	0.7
Feces, major elements in, g/day		
Carbon	4.2	1.2
Hydrogen	8.6	2.5
Nitrogen	1.0	0.3
Oxygen	62	17
Lung volume and respiration		
Minute volume, l/min		
Resting	1.5	0.5
Light activity	4.2	1.5
Liters of air breathed, l/day		
Light activity	2500 (10 h)	90 (1 h)
Resting	1300 (14 h)	690 (23 h)
Metabolic rate, cal/min-kg W	25	35
Urine values		
Volume, ml/day	450	
Solids, g/day	19	
Urinary loss of major elements, g/day		
Nitrogen	8	
Hydrogen	50	
Oxygen	420	
Carbon	0.5	

Table 17(4).3: Composition of human milk

[based on ICRP 23 (collated from Albritton, 1954; Altman and Dittmer, 1968; Diem, 1962; Macy, 1949; Spector, 1956; WHO, 1970; others; further detailed references are given in ICRP 23]

Water	870	ml/l
Specific gravity	1.03	g/cc
Total solids	12.9	g/100 ml
Fats	4.5	g/100 ml
Carbohydrates	7.1	g/100 ml
Proteins	1.2 - 2.7	g/100 ml
Calcium	34	mg/100 ml
Chlorine	37	mg/100 ml
Cobalt	0.0001	mg/100 ml
Copper	0.045	mg/100 ml
Fluorine	< 0.01 - 0.02	ppm
Iodine	0.007	mg/100 ml
Iron	0.15	mg/100 ml
Magnesium	3.5	mg/100 ml
Phosphorus	14	mg/100 ml
Potassium	51	mg/100 ml
Silicon	0.034	mg/100 ml
Sodium	17	mg/100 ml
Sulphur	14	mg/100 ml
Zinc	0.53	mg/100 ml

Table 17(4).4 : Summary of model values for daily balance of elements in reference man

(based on ICRP 23)

Element	Daily intake		Daily losses				Units
	Food and fluids	Air-borne	Urine	Feces	Others		
Aluminium	45	0.10	0.10	43	1	(S)	mg
Antimony	50	0.05	40	9	1	(H)	µg
Arsenic	1.0	0.0014	0.05	0.8	0.15		mg
Barium	0.75	0.0001-0.026	0.05	0.69	0.08 0.01	(H) (S)	mg mg
Beryllium	12	< 0.01	1.0	10	1		µg
Bismuth	20	< 0.01	1.6	18	?		µg
Boron	1.3	-	1.0	0.27	-		µg
Bromine	7.5	-	7.0	0.07	0.2	(S)	mg
Cadmium	150	< 1	100	50	-		µg
Carbon	300	-	5.0	7.0	270 + 18	(E)	g
Cesium	10	0.025	9.0	< 1.0	-		µg
Chlorine	5.2	-	4.4	0.05	0.8	(S)	g
Chromium	150	0.1	70	80	1.6	(S,H)	µg
Cobalt	300	< 0.01	200	90	6.4	(S,H)	µg
Copper	3.5	0.02	0.05	34	0.2	(S)	mg
Fluorine	1.8	-	1.0	0.15	0.65	(S)	mg
Germanium	1.5	-	1.4	0.10	-		mg
Hydrogen	350	-	160	13	72 + 95 + 10	(S) (I)	g
Iodine	200	0.5 - 35	170	20	8.3	(S,H)	µg
Iron,							
Males	16	0.03	0.25	15	0.5	(S)	mg
Females	12	0.03	0.20	11	0.6 + 0.5	(M) (S)	mg
Lead	0.44	0.01	0.045	0.3	0.1	(S)	mg
Lithium	2.0	-	0.8	1.2	-		mg
Magnesium							
Males	0.34	-	0.13	0.21	-		g
Females	0.27	-	0.11	0.16	-		g
Manganese	37	0.002	0.03	36	0.04	(S)	mg
Mercury	15	1	0.35	10	1	(H)	µg
Molybdenum	300	< 0.1	150	120	20	(S)	µg

(contd ...)

Element	Daily intake		Daily losses				Units
	Food and fluids	Air-borne	Urine	Feces	Others		
Nickel	400	0.6	11	370	21	(S)	µg
Niobium	620	-	360	260	0.3	(H)	µg
Nitrogen							
Males	16	-	15	1.5	0.3	(S)	g
Females	13	-	13	1.3	0.3	(S)	g
Oxygen							
Males	2600	920	1300	100	720	(E)	g
					+ 586	(S)	
					+ 760	(I)	
Females	1800	640	1100	90	510	(E)	g
					+ 370	(S)	
					+ 530	(I)	
Phosphorus	1.4	-	0.90	0.50	-		g
Polonium-210	3.2	< 0.0	0.011	3.3	-		pCi
Potassium	3.3	-	2.8	0.36	0.1	(S)	g
Radium-226	2.3	-	0.08	2.2	-		pCi
Rubidium	2.2	-	1.9	0.3	0.05		mg
Selenium	150	-	50	20	80	(S)	µg
Silicon	3.5	15 ?	10	10	0.3	(H)	mg
Silver	70	-	9	60	1.0	(S,H)	µg
Sodium	4.4	-	3.3	0.1	1.0	(S)	g
Strontium	1.9	-	0.34	1.5	-		mg
Sulphur	0.85	-	0.8	0.14	0.06	(S,H)	g
Tellurium	0.6	0	0.53	0.10	-		mg
Thallium	1.5	0.05	0.5	1.0	-		µg
Thorium	3	-	0.1	2.9	-		µg
Tin	4	-	0.02	3.5	0.5	(S)	mg
Titanium	0.85	0.001	0.33	0.52	-		mg
Uranium	1.9	0.007	0.05-0.5	1.4-1.8	0.02	(H)	µg
Vanadium	2	0.002	0.015	2	-		mg
Zinc	13	< 0.1	0.5	11	0.8	(S)	mg
					+ 0.8	(M)	
Zirconium	4.2	-	0.15	4	-		mg

Note: S -sweat; H -hair; E - exhaled; I -insensible sweat loss; M -menstrual loss.

CHAPTER 17. SECTION 5

SPECIFIC ABSORBED FRACTIONS OF PHOTON ENERGIES FOR REFERENCE MAN

The parameters 'absorbed fraction', ϕ, and 'specific absorbed fraction', Φ, have been defined, and the basic principles for their computation are briefly explained in Chapters 18 and 19.

In Appendix 1 to ICRP 23, values of the specific absorbed fraction for photons have been tabulated as a dosimetric characteristic of Reference Man. It is assumed that the radioactivity is uniformly distributed in the source organ. Most of the data have been computed by the Monte Carlo method on a modification of the MIRD 5 anthropomorphic phantom (Snyder *et al.*, 1969). The source was placed in 16 different 'organs' including the total body. Computations were made for 12 source energies ranging from 10 keV to 4 MeV. The sample size was at least 60,000 photons.

Some computations were also made by integrating the build-up factor tabulated by Berger (1968) for an infinite homogeneous medium; the values of the specific absorbed fraction obtained by this method did not differ by more than a factor of 2 from the Monte Carlo values; the variation was reduced to 20-30% as the statistics of the Monte Carlo estimates were improved (Snyder *et al.*, 1971, 1972a, b).

The specific absorbed fraction is relatively insensitive to the mass of the target organ for which it is defined provided that the source is not in the organ (Loevinger and Berman, 1968; Snyder, 1971). ICRP 23 gives a simple formula for computation of the specific absorbed fraction in case one wants to use a mass different from that of the phantom used for the calculations.

Some specific points used in the methodology of computations may be mentioned. Red bone marrow has been distinguished as a target organ but has not been used as a source organ. Snyder *et al.* (1969), in MIRD Pamphlet 5, considered the 4 sections of the GI tract and the bladder as solid organs. When the source is in the contents of these organs, the dose varies by as much as a factor of 2 in the wall and the contents. This point

has been taken care of in the present computations. Further, corrections have been made to take into account the inhomogeneity of certain organs (such as bone).

There are some ambiguities in the choice of source and target organs in certain cases. Thus skeleton is suggested as the organ to be used if cortical or cancellous bone or red or yellow marrow are sources, or as target organ if dose to total endosteal cells is required; strictly speaking, these approximations would not be valid for localized sources or other forms of radiation (alphas, beta-like radiations). However, to a first approximation, a nuclide depositing in bone or in marrow will produce a dose commitment, averaged over this tissue, which is equal to these averages in the total marrow or in the skeleton.

There is no region of the phantom which is designated as 'muscle'. Whenever muscle is a target organ, it can be considered as having the same specific absorbed fraction as the total body minus all the designated target organs. This compartment, termed 'other tissues', totals some 48 kg, and thus includes much other tissue in addition to muscle. However, muscle is rather well distributed throughout this mass, and hence the absorption per gram may be expected to be rather similar, and the specific absorption factor for 'other tissues' can be taken as approximately the value for muscle. When muscle is indicated as a source organ, the reciprocity theorem can be used. (For further details, the original may be consulted.)

Table 17(5).1 gives in a very brief form some of the data on specific absorbed fractions contained in the original publication. The specific absorbed fractions are tabulated for all source organs but for only two target organs (out of the 18 considered in the original), viz. (a) source and target organ the same (for which the value of the specific absorbed fraction would be the highest), and (b) total body (TB) as target organ.

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Table 17(5).1: Specific absorbed fractions of photon energy
(based on ICRP 23)

Photon energy (MeV)	Specific absorbed fraction		Specific absorbed fraction	
	S<—S	TB<—S	S<—S	TB<—S
	S: bladder contents (a)		*S: stomach contents (b)*	
0.010	8.49E-04	1.43E-05	3.21E-04	1.43E-05
0.015	1.40E-03	1.43E-05	7.61E-04	1.43E-05
0.020	1.43E-03	1.42E-05	8.96E-04	1.42E-05
0.030	9.83E-04	1.31E-05	6.87E-04	1.31E-05
0.050	4.49E-04	1.00E-05	3.28E-04	9.75E-06
0.100	2.56E-04	7.28E-06	1.88E-04	6.87E-06
0.200	2.47E-04	6.58E-06	1.78E-04	6.21E-06
0.500	2.56E-04	6.44E-06	1.79E-04	6.11E-06
1.000	2.22E-04	5.98E-06	1.65E-04	5.74E-06
1.500	2.07E-04	5.63E-06	1.48E-04	5.39E-06
2.000	1.97E-04	5.35E-06	1.41E-04	5.07E-06
4.000	1.57E-04	4.44E-06	1.15E-04	4.22E-06
	S:SI + wall (c)		*S: ULI contents (d)*	
0.010	7.40E-04	1.43E-05	3.63E-04	1.43E-05
0.015	6.61E-04	1.43E-05	8.01E-04	1.43E-05
0.020	5.60E-04	1.43E-05	8.62E-04	1.43E-05
0.030	3.71E-04	1.37E-05	5.57E-04	1.35E-05
0.050	1.87E-04	1.08E-05	2.62E-04	1.05E-05
0.100	1.13E-04	7.82E-06	1.52E-04	7.61E-06
0.200	1.07E-04	7.02E-06	1.47E-04	6.79E-06
0.500	1.07E-04	6.74E-06	1.47E-04	6.57E-06
1.000	9.57E-05	6.27E-06	1.39E-04	6.17E-06
1.500	9.07E-05	5.92E-06	1.24E-04	5.77E-06
2.000	8.23E-05	5.51E-06	1.18E-04	5.46E-06
4.000	6.68E-05	4.60E-06	9.33E-05	4.50E-06

(contd ...)

Photon energy (MeV)	Specific absorbed fraction		Specific absorbed fraction	
	S<—S	TB<—S	S<—S	TB<—S
	S:LLI contents (e)		*S: kidneys*	
0.010	5.53E-04	1.43E-05	3.28E-03	1.43E-05
0.015	1.13E-03	1.43E-05	2.74E-03	1.43E-05
0.020	1.16E-03	1.43E-05	2.04E-03	1.40E-05
0.030	7.02E-04	1.37E-05	1.03E-03	1.23E-05
0.050	2.97E-04	1.08E-05	3.93E-04	9.01E-06
0.100	1.76E-04	7.62E-06	2.35E-04	6.45E-06
0.200	1.74E-04	6.77E-06	2.39E-04	5.90E-06
0.500	1.77E-04	6.53E-06	2.52E-04	5.84E-06
1.000	1.62E-04	6.10E-06	2.26E-04	5.48E-06
1.500	1.52E-04	5.76E-06	2.14E-04	5.21E-06
2.000	1.39E-04	5.44E-06	1.93E-04	4.87E-06
4.000	1.08E-04	4.48E-06	1.63E-04	4.12E-06
	S: liver		*S: lungs*	
0.010	5.36E-04	1.43E-05	8.17E-04	1.43E-05
0.015	4.96E-04	1.43E-05	6.58E-04	1.43E-05
0.020	4.34E-04	1.42E-05	4.71E-04	1.41E-05
0.030	2.97E-04	1.29E-05	2.30E-04	1.26E-05
0.050	1.52E-04	9.48E-06	8.99E-05	8.78E-06
0.100	9.14E-05	6.54E-06	5.05E-05	5.70E-06
0.200	1.36E-05	5.94E-06	5.00E-05	5.11E-06
0.500	1.29E-05	5.86E-06	5.01E-05	4.99E-06
1.000	1.18E-05	5.49E-06	4.55E-05	4.66E-06
1.500	1.14E-05	5.16E-06	4.32E-05	4.36E-06
2.000	1.01E-05	4.86E-06	3.92E-05	4.12E-06
4.000	8.23E-06	4.06E-06	3.08E-05	3.41E-06

(contd ...)

Photon energy (MeV)	Specific absorbed fraction		Specific absorbed fraction	
	S<—S	TB<—S	S<—S	TB<—S
	S: muscle (f)		*S: ovaries*	
0.010	1.81E-05	1.43E-05	9.68E-02	1.43E-05
0.015	1.70E-05	1.37E-05	5.91E-02	1.43E-05
0.020	1.52E-05	1.25E-05	3.26E-02	1.43E-05
0.030	1.15E-05	9.93E-06	1.15E-02	1.39E-05
0.050	7.13E-06	6.60E-06	3.58E-03	1.13E-05
0.100	5.08E-06	4.86E-06	2.23E-03	8.10E-06
0.200	5.03E-06	4.82E-06	2.48E-03	7.16E-06
0.500	5.22E-06	4.97E-06	2.62E-03	6.88E-06
1.000	4.99E-06	4.75E-06	2.42E-03	6.38E-06
1.500	4.65E-06	4.45E-06	2.28E-03	5.97E-06
2.000	4.36E-06	4.18E-06	2.12E-03	5.65E-06
4.000	3.66E-06	3.51E-06	1.72E-03	4.70E-06
	S: pancreas		*S: skeleton (g)*	
0.010	1.47E-02	1.43E-05	9.34E-05	1.43E-05
0.015	1.09E-02	1.43E-05	8.97E-05	1.42E-05
0.020	7.14E-03	1.43E-05	8.42E-05	1.40E-05
0.030	3.16E-03	1.39E-05	6.84E-05	1.28E-05
0.050	1.10E-03	1.11E-05	4.10E-05	9.31E-06
0.100	6.51E-04	7.92E-06	1.81E-05	5.73E-06
0.200	6.88E-04	6.99E-06	1.29E-05	5.01E-06
0.500	7.29E-04	6.74E-06	1.19E-05	4.93E-06
1.000	6.74E-04	6.22E-06	1.11E-05	4.70E-06
1.500	6.33E-04	5.90E-06	1.01E-05	4.43E-06
2.000	5.72E-04	5.55E-06	9.39E-05	4.16E-06
4.000	4.58E-04	4.53E-06	7.79E-05	3.50E-06

(contd ...)

Photon energy (MeV)	Specific absorbed fraction		Specific absorbed fraction	
	S<—S	TB<—S	S<—S	TB<—S
	S: skin		*S: spleen*	
0.010	2.05E-04	1.13E-05	5.44E-03	1.43E-05
0.015	1.03E-04	9.06E-06	4.66E-03	1.43E-05
0.020	5.57E-05	7.77E-06	3.59E-03	1.42E-05
0.030	2.24E-05	6.17E-06	1.86E-03	1.30E-05
0.050	8.07E-06	4.21E-06	7.24E-04	9.47E-06
0.100	5.33E-06	2.87E-06	4.21E-04	6.52E-06
0.200	6.05E-06	2.81E-06	4.32E-04	5.93E-06
0.500	6.93E-06	3.15E-06	4.49E-04	5.82E-06
1.000	6.93E-06	3.15E-06	4.10E-04	5.46E-06
1.500	6.38E-06	2.97E-06	3.77E-04	5.16E-06
2.000	6.01E-06	2.81E-06	3.55E-04	4.81E-06
4.000	4.78E-06	2.35E-06	2.73E-04	4.02E-06
	S: testes		*S: thyroid*	
0.010	2.43E-02	1.43E-05	4.29E-02	1.43E-05
0.015	1.86E-02	1.38E-05	2.93E-02	1.42E-05
0.020	1.23E-02	1.26E-05	1.81E-02	1.35E-05
0.030	5.34E-03	1.00E-05	7.41E-03	1.08E-05
0.050	1.77E-03	6.84E-06	2.42E-03	6.95E-06
0.100	1.07E-03	5.03E-06	1.44E-03	4.71E-06
0.200	1.17E-03	4.82E-06	1.55E-03	4.42E-06
0.500	1.23E-03	4.91E-06	1.66E-03	4.50E-06
1.000	1.16E-03	4.69E-06	1.54E-03	4.26E-06
1.500	1.04E-03	4.47E-06	1.45E-03	4.03E-06
2.000	9.82E-04	4.23E-06	1.31E-03	3.78E-06
4.000	7.66E-04	3.54E-06	1.05E-03	3.18E-06

(contd ...)

Photon energy (MeV)	Specific absorbed fraction		Specific absorbed fraction	
	S<—S	TB<—S	S<—S	TB<—S
	S: total body			
0.010		1.42E-05		
0.015		1.38E-05		
0.020		1.32E-05		
0.030		1.14E-05		
0.050		8.13E-06		
0.100		5.41E-06		
0.200		4.95E-06		
0.500		4.97E-06		
1.000		4.72E-06		
1.500		4.40E-06		
2.000		4.16E-06		
4.000		3.48E-06		

S: source organ; S<—S indicates that source and target organs are the same; TB<—S indicates that total body is the target organ.

(a) target organ: bladder wall; (b) target organ: stomach wall; (c) source organ: small intestine plus walls; target organ: small intestine plus contents; (d) source organ: upper large intestine contents; target organ: upper large intestine wall; (e) source organ: lower large intestine contents; target organ: LLI wall; (f) target organ: 'other tissues' (suggested for muscle); and (g) source organ: skeleton (suggested for cancellous bone, cortical bone, bone marrow and yellow marrow); target organ: skeleton (suggested for total endosteal cells).

CHAPTER 18

INTERNAL DOSIMETRY:INTRODUCTION TO ICRP 30 METHODOLOGY

18.1 INTRODUCTION

ICRP publication 30, 'Limits for intakes of radionuclides by workers', a report of Committee 2, is by far the most voluminous report published so far by ICRP. It consists of 4 main parts, Part 1 (1979), Part 2 (1980), Part 3 (including addendum to Parts 1 and 2) (1981), and Part 4 (An addendum) (1988), running together to a total of 474 pages; Supplement to Part 1 (1979), Supplement to Part 2 (1981), and Supplements A and B to Part 3 (1982) running together to a total of 2254 pages; and an Index ('A subject index and a comprehensive index of radionuclides considered in all parts and supplements to ICRP publication 30') (1982) running to 73 pages. ICRP 30 supersedes ICRP publication 2, 'Report of Committee II on permissible dose for internal radiation' (1959).

A somewhat detailed account of the method of presentation of the data in ICRP 30, its Supplements, as well as in ICRP publication 38, 'Radionuclide transformations: energy and intensity of emissions' (1983) (which gives the relevant physical data on radionuclides necessary for dosimetry), is given in Chapter 20.

A major revision of ICRP 2 became necessary for a variety of reasons:

(a) Advances in our understanding of the metabolic behaviour of radionuclides and development of newer dosimetric models, subsequent to the publication of ICRP 2, as evidenced by the publication of several reports of ICRP and its Task Groups. Important among these may be mentioned: Eve's GI tract model (Eve, 1966), report of the Task Group on Lung Dynamics (1966), ICRP publication 11, 'A review of the radiosensitivity of tissues in bone' (1968), ICRP publication 19, 'The metabolism of compounds of plutonium and the actinides' (1972), ICRP publication 20, 'Alkaline earth metabolism in adult man' (1973); and ICRP publication 23, 'Reference Man: anatomical, physiological and metabolic characteristics' (1975);

(b) Availability and utilization of several newer radionuclides;

(c) Major revision of ICRP's main recommendations as given in ICRP publication 26 (1977).

ICRP publication 48, 'The metabolism of plutonium and related elements'(1986) which supplemented ICRP 19 with a lot of newer information on the metabolic behaviour of the transuranics led to the work reported in ICRP 30, Part 4.

ICRP 2 dealt with 235 radionuclides of 88 elements. Some changes and additional recommendations concerning 20 radionuclides of 8 elements were subsequently made in ICRP 6 (1964), but no systematic revision of ICRP 2 had been published. In ICRP 30, dosimetric data were computed for 1351 inhalation and 855 ingestion intakes of 735 radionuclides and 101 submersion exposures for 26 noble gas radionuclides.

ICRP publication 56, 'Age-dependent doses to members of the public from intakes of radionuclides: Part I' (1989) gave additional dosimetric data for selected radionuclides based on newer biokinetic information. It gave organ doses and effective doses to members of the public of various age groups from 3 months to adults.

Consequent on the revisions of the main recommendations in 1990 as given in ICRP publication 60, a further revision of permissible intakes by inhalation and ingestion became necessary. New values of ALIs which incorporate the new dose limits, radiation and tissue weighting factors, and the metabolic and biokinetic information from ICRP 30, have been calculated. These have been reported in ICRP publication 61, 'Annual limit on intakes of radionuclides by workers based on the 1990 recommendations' (1991). It gives f_1 and ALI values (but not DAC values) for 855 cases of ingestion and 1351 cases of inhalation (D class: 465; W class: 599; Y class: 284; Vapour class: 23) for 734 radionuclides of 91 elements from hydrogen to mendelevium (excluding lithium, boron, nitrogen, oxygen, and the noble gases helium, neon, krytpon, xenon and radon); it does not deal with submersion exposures. (The terms f_1, ALI and DAC are explained later in this chapter; the terms D, W and Y are explained in Chapter 21.)

Eventually, a new complete revision of ICRP 30 will be issued, taking into account a new respiratory tract model, a new Reference Man report, and new biokinetic models.

No radical departures from the methodology or dosimetric models used by ICRP 30 have been effected in either ICRP 56 or ICRP 61. We shall therefore first discuss in some detail the dosimetric formalism adopted in ICRP 30. The contents of ICRP 61 will be presented subsequently.

ICRP 2 served as a satisfactory guide for the control of intakes of radionuclides into the body to meet the basic standards of ICRP in force at that time, although there have been some misconceptions about its intent and some misuse of its recommendations. ICRP 2 recommended values for the maximum permissible concentrations (MPC) in air and in water and of maximum permissible body burdens (MPBB) for a number of radionuclides. For any radionuclide, continuous exposure of a Standard Man to either of the MPC values during the whole of a working lifetime of 50 years would result in his containing the MPBB of the radionuclide at the end of that period. The MPBB was estimated to deliver a dose equivalent rate in the appropriate critical organ such that the ICRP limits were not exceeded. Although ICRP 2 emphasized that the rate of intake of a radionuclide could be varied, provided that the intake in any quarter year was no greater than that resulting from continuous exposure to the appropriate MPC for 13 weeks, the concept of MPC was being misused to imply a maximum concentration in air or water that should be never exceeded under any circumstances. Similarly, MPBB had been misused to imply a maximum for the activity in the body, although it is evident that an intake in excess of MPBB for a radionuclide with a short residence time in the body would not result in the dose limits being exceeded. For these and other reasons discussed below the concepts of MPC and MPBB have been discarded in ICRP 30.

ICRP 30 is concerned with the derivation of the secondary standards that limit the intake of radionuclides by workers so that the primary occupational dose limits are not exceeded. The time over which the dose equivalent should be integrated is a working life of 50 years. The total dose equivalent to any tissue over the 50 years after intake of a radionuclide is termed the 'committed dose equivalent', H_{50}. Values of this parameter are given for a number of organs and tissues in the Supplements which give dosimetric data for individual radionuclides based on the characteristics of Reference Man.

Several organs and tissues will be irradiated following the intake. The intake in any year must be limited to satisfy the following conditions:

$$\sum_T W_T H_{50,T} \leqslant 0.05 \text{ Sv} \quad \text{(ICRP 26 stochastic limit);}$$

and

$$H_{50,T} \leqslant 0.5 \text{ Sv} \quad \text{(ICRP 26 non-stochastic limit),}$$

where W_T is the (ICRP 26) tissue weighting factor, and $H_{50,T}$ (in Sv) is the total committed dose equivalent in tissue (T) resulting from intakes of radioactive materials from all sources during the year in question. Table APP 8(2).1 of Appendix 8.2 of this Handbook gives the (ICRP 26) weighting factors for stochastic risks.

Secondary and derived limits for use in practice are discussed subsequently. For each radionuclide in each of several forms, a secondary standard is given for the Annual Limit on Intake (ALI) either by ingestion or inhalation. A Reference Man (ICRP 23) receiving any such intake would be irradiated to the dose limit for each year of occupational exposure. Values are also given for the Derived Air Concentration (DAC) of each radioactive material. This is obtained by dividing the ALI by the volume of air inhaled by the Reference Man in a working year. DACs are also derived for submersion of Reference Man in a cloud of a radioactive inert gas and in a cloud of elemental tritium; in this case the limit is set by the doses to the organs and tissues from external irradiation.

DAC values are analogous to the values of MPC (air) derived in ICRP 2. DAC values must be used circumspectly. For inhalation the overriding limit is the ALI and for submersion it is the time integral of the concentration of the radionuclide in air during any year of practice. No standard is developed here for a derived concentration in water because water is only one source of ingested material. The total activity ingested in any year should be controlled by use of the value of ALI for ingestion.

The general principles used to calculate H_{50} per unit intake are described in the next Chapter (Chapter 19). H_{50} in any target organ includes the dose from photons emitted by the radionuclide in other organs of the body as well as that from radiations arising from the radionuclide in the target organ itself. The total energy absorbed per unit mass in any target organ for each transformation of the radionuclide in any source organ, suitably weighted by the Quality Factor, is known as the Specific Effective Energy (*SEE*). H_{50} in any target organ is directly related to *SEE* and to the number of transformations, U, which occur in source organs during the 50 years after the intake of the radionuclide. The values of H_{50} given here refer to unit intake of the stated radionuclide alone, but include the

committed dose equivalent contributed by any daughter radionuclide produced from transformations of the parent radionuclide within the body.

Specific models for the routes of entry of radioactive materials into the body, namely the Respiratory System (Lung Model) and the Gastrointestinal (GI) Tract are described in Chapters 21 and 22. The lung model is used to calculate the average dose equivalent to the lung, considered for dosimetric purposes as a composite tissue comprising the trachea and bronchi, the pulmonary region and its associated regional lymph nodes. The model is also used to calculate those fractions of the inhaled activity which are transferred either to body fluids or to the GI tract. Similarly, the GI tract model is used first to calculate the average committed dose equivalents in anatomically distinct sections of the tract, which are regarded as separate organs in the application of the basic dose limits, and secondly to calculate f_1, the fraction of the ingested activity transferred to body fluids.

Chapter 23, Section 1 describes the model used for bone dosimetry. Committed dose equivalent is estimated for the relevant target tissues, identified as certain cells near bone surfaces and the red bone marrow.

Chapter 24 describes the model used to derive DAC values for submersion of an individual in a radioactive, chemically inert gas or in elemental tritium. (The limiting factor for tritium is irradiation of the lung, and, for inert gases, irradiation of the body. Internal irradiation from absorbed gas can be disregarded.)

After a general discussion of the basic methodologies, ICRP 30 goes on to present the relevant metabolic data required to calculate ALI and DAC for a number of radionuclides. Data on daily intake, excretion, body content and distribution for the stable element are given for the Reference Man (ICRP 23). A description of the metabolism of compounds of the element is given, together with values for their absorption from the GI tract to the body fluids, their lung clearance classification as per the corresponding models, and models describing the retention in the whole body and relevant organs of the adult. After each set of metabolic data for an element, values are given for ALI and DAC for radioisotopes with radioactive half-life in excess of 10 min. The relevant dosimetric data for individual radionuclides are given in the Supplements in the form of a computer print-out. They include information on the radioactive transformations of the radionuclide, values of *SEE* for a number of target organs from activities in different source organs, the number of transformations in several source organs per unit intake by ingestion and inhala-

tion of various chemical forms, corresponding values of H_{50} and of H_{50} weighted by the tissue weighting factor for that organ or tissue, and finally a table of values of ALI and DAC for occupational exposure. Except for Part 4 of ICRP 30 dealing with the transuranics, values for the committed effective dose, $E(50)$, are not given in ICRP 30 and its Supplements. However, since the values of H_{50} for organs that receive a substantial dose, weighted by the tissue weighting factor appropriate to each of those organs, are tabulated in the Supplements, these values can be summed up to give $E(50)$.

A Chapter in ICRP 30, Part 1 is devoted to the limitations on the use of the data. The total dose equivalent (received and committed) is the primary factor, and the values of H_{50}, ALI and DAC are criteria that are useful only for interpreting the occupational exposure of adults. Information is not given for other ages. The values of ALI are based on consideration of radiation dose alone; chemical toxicity has not been considered. The models used here have been chosen, often conservatively, to derive values of ALI and DAC to ensure the protection of workers.

18.2 SECONDARY AND DERIVED LIMITS FOR THE CONTROL OF INTERNAL DOSE

The units of relevance here are the Committed Dose Equivalent, the Annual Limit on Intake and Derived Air Concentration, which have already been defined in Chapter 16.

18.2.1 Annual Limit on Intake (ALI)

ALI is a secondary limit derived as follows: ALI is the greatest value of the annual intake I which satisfies both the following inequalities:

$$I \sum_T W_T (H_{50,T} \text{ per unit intake}) \leq 0.05 \text{ Sv}$$

$$I \ (H_{50,T} \text{ per unit intake}) \leq 0.5 \text{ Sv}$$

where I (in Bq) is the annual intake of the specified radionuclide either by ingestion or inhalation; W_T is the weighting factor for tissue T; and $H_{50,T}$ per unit intake (in Sv/Bq) is the committed dose equivalent in tissue T from the intake of unit activity of the radionuclide by the specified route.

Values of ALI are estimated for exposure, both by ingestion and inhalation, to the specified radionuclide. Any daughter radionuclides produced in the body after intake of the specified radionuclide are taken into

account. When the intake consists of a mixture of radionuclides, and/or exposure occurs both by ingestion and inhalation, the values of ALI do not directly apply. In such cases the intakes in any one year should be controlled so that the sum of the weighted committed dose equivalents from all sources is less than 0.05 Sv (stochastic limit), and, in addition, the committed dose equivalent to any organ does not exceed 0.5 Sv (non-stochastic limit).

Values of ALI and $H(50)$ have been calculated for the parameters of a Reference Man. However, when there is reason to believe that a particular individual has taken in more than the ALI, it is recommended that his age and known biological parameters should be taken into account as far as it is practicable, in the estimate of the committed dose equivalent.

18.2.2 Derived Air Concentration (DAC)

The concentration of a radionuclide in air during any year is limited as follows:

$$\int C(t)\ B(t)\ dt \leq ALI \qquad \text{Bq}$$

where at any time t, $C(t)$ (in Bq m^{-3}) is the concentration of the radionuclide in air; and $B(t)$ (in m^3 per unit time) is the volume of air breathed by the worker per unit time, and the limits on integration are over a working year.

The value of $B(t)$ depends on the type of work being performed and for heavy work it could be more than twice the reference value for 'light activity' given in ICRP 23.

For convenience, ICRP recommends values for DAC. The DAC for any radionuclide is that concentration in air (expressed in Bq m^{-3}) which, if breathed by Reference Man for a working year of 2000 h (50 weeks at 40 h per week) under conditions of 'light activity' would result in the ALI by inhalation.

$$\text{DAC} = \text{ALI}/(2000 \times 60 \times 0.02)$$

$$= \text{ALI}/(2.4 \times 10^3) \text{ Bq m}^{-3}$$

where 0.02 m^3 is the volume of air breathed at work by Reference Man per minute under conditions of 'light activity'. It is emphasized that the ALI is the overriding limit and the derived limit DAC should always be used circumspectly.

18.2.3 Derived Air Concentration for Submersion, DAC (Submersion)

The parameter DAC (submersion) has already been defined. The methods used to derive these values are described in the following and, in more detail, in Chapter 24.

18.2.3.1 DAC (Submersion) for Elemental Tritium

Exposure to elemental tritium in air during any year is limited by consideration of stochastic effects in the lung as follows:

$$W_{Lung}\ \dot{H}_{Lung} \int C(t)\ dt \leq 0.05 \quad \text{Sv}$$

where W_{Lung} is the weighting factor for lung; $\dot{H}_{Lung}$ (in Sv m^3 $Bq^{-1}h^{-1}$) is the dose-equivalent rate to lung from exposure to unit concentration of tritium in air (i.e. 1 Bq m^{-3}); and $C(t)$ (in Bq m^{-3}) is the concentration of elemental tritium in air at any time t and the limits of integration are over a working year.

For convenience, a value of DAC is recommended which is 1/2000th of the greatest value of $\int C(t)\,dt$ that satisfies the above equation. A worker may be exposed to concentrations of elemental tritium in air greater than the DAC provided that the basic conditions of the above relation are satisfied.

18.2.3.2 DAC (Submersion) for a Radioactive Noble Gas

Exposure to a radioactive noble gas (other than radon and thoron which are the subject of a separate ICRP report) during any year is limited by consideration of external exposure of the body as follows:

$$W_T\ \dot{H}_T \int C(t)\,dt \leq 0.05 \quad \text{Sv}$$

and

$$\dot{H}_T \int C(t)\,dt \leq 0.5 \quad \text{Sv}$$

and

$$\dot{H}_{Lens} \int C(t)\, dt \leq 0.3 \quad \text{Sv}$$

where $C(t)$ (in Bq m^{-3}) is the concentration of the radioactive noble gas in air at any time t and the limits on integration are over a year; W_T is the weighting factor for tissue T; $\dot{H}_T$ (in Sv m^3 Bq^{-1} h^{-1}) is the dose-equivalent rate in any tissue T; and $\dot{H}_{Lens}$ is the corresponding value for the lens of the eye resulting from submersion of Reference Man in unit concentration of the noble gas in air (i.e. 1 Bq m^{-3}).

For convenience the DAC (submersion) is taken as 1/2000th of the greatest value of $\int C(t)\, dt$ that satisfies the relationship given above.

18.3 REVISIONS TO ICRP 30 CARRIED OUT IN ICRP 61

18.3.1 Dosimetric Data

ICRP 61 has utilized, in addition to data given in ICRP 30, information from the related unabridged tabulations retained in the dosimetric data files at Oak Ridge National Laboratory, Tennessee. ICRP 30 database contains no information for the esophagus and since the transit time in the esophagus is quite rapid, the dose to the thymus was used to approximate the dose to the esophagus. In addition, skin is no longer excluded from the definition of effective dose. As explained in the discussion on ICRP 60 recommendations, the 'remainder' is now composed of 10 organs (see Table 2.2, Chapter 2).

The committed effective dose $E(50)$ for the worker is computed here as:

$$E(50) = \sum_{T=1}^{T=j} w_T H_T(50) + w_{remainder} \frac{\sum_{T=k}^{T=l} m_T H_T(50)}{\sum_{T=k}^{T=l} m_T}$$

where $H_T(50)$ is the committed equivalent dose and W_T is the specific weighting factor for the tissues and organs T_i to T_j (the 12 named in Table 2.2, Chapter 2); and m_T is the mass of the remainder tissues, T_k to T_j (given in the note to the Table referred to) and $W_{remainder}$ is the W_T (0.05) assigned to these remainder tissues.

In the exceptional cases in which one of the remainder tissues or organs is calculated to receive a committed equivalent dose in excess of

the highest dose in any of the organs for which a weighting factor is specified, a weighting factor of 0.025 (half of $W_{remainder}$) is applied to that tissue or organ and 0.025 (half of $W_{remainder}$) to the average equivalent dose in the rest of the remainder tissues and organs. The committed effective dose $E(50)$ is computed in this case as:

$$E(50) = \sum_{T=i}^{T=j} w_T H_T(50) + 0.025 \frac{\sum_{T=k}^{T=l} m_T H_T(50) - m'_T H'_T(50)}{\sum_{T=k}^{T=l} m_T - m'_T}$$

$$+ 0.025\ H'_T(50)$$

where m_T is the mass of the remainder tissue or organ in which the committed equivalent dose is calculated to be higher than that in any tissue or organ with a specified W_T, and $H_T(50)$ is the committed equivalent dose in that tissue.

18.3.2 Secondary Limits

In accordance with the ICRP 60 recommendations, the ALI for any radionuclide is obtained by dividing the annual average effective dose limit (0.02 Sv) by the committed effective dose, $E(50)$, resulting from the intake of 1 Bq of that radionuclide.

In ICRP 30, it was pointed out that if the behaviour of any specific material was expected to vary significantly from that of the dosimetric model employed, then alterations should be made in the application of the model when specific data are available to justify such alterations. This advice still applies.

18.3.3 Conclusions of ICRP 61

The effective dose is a rather robust quantity and thus changes in tissue weighting factors have minor numerical effects on the effective dose per unit intake. However, in the absence of any explicit limits on equivalent dose to organs, the weighting factors strongly influence the equivalent dose in organs at intakes corresponding to the ALI. ALIs developed under the 1990 recommendations for the most part are more restrictive; however, the limitation on effective dose does permit intakes which result in committed equivalent dose to bone surfaces (and the kidneys) of the order of 1 Sv. Due to the protracted nature of the exposure

from these internally deposited radionuclides, it is unlikely that the lifetime equivalent dose would be sufficient to result in deterministic effects.

In addition, for the alpha emitting radionuclides, the committed effective dose (or the equivalent dose) as calculated here incorporates a radiation weighting factor of 20 based on potential stochastic effects. This almost certainly overestimates the potential for deterministic effects.

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CHAPTER 19

METHODOLOGIES FOR COMPUTATION OF COMMITTED DOSE EQUIVALENT, ANNUAL LIMIT ON INTAKE AND DERIVED AIR CONCENTRATION

19.1 INTRODUCTION

A brief account is given in this chapter of the methodology for computation of committed dose equivalent (H_{50}), annual limit on intake (ALI) and derived air concentration (DAC). For details, reference may be made to ICRP publication 30, Part 1.

19.2 COMMITTED DOSE EQUIVALENT

The committed dose equivalent H_{50} is given by

$$H_{50} = \sum_i Q_i \overline{D}_{50,i}$$

where $D_{50,i}$ is the total absorbed dose during the 50 years after the intake averaged throughout the specified organ or tissue for each radiation of type i. A specified organ or tissue is one of those designated by ICRP 26 and in the metabolic data for individual elements.

Estimates have been made in ICRP 30 for the committed dose equivalents in a number of target organs from the activity in a given source organ. For each type of radiation i, $H_{50,i}$ in target organ T resulting from radionuclide j in source organ is the product of two factors:

(a) the total number of transformations of radionuclide j in S over a period of 50 years after intake;

(b) the energy absorbed per g in T, suitably modified for quality factor, from radiation of type i per transformation of radionuclide j in S, i.e. for each radiation of type i from radionuclide j

$$H_{50}(T \leftarrow S)_i = Q_i \overline{D}_{50}(T \leftarrow S)_i$$

$$= U_S \times 1.6 \times 10^{-13}\, SEE(T \leftarrow S)_i \times 10^3 \quad \text{Sv}$$

where U_S is the number of transformations of j in S over 50 years following intake; 1.6×10^{-13} is the number of joules in 1 MeV; and $SEE\ (T \leftarrow S)_i$ (in MeV g^{-1} per transformation) is the specific effective energy for radiation type i, suitably modified by quality factor, absorbed in T from each transformation in S and 10^3 is the conversion factor from g^{-1} to kg^{-1}.

Hence,

$$H_{50}(T \leftarrow S)_i = 1.6 \times 10^{-10}\, U_S\, SEE(T \leftarrow S)_i \quad \text{Sv}$$

and for all types of radiations emitted by radionuclide j:

$$H_{50}\ (T \leftarrow S)_j = 1.6 \times 10^{-10} \left[U_S \sum_i SEE\ (T \leftarrow S)_i \right]_j \quad \text{Sv}$$

When the radionuclide has a radioactive daughter j',

$$H_{50}(T \leftarrow S)_{j+j'} = 1.6 \times 10^{-10} \left[\left\{ U_S \sum_i SEE\,(T \leftarrow S) \right\}_j + \left\{ U_S \sum_i SEE\,(T \leftarrow S)_{j'} \right] \right. \quad \text{Sv}$$

In general, for the intake of any mixture of radionuclides, i.e. parent with daughters and/or other radionuclides, H_{50} in target T from activity in source S is given by

$$\sum_j H_{50}(T \leftarrow S)_j = 1.6 \times 10^{-10} \sum_j \left[U_S \sum_i SEE\,(T \leftarrow S)_i \right]_j \quad \text{Sv}$$

where the summation in j is over all the radionuclides involved.

Finally, target T may be irradiated by radiations arising in several sources S. The total value of H_{50} in target T is then given by

$$H_{50,T} = 1.6 \times 10^{-10} \sum_S \sum_j \left[U_S \sum_i SEE\,(T \leftarrow S)_i \right]_j \quad \text{Sv}$$

19.3 CELLULAR DISTRIBUTION OF DOSE

Values of the committed dose equivalent refer to the average value in the specified target tissue. For parts of the GI tract, the target tissue is considered to be the mucosal layer, for the bone the cells lying within 10 μm of bone surfaces, and for skin the basal layer of the epidermis taken to be at a depth of 70 μm. In all other cases the committed dose equivalent refers to the mean value in a target tissue arising from activity in a source organ.

It is recognized that there may be circumstances where the effects produced may be different from those expected from considerations of average dose, e.g. for radionuclides that emit radiations of very short range and which concentrate near sensitive microvolumes. Cases in which the microdistribution of dose is thought to be important are referred to specifically in the metabolic data for the element concerned in ICRP 30.

19.4 SPECIFIC EFFECTIVE ENERGY (SEE)

In the dosimetric data for individual radionuclides given in the Supplements to ICRP 30, values are given for

$$SEE\ (T \leftarrow S) = \sum_i SEE\ (T \leftarrow S)_i$$

for a number of target and source organs. (Values for daughter radionuclides are given separately.) For any radionuclide j, $SEE(\mathrm{T} <\!\!- \mathrm{S})_j$ for target T and source S is given by

$$SEE\ (T \leftarrow S)_j = \sum_i \frac{Y_i E_i A F\ (T \leftarrow S)_i Q_i}{M_T} \qquad \text{MeV g}^{-1} \text{ per transformation}$$

where the summation is over all radiations produced per transformation of radionuclide j in source organ S; Y_i is the yield of radiations of type i per transformation of radionuclide j; E_i (in MeV) is the average or unique energy of radiation i, as appropriate; $AF(\mathrm{T} <\!\!- \mathrm{S})$ is the fraction of energy absorbed in target organ T per emission of radiation i in S. For most organs it is assumed that the energies from alpha particles and electrons are completely absorbed within the source organ. Notable exceptions are mineral bone and the contents of the GI tract. The absorbed fraction of energy from photons is estimated by the use of data on specific absorbed fraction (absorbed fraction per g of target) given in ICRP 23. The absorbed

fractions for fission neutrons quoted in ICRP 30 have been obtained from data given by Dillman and Jones (1975), and Ford *et al.* (1977); Q_i is the quality factor appropriate for radiation of type i; and M_T (in g) is the mass of the target organ.

19.4.1 Decay Schemes

Only the principal modes of decay are listed in the dosimetric data in ICRP 30 and these are given to provide some information as a basis for monitoring rather than as an adequate basis for dosimetry. The yields and energies of internal conversion electrons, of Auger electrons and X-rays, and of beta and positron emitters have been estimated by the methods of Dillman (1979). Data on the decay schemes used in the calculations have subsequently been published separately in ICRP Publication 38, 'Radionuclide transformations: Energy and intensity of emissions' (1983).

19.4.2 Masses of Organs in the Body

The masses of target organs have been taken from ICRP 23.

19.5 NUMBER OF TRANSFORMATIONS IN A SOURCE ORGAN OVER 50 YEARS

The number of transformations of a radionuclide in any organ or tissue during any period of time, U, is the time integral of activity of the radionuclide within that organ or tissue over the stated period of time. The function describing uptake and retention of a radionuclide in the body following its ingestion or inhalation may be very complex and therefore it is convenient to describe the transfer of radionuclides within the body by simple models which facilitate calculation and yet yield sufficiently accurate dose estimates. With certain exceptions (e.g. alkaline earth radionuclides in bone), the models are based on the assumption that the body consists of a number of separate compartments. Any organ or tissue may comprise one or more compartments. Loss of the radionuclide from any compartment is taken to be governed by first order kinetics. Therefore, the retention of an element in any organ or tissue will usually be described by either a single exponential term or the sum of a number of exponential terms, details of which are given in the metabolic data for individual elements in ICRP 30. Exceptions are noted in the metabolic data.

The compartmental models used for the respiratory system and the GI system are described in separate chapters. After a radionuclide has been

inhaled or ingested it will be translocated to the body fluids at a rate determined by the rate constants for the different compartments and by the radioactive decay constant of the radionuclide. Its translocation thereafter to the compartments representing the various organs and tissues of the body is shown schematically in Fig. 19.1. The finite time taken for translocation to the organs and tissues of deposition following entry into the body fluids is represented in the model by transfer compartment *a* , which is assumed to be cleared by first order kinetics with a half-life of 0.25 day, unless otherwise stated in the metabolic data for a particular element. Transformations occurring in the transfer compartment are assumed to be uniformly distributed throughout the whole body of mass 70 kg. Each organ or tissue of deposition is assumed to consist of one or more compartments, and from each of these compartments the radionuclide

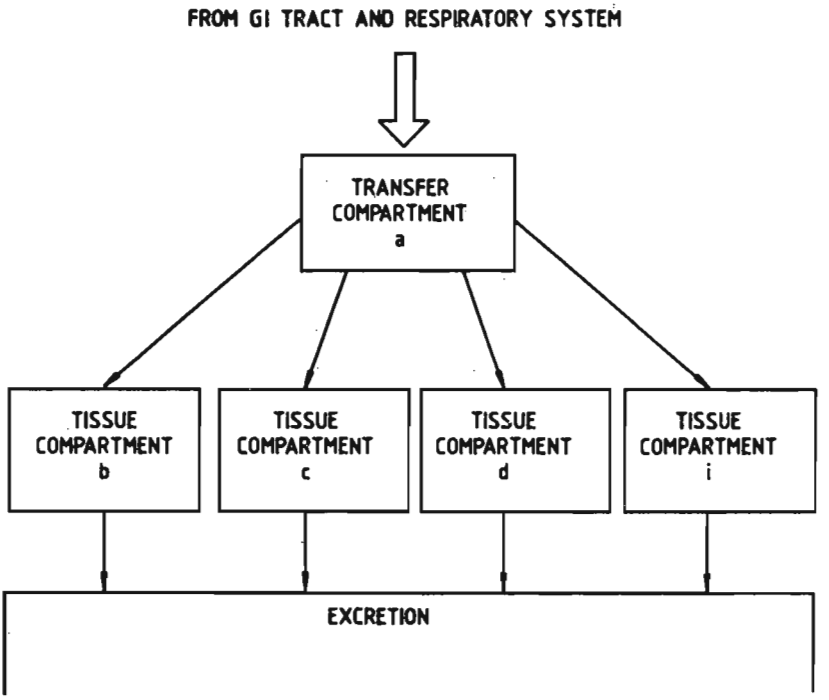

Figure 19.1. Mathematical model to describe the kinetics of radionuclides in the body (from ICRP 30).

is translocated at an appropriate rate to the excretion pathways. For simplicity, it is usually assumed that there is no feedback to the transfer

compartment either from the routes of excretion or from the organ compartments, although it is recognized that transfer to body fluids happens in practice, and thus no estimate is usually made of dose along the routes of excretion. (Because of the above assumption the amount of a radionuclide in a transfer compartment a at any time after inhalation or ingestion cannot be used to estimate the amount of the radionuclide present in body fluids at that time.)

From the above model the activity $q(t)$ in any compartment at time t is derived using the following equations:

In transfer compartment a,

$$\frac{d}{dt} q_a(t) = I(t) - \lambda_a q_a(t) - \lambda_R q_a(t)$$

In tissue compartment b,

$$\frac{d}{dt} q_b(t) = b\lambda_a q_a(t) - \lambda_b q_b(t) - \lambda_R q_b(t)$$

and so on, for any number of compartments,

where $\dot{I}(t)$ is the rate of entry of activity of the radionuclide into body fluids at time t after inhalation or ingestion; λ_a is the clearance rate of stable isotopes of the element from transfer compartment a; b, c, etc. are the fractions of stable isotopes of the element transferred from the body fluids to compartments b, c, etc.; λ_b, λ_c, etc. are the clearance rates of stable isotopes of the element from the compartments b, c, etc. and λ_R is the radioactive decay constant of the radionuclide.

Values of b, c, etc.; λ_b, λ_c, etc. can be derived from the metabolic data for individual elements.

Any exceptions to this general method of deriving the activity in a compartment are noted in the metabolic data for that element, e.g. iodine.

19.6 SUMMARY OF THE MATHEMATICAL TREATMENT FOR SOLUTION TO THE COMPARTMENTAL MODELS

The details of the methods of solution (exact and approximate) are described in ICRP 30. Only a very brief indication of the methodology and the solutions is given in the following.

All the metabolic models used, except for that for the alkaline earths, can be expressed in the form of first order differential equations with

constant coefficients. Most of the models used here do not involve feedback and can be reduced to a linear chain of compartments as described by the system of differential equations of the form:

$$\frac{dq_1}{dt}(t) = I_1(t) - \lambda_1 q_1(t)$$

$$\frac{dq_i}{dt} = I_i(t) + \lambda_{(i-1,i)} q_{(i-1)}(t) - \lambda_i q_i(t) \qquad i = 2 \text{ to } n$$

where $q_i(t)$ is the total quantity of activity in compartment i at any time, $I_i(t)$ is the rate of intake of activity from outside the system into compartment i at time t; $\lambda_{(i-1,i)}$ is the rate constant for transfer of material from compartment $i-1$ to compartment i, and λ_i is the rate constant for loss of material from compartment i.

When evaluating the number of transformations U in the various compartments of the chain, calculations are simplified because a quantity of activity is supposed to be instantaneously deposited in the first compartment of the chain and that, subsequent to this, no further intakes of activity from outside the system occur in any of the compartments of the chain. Thus, taking t to be zero at the time of this single instantaneous intake, $I_i(t)$ may be set equal to zero for all i, $q_i(0)$ may be set equal to the amount of activity instantaneously deposited in the first compartment and $q_i(0)$ may be set equal to zero for all i from 2 to n, i.e. for all other compartments of the chain. It can then be shown (Skrable *et al.*, 1974) that

$$q_i(t) = \left[\left(\prod_{k=1}^{i-1} \lambda_{(k,k+1)} \right) \sum_{k=1}^{i} \left(\frac{q_1(0)\, e^{-\lambda_k t}}{\prod_{\substack{p=1 \\ p \neq k}}^{i} (\lambda_p - \lambda_k)} \right) \right]$$

where

$$\prod_{i=m}^{n} a_i = a_m \times a_{m+1} \times \ldots a_n \qquad \text{if } n \geq m$$

and

$$\prod_{i=m}^{n} = 1 \quad \text{if } m > n$$

The above expression for $q_i(t)$ may be integrated between time zero and time T to yield:

$$U_i(T) = \int_0^T dtq_i(t) = \left[\left(\prod_{k=1}^{i-1} \lambda_{(k,k+1)} \right) \sum_{k=1}^{i} \left(\frac{q_1(0)\,(1-e^{-\lambda_k T})}{\lambda_k \prod_{\substack{p=1 \\ p \neq k}}^{i} (\lambda_p - \lambda_k)} \right) \right]$$

Thus, if $q_i(t)$ is taken to be the activity of a radionuclide in compartment i at time t, and if T is taken to be 50 years, the above expression gives the integrated activity of that radionuclide in the ith compartment of the chain in the 50 years following its intake into the first compartment of that chain. The equation can also be used to determine the integrated activities of the daughter radionuclides in various compartments of the lung and GI tract.

(The equations for $q_i(t)$ and $U_i(t)$ break down in the case of $\lambda_p = \lambda_k$ (p = k). However, in this case it can be shown that $\lim \lambda_p \to \lambda_k [q_i(t)]$ and $\lim \lambda_p \to \lambda_k [U_i(t)]$ still exist since these equations are completely antisymmetric in exchange of λ.)

19.6.1 Illustrative Solution for Integrated Activities in a Two-Chain Compartment

In this case the original equations reduce to:

$$\frac{dq_1}{dt} = -\lambda_1 q_1(t)$$

$$\frac{dq_2}{dt} = \lambda_{(1,2)} q_1(t) - \lambda_2 q_2(t)$$

provided that the boundary conditions discussed above are employed. An example of this might be the distribution of a radionuclide between the transfer compartment and a particular tissue compartment following its instantaneous transfer to the transfer compartment. In this case, $q_1(0)$ will be equal to Q, the amount of activity initially introduced, $q_1(t)$ the activity in the transfer compartment at any time, $q_2(t)$ the activity in the tissue compartment at time t, $\lambda_1 = \lambda_a + \lambda_k$, $\lambda_2 = \lambda_b + \lambda_R$ and $\lambda_{(1,2)} = b\lambda_a$, where λ_a is the rate of loss of the stable element from the transfer compartment, λ_R the radioactive decay constant of the radionuclide, $b\lambda_a$ the rate of translocation from the transfer compartment to tissue compartment b and λ_b the rate of loss from that compartment (Fig. 19.1).

Solution of these equations gives:

$$q_1(t) = Qe^{-(\lambda_a + \lambda_R)t}$$

$$q_2(t) = \frac{b\lambda_a Q}{(\lambda_a - \lambda_b)} \left\{e^{-(\lambda_b + \lambda_R)t} - e^{-(\lambda_a + \lambda_R)t}\right\}$$

The build-up and subsequent decline in activity in the second compartment of the chain is reflected in the expression for $q_2(t)$.

Similarly, we get:

$$U_1(T) = \frac{Q(1 - e^{-(\lambda_a + \lambda_R)T})}{(\lambda_a + \lambda_R)}$$

$$U_2(T) = \frac{b\lambda_a Q}{(\lambda_a - \lambda_b)} \left\{\frac{(1 - e^{-(\lambda_b + \lambda_R)T}}{(\lambda_b + \lambda_R)} - \frac{(1 - e^{-(\lambda_a + \lambda_R)T})}{(\lambda_a + \lambda_R)}\right\}$$

The first term within the bracket represents the integrated activity that would have occurred in the organ, had transfer to it taken place instantaneously. The second term represents a correction for the finite residence time in the transfer compartment.

ICRP 30 discusses details of computation of U values in several special cases, including, in particular, (a) Build-up of radioactive daughters; (b) Inhaled and ingested radionuclides in various regions of the lung and GI tract.

19.7 ANNUAL LIMIT ON INTAKE (ALI)

In the dosimetric data for each radionuclide j, values are given in ICRP 30 for H_{50} per unit activity ingested or inhaled for a number of organs and tissues irradiated to a significant extent. Corresponding values are given for H_{50} per unit intake weighted by the factor W_T. Except in ICRP 30, Part 4, dealing with the transuranics, no values are tabulated for the committed effective dose, but this quantity can be arrived at by adding the weighted dose commitment contributions from each organ, i.e. $\Sigma\, W_T H_{50,T}$ Values are also given for ALI by inhalation and by ingestion of a radionuclide in various chemical forms calculated as described in Chapter 18, i.e. ALI_j is the greatest value of I_j which satisfies both the following inequalities

$$I_j \sum_T W_T (H_{50,T} \text{ per unit intake}) \leq 0.05 \text{ Sv for stochastic effects, and}$$

$$I_j \, (H_{50,T} \text{ per unit intake}) \leq 0.5 \text{ Sv for non-stochastic effects.}$$

In the relationship for stochastic effects, the summation is for all those tissues specified in ICRP 26 (refer to table App. 8(2).1, of Appendix 8(2)) and the five tissues in the remainder which receive the greatest committed dose equivalents. A tissue T is deemed to be irradiated to a significant extent if for that tissue the weighted value of H_{50} per unit intake is greater than 10% of the maximum weighted value of H_{50} per unit intake in any tissue (the 10% rule); that is,

$$W_T H_{50,T} \text{ per unit intake} \geq 0.1 \, (W_T H_{50,T} \text{ per unit intake})_{\max}$$

For any radionuclide only those target tissues that satisfy the above equation are listed. Values of SEE(T <— S) are given for every source organ that contributes 1% or more to the committed dose equivalent in a target tissue which satisfies the above inequality (the 1% rule).

In most cases it is the stochastic limit that determines the ALI, unless the non-stochastic relationship overrides because of the non-stochastic effects on bone surfaces, thyroid or any one of the five tissues in the remainder. This follows as a consequence of the small weighting factor ($W_T \leqslant 0.06$) assigned to these tissues. When the ALI is limited by consideration of non-stochastic effects, the organ or tissue concerned is named in the ICRP 30 tabulations below the value of ALI. In all such

cases the greatest annual intake that meets the stochastic limits is given in parentheses. This value is useful when considering the limitation of exposure from several sources. Values of ALI by ingestion are given in ICRP 30 for each appropriate value of f_1, the fraction of an ingested compound of the element which is absorbed into blood. Values of ALI by inhalation are given for each inhalation class D, W or Y appropriate to various chemical compounds of the element concerned. The revised ALI values for inhalation (based on ICRP 60 recommendations) are given in ICRP 61.

19.8 DERIVED AIR CONCENTRATION (DAC)

As described in chapter 18, the derived air concentration (DAC) for a radionuclide is related to the appropriate ALI by inhalation as follows:

$$DAC_j = ALI_j / (2.4 \times 10^3) \quad \text{Bq m}^{-3}$$

All the values given for DAC and ALI by inhalation are calculated for an aerosol of Activity Median Aerodynamic Diameter (AMAD) of 1 μm. However, values are given in the dosimetric data for the percentage of the committed dose equivalent arising from deposition in the nasal-passage (NP), the tracheo-bronchi (TB) and the pulmonary region (P) of the respiratory system to enable an appropriate correction to be made for aerosols of different AMAD. The method of calculation has been described by ICRP 30 while discussing the respiratory tract model.

DAC values are also given in ICRP 30 for submersion in a radioactive cloud of elemental tritium and of the noble radioactive gases. In the latter case the DAC is generally limited by the non-stochastic dose limit to the skin. (It may be noted that skin is not one of the organs in ICRP 26 for which a specific value for W_T is given.)

19.9 USE AND LIMITATIONS OF THE DOSIMETRIC DATA

19.9.1 Precision of the Data

Values of ALI and DAC are given in ICRP 30 and ICRP 61 to one significant figure; greater precision is hardly justified. Values of *SEE*, U_S and H_{50} per unit intake collected in the Supplements to ICRP 30 are given to two significant figures.

19.9.2 Assumptions Concerning Chemical and Physical Form

As discussed earlier, in the lung model, three general classes of compounds are considered and more than one value for absorption from the GI tract (f_1) is employed where information is available. The values of f_1 listed in ICRP 30 and ICRP 61 may not always be applicable in practice and the user should make his own choice from the values given, interpolating between values where appropriate. Alterations should be made in the application of the model when specific data are available to justify such alterations.

19.9.3 Assumptions Concerning Metabolic Models

The metabolic data given for the different elements may be quite detailed in some cases and supported by experimental evidence from observations on humans, while in other cases they may be more limited. This lack of knowledge about metabolism represents the largest factor of uncertainty in most estimates of committed dose equivalent.

19.9.4 Assumptions Concerning the Exposed Individual

The dosimetric data are calculated for an occupationally exposed adult with the characteristics of the ICRP 23 Reference Man. ICRP does not recommend the use of the data and the models described in ICRP 30 to estimate committed dose equivalents to members of a population, for example from radionuclides in the environment, by adjustment solely on the basis of differences in mass of organs or magnitude of intake. A bibliography is given in ICRP 30 concerning the estimation of committed dose equivalent in people of different ages from intakes of radionuclides.

19.9.5 Assumptions Concerning Chemical Toxicity

ALI values and dosimetric data relate solely to limitations on the radiation doses received by organs and tissues of the body and not to chemical toxicity. Certain elements like uranium may present a greater risk from chemical toxicity than due to radioactivity. For some nuclides of very low specific activity, e.g. ^{115}In, the mass of the material associated with ALI is greater than it would be reasonable to expect a worker might inhale or ingest in any one year. Accordingly, the values of ALI should always be used with circumspection.

19.9.6 Assumptions Concerning Daughter Radionuclides

All the values quoted relate to intake by the specified route of entry into the body of the single radionuclide named. They include an appropriate allowance for the activity of any daughter radionuclides produced in the body as a result of intake of the parent. It is usually assumed that daughter radionuclides produced from their parent within the body stay with and behave metabolically like their parent. Intakes which include both parent and daughter radionuclides should be treated by the general method appropriate to mixtures.

19.9.7 Exposure to a Mixture of Radionuclides in Inhalation, Ingestion and Submersion

Each ALI has been derived by assuming that a worker takes the radionuclide into his body by only one of the specified routes of ingestion or inhalation. In practice, workers may ingest or inhale a mixture of radionuclides, may also be submerged in a cloud of a radioactive, chemically inert, gas or a cloud of gaseous tritium, and may also be exposed to other external sources of radiation. In such circumstances the tabulated values of ALI cannot be used directly and exposures must be limited as described below.

The ALI for a particular radionuclide is derived from a consideration of both stochastic and non-stochastic effects and this is true also of the DAC for submersion in a radioactive cloud. Thus, when considering such cases, the total exposures should be limited by ensuring that the inequalities set out in Chapter 18 are satisfied. However, since it is often more convenient to use the appropriate ALIs and DACs than to compute the various appropriate values of H_{50}, the method for control of exposure using the ALI values is discussed in ICRP 30.

When a worker is exposed to external sources of radiation in addition, the total exposure in any year of practice can be limited by ensuring that both the following conditions are met [ICRP 26; ICRP 1978 Stockholm meeting statement (*Ann. ICRP*, 1978)]:

$$\frac{H_{I,d}}{H_{E,L}} + \sum_j \frac{I_j}{I_{j,L}} \leq 1$$

and

$$\frac{H_{I,s}}{H_{sk,L}} \leq 1$$

where $H_{I,d}$ is the annual deep dose-equivalent index, $H_{I,s}$ is the annual shallow dose-equivalent index, $H_{E,L}$ is the annual limit of the effective dose (50 mSv as per ICRP 26), $H_{sk,L}$ is the annual limit of equivalent dose to the skin (500 mSv as per ICRP 26), I_j is the annual intake of radionuclide j, and $I_{j,L}$ is the annual limit on intake of radionuclide j.

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CHAPTER 20

METHOD OF PRESENTATION OF THE DATA IN ICRP PUBLICATION 30, ITS SUPPLEMENTS AND ICRP PUBLICATION 38

20.1 ICRP PUBLICATION 30 AND ITS SUPPLEMENTS

ICRP 30 Part 1 is divided into two sections. The first section is the main text, running into 62 pages, having 9 chapters and an appendix. It is concerned with the basic principles and methodologies of internal dosimetry. It starts with a glossary of terms and then discusses the basic, secondary and derived limits for the control of internal doses. Methods of computing committed equivalent doses are then described in detail. Then follow the dosimetric models for the respiratory tract, GI tract, bone and submersion in a radioactive cloud. The last chapter discusses the use and limitation of the dosimetric data. The appendix deals with the exact and approximate solutions of the compartmental models used in ICRP 30. The rest of ICRP Part 1, as well as Parts 2, 3 and 4 give relevant metabolic data for each element followed by a table of values of ALI and DAC for radioisotopes of that element having radioactive half-lives greater than 10 min. There is a separate volume giving a comprehensive index.

The metabolic models described are for compounds of a stable isotope of the element. Retention data given in the literature have, where necessary, been corrected for radioactive decay. Because of the considerable variation of GI absorption from individual to individual, values of f_1, the fraction of a stable element reaching body fluids after its entry into the gastrointestinal tract, are given to one significant figure only. A method for correcting the ALI values for particles of sizes other than a value of AMAD = 1 μm is described in the chapter on the respiratory tract model (Chapter 21 in this Handbook) and the required numerical data are given in the Supplements to ICRP 30.

The Supplements give the relevant dosimetric data for radionuclides considered in the main text of ICRP 30. Only abbreviated decay schemes are shown as an aid to monitoring. The full decay schemes can be found in ICRP 38.

20.1.1 Tables of Data

Except for tritium the tables in the Supplements are directly reproduced from the computer print-out used for determining ALI and DAC.

20.1.2 Specific Effective Energy

Values of *SEE* in a number of target organs from transformations arising in a number of source organs are given for the specific radionuclides (and, where appropriate, for any radioactive daughters that are produced from transformations of the specific radionuclide within the body).

The total body is often given as a source organ in a *SEE* table although it is never listed as a source organ in a table of number of transformations. The committed dose equivalents are calculated separately using data for the *U* values in each of certain specific organs and adding the *U* values for all the other organs comprising the rest of the body.

20.1.3 Number of Transformations

Values are given for the number of transformations, *U*, that occur in source organs during the 50 years following intake of unit activity by both ingestion and inhalation.

The source organs considered comprise a complete inventory of those tissues that contain the radionuclide after its entry into the body. 'Other tissue' (with its mass specified in grams in parentheses) is the name given to the rest of the body when it comprises the whole body less those organs and tissues which are mentioned in the metabolic model and metabolic data for an element, and which are also named as source organs.

Transformations in the transfer compartment are assumed to be uniformly distributed throughout all organs and tissues.

The committed dose equivalent in the gonads is taken to be the larger of the corresponding values for the testes and ovaries. The committed dose equivalent for muscle is used for breast.

'Remainder' is the name given to describe collectively all those organs and tissues not mentioned in : (a) the metabolic model; (b) the GI tract model; and (c) the table of weighting factors. The committed dose equivalent assigned to the 'Remainder' is the maximum committed dose equivalent in any target organ or tissue shown in (a), (b) and (c) above.

In the table of committed dose equivalents, a weighting factor of 0.06, 0.12, 0.18, 0.24 or 0.30 is given in parentheses under the committed dose equivalent for the 'Remainder'. This weighting factor is determined by the number of target organs and tissues, up to a maximum of 5, which are not eliminated under the 10% rule or included in (a), (b) and (c) above, which also qualify for a weighting factor of 0.06. If there are no such organs or tissues, then no committed dose equivalent, or weighted committed dose equivalent, is given for the remainder class.

(Author's note: With the exception of the transuranic nuclides discussed in ICRP 30, Part 4, values for the effective dose commitments have not been given in ICRP 30 for any of the radionuclides. The Supplements, however, give the committed equivalent doses to all the target organs that get a substantial dose as well as the committed equivalent doses for each of these organs weighted by the tissue weighting factor appropriate to that organ. By adding these weighted committed equivalent doses one can arrive at the committed effective doses.)

20.2 ICRP PUBLICATION 38

ICRP publication 38, 'Radionuclide transformations: energy and intensity of emissions' (1983) is a Report of a Task Group of ICRP Committee 2 on data used in ICRP 30. It is a volume of 1250 pages, giving data on 817 radionuclides of 98 elements from hydrogen to mendelevium (no data are given for helium, lithium and boron).

It provides nuclear transformation data and associated decay scheme drawings for the radionuclides discussed in ICRP 30; data for a few additional radionuclides (with half-lives less than 10 min) of interest in nuclear medicine are also included. The yields and energies of the various radiations emitted for each radionuclide considered are presented in tabular form. These data and drawings are directly reproduced from the input of a computer code, EDISTR (Dillman, 1980). A brief discussion of the methodology implemented in EDISTR is given in ICRP 38. EDISTR requires as input the basic nuclear decay data information contained in the Evaluated Nuclear Structure Data Files (ENSDF) which are part of the computer data developed and maintained by the Nuclear Data Project at Oak Ridge National Laboratory (and later transferred to the National Center at Brookhaven National Laboratory) from which the radioactive data published in *Nuclear Data Sheets* (Academic Press, New York) are generated (Ewbank and Schmorak, 1978).

There are four columns of information for each radionuclide. The first column identifies the type of radiation. The next column, labelled

$y(i)$, provides the corresponding mean number of events per nuclear transformation. The unit, $(Bq.s)^{-1}$, given for this column is equivalent to transformation^{-1}. In the third column, labelled $E(i)$, the corresponding transition energy in MeV is listed. The fourth column, labelled $y(i)$ x $E(i)$, is the average energy emitted per transformation for the associated radiation type specified in the first column.

At the beginning of a decay table, the modes of decay are given: alpha, electron capture, beta plus, beta minus, isomeric transition, and spontaneous fission.

Except for spontaneous fission, the radiations are placed in 3 categories: (i) gamma rays, X-rays (characteristic X-rays and those associated with Auger transitions) and annihilation quanta; (ii) beta particles, conversion electrons, and Auger electrons; and (iii) alpha particles and recoil nuclei.

At the conclusion of every table there is an indication whether the daughter nuclides are stable or radioactive; when there is more than one daughter nuclide resulting from two or more modes of decay, the branching ratios (i.e. the fraction of the total number of decays proceeding by each mode) are also given.

If a radionuclide decays to an isomeric level in a daughter nucleus, the fractions of the transformations that feed the isomeric level and the ground level are given separately at the conclusion of the data table.

At the conclusion of each data table, the energy emitted per transformation by the listed radiation for each of the 3 major radiation types, as well as the total energy emitted per transformation by all listed radiations, is given.

To keep the tables within reasonable size, radiations that contribute below a percentage cutoff value of energy emitted per transformation have been omitted.

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CHAPTER 21

DOSIMETRIC MODEL FOR THE RESPIRATORY SYSTEM

21.1 INTRODUCTION

The dosimetric model discussed here relates to the inhalation of radioactive aerosols. The special cases of gas and free ion deposition will receive further consideration.

When radioactive aerosols are inhaled, parts of the respiratory system are irradiated. Other organs and tissues are also irradiated both by radiations originating from the lungs and as a result of translocation of inhaled material to body tissues from the respiratory system.

After inhalation of radioactive aerosols the doses received by various regions of the respiratory system will differ widely, depending upon the size of the inhaled material. The dose received by the nasopharyngeal region is neglected since for most particle sizes it is usually small in comparison with doses received by other regions.

The dose delivered to some of the pulmonary lymph nodes by insoluble particles cleared from the pulmonary region may be many times greater than that received by the lung tissues. After careful consideration ICRP has decided that irradiation of the lung is likely to be more limiting than that of lymphoid tissue in such cases. The distribution of doses to cells in the lung from inhaled particles may also be very inhomogeneous. For the induction of malignancy, the hazard of radioactive particles from such inhomogeneous distribution in the lung is likely to be less than that of the same amount of material uniformly distributed in the lung. For purposes of radiological protection, in adults it will be satisfactory to consider the tracheobronchial region, pulmonary region and pulmonary lymph nodes as one composite organ of mass 1000 g to which the ICRP 26 weighting factor of 0.12 applies.

The methods used to calculate U_S and $SEE(T \leftarrow S)$ for parts of the respiratory system and to calculate H_{50} for the composite lung tissue defined above, are described in what follows.

21.2 DEPOSITION AND RETENTION MODEL

The lung model proposed in the ICRP Task Group Report on Lung Dynamics (1966) takes account of particle size and also defines 3 classes of retention which, in part, reflect the chemical form of the aerosol. Additionally, the model provides information on the various routes of elimination from the lungs. The present model is substantially that of the Task Group but with many changes in values of deposition and clearance parameters.

The respiratory system is divided into 3 distinct regions : the nasal passage (N-P), the trachea and bronchial tree (T-B), and the pulmonary parenchyma (P). Deposition is assumed to vary with the aerodynamic properties of the aerosol distribution and is described by the 3 parameters $D_{N\text{-}P}$, $D_{T\text{-}B}$, and D_P, which represent the fractions of inhaled material initially deposited in the N-P, T-B and P regions, respectively. For a log-normal distribution of diameters, which seems typical of aerosols, the pattern of deposition can be related to the activity median aerodynamic diameters (AMAD) of the aerosol (Fig.21.1). Estimates of the committed dose equivalent are for an aerosol with an AMAD of 1 μm. Estimates for other AMADs can be made using the data given in Fig.21.1 as discussed later in this Chapter.

To describe the clearance of inhaled radioactive materials from the lung, materials are classified as D, W or Y which refer broadly to their retention times in the pulmonary region (of the order of days, weeks and years respectively). The classification applies to a range of half-times for D of less than 10 days, for W from 10 to 100 days, and for Y greater than 100 days. The three regions N-P, T-B and P are each divided into 2 or 4 compartments as shown in Fig.21.2. Each of these compartments is associated with a particular pathway of clearance for which the half-time of clearance is T days and the fraction leaving the region by that rate is F. Thus, compartments *a*, *c* and *e* are associated with absorption processes, whereas *b*, *d*, *f* and *g* are associated with particle transport processes, including mucociliary transport, which translocate material to the GI tract. The pulmonary lymphatic system (L) also serves to remove dust from the lungs. It is associated with compartment *h* in the P region of the lungs from which material is translocated to compartments *i* and *j* in the pulmonary lymph nodes. Material in compartment *i* is translocated to body fluids but that in compartment *j* is assumed to be retained there indefinitely. This compartment is considered appropriate only for class Y aerosols; for class D and W aerosols the fraction (F_j) of material entering compartment *j* from compartment *h* is set equal to zero. The clearance of

material from each of the compartments is assumed to be governed by first order kinetics so that each compartment is associated with a clearance constant λ and half-time of clearance $T = 0.693/(86{,}400\lambda)$. The clearance of inhaled material can be described by a set of interlinked first order differential equations (as described for the general methodology in Chapter 19). Equations can also be set up for the transfer of a radionuclide from the lungs directly to body fluids or to the GI tract.

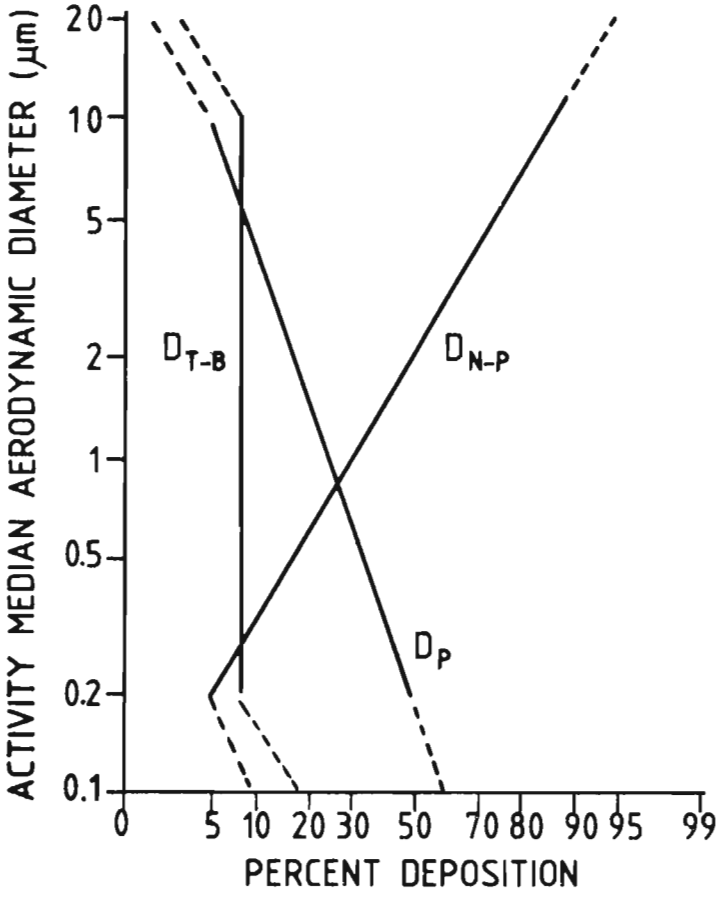

Figure 21.1. Deposition of dust in the respiratory system (from ICRP 30).

Such equations have been used to calculate the number of transformations, U, in the lung and other organs. (For details reference may be made to ICRP 30, Part 1.) Table 21.1 gives approximate expressions

REGION	COMPARTMENT	CLASS D T day	D F	W T day	W F	Y T day	Y F
N-P	a	0.01	0.5	0.01	0.1	0.01	0.01
(D_{N-P} = 0.3)	b	0.01	0.05	0.40	0.9	0.40	0.99
T-B	c	0.01	0.95	0.01	0.5	0.01	0.01
(D_{T-B} = 0.08)	d	0.2	0.05	0.2	0.5	0.2	0.99
	e	0.5	0.8	50	0.15	500	0.05
P	f	N.A	N.A	1	0.4	1	0.4
(D_P = 0.25)	g	N.A	N.A	50	0.4	500	0.4
	h	0.5	0.2	50	0.05	500	0.15
L	i	0.5	1.0	50	1.0	1000	0.9
	j	N.A	N.A	N.A	N.A	∞	0.1

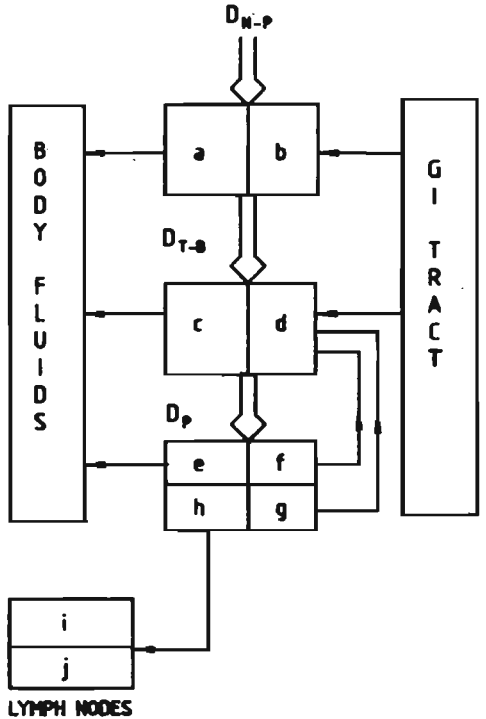

Figure 21.2. Mathematical model to describe clearance from the respiratory system (from ICRP 30).

for the number of transformations in the various compartments following unit intake. Table 22.1 of Chapter 22 dealing with the GI tract model gives similar approximate expressions for the number of transformations in the various regions of the GI tract following ingestion (either directly or transferred from the lungs) of unit activity.

21.3 CALCULATION OF COMMITTED DOSE EQUIVALENT, H_{50}, IN THE LUNG

Neglecting the dose in the N-P region, the target tissue T in the equation for $H_{50,T}$ is the lung comprising the T–B, P and L regions of total mass 1000 g. $H_{50,T}$ has two components – the committed dose equivalent from radioactive material in the lung (i.e. in target tissue comprising T–B, P and L) and the committed dose equivalent from photons arising in other organs and tissues of the body, S.

$$H_{50,\,T} = 1.6 \times 10^{-10} \left[\sum_j U_T^{\,j} \sum_i SEE\ (T \leftarrow T)_i + \sum_S \sum_j U_s^j \sum_i SEE\ (T \leftarrow S)_i \right] \quad \text{Sv}$$

In each case the summation is for all radionuclides, including those daughter nuclides which build up after inhalation of their parent. Values of $U^j_T\ (= U^j_{T\text{-}B} + U^j_P + U^j_L)$ and corresponding values for daughters are given in the dosimetric data for the parent radionuclide j. If they contribute significantly to dose, values of U_S and corresponding values for daughters are also given in the dosimetric data for radionuclide j in ICRP 30.

Values of SEE for any radionuclide j can be arrived at from the equation discussed in Chapter 19. It can be shown that SEE and $AF(T \leftarrow S)_i$ take the following values:

(a) For $SEE\ (T \leftarrow T)_i$, i.e. for radionuclide material in the lung itself, $AF(T \leftarrow T)_i = 1$ for all radiations other than photons and neutrons. For photons and fission neutrons, values of $AF(T \leftarrow T)/M_T$, the specific absorbed fractions, are taken either from ICRP 23 or other sources.

(b) For $SEE\ (T \leftarrow S)_i$, i.e. for radioactive materials in organs and tissues S outside the respiratory system, for all radiations other than photons and neutrons $AF(T \leftarrow S)_i = 0$. For photons and fission neutrons values of $AF(T \leftarrow S)/M_T$, the specific absorbed fractions are similarly computed from ICRP 23 or other sources.

21.4 PARTICLE SIZE CORRECTION

ICRP 30 and its Supplements, in addition to giving dosimetric data for an aerosol of 1 μm AMAD, also give values in parentheses for the fractions $f_{N\text{-}P}$, $f_{T\text{-}B}$ and f_P of the committed dose equivalent in the reference tissue resulting from deposition in the N-P, T-B and P regions respectively. H_{50} for an aerosol of AMAD other than 1 μm may then be estimated by

$$\frac{H_{50}(AMAD)}{H_{50}(1\ \mu m)} = f_{N-P}\frac{D_{N-P}(AMAD)}{D_{N-P}(1\ \mu m)} + f_{T-B}\frac{D_{T-B}(AMAD)}{D_{T-B}(1\ \mu m)} + f_P\frac{D_P(AMAD)}{D_P(1\ \mu m)}$$

where $D_{N\text{-}P}$, $D_{T\text{-}B}$, and D_P are the deposition probabilities in the respiratory region for a given AMAD (Fig.21.1).

If the AMAD is unknown, the values for an aerosol of 1 μm AMAD may be used for dosimetric purposes.

21.5 PROPOSED NEW ICRP LUNG MODEL

The ICRP Task Group on the Lung Model has developed a new (revised) lung model which is expected to be published in the near future. The summary account presented here is based on a paper by Bailey and Birchall (1991).

The new model will be quite comprehensive. Figure 21.3 (Bailey and Birchall, 1991) gives a simplified diagram of the compartment model. The old partitioning of the respiratory tract into N-P, T-B and P regions has been significantly modified and new regions postulated. Newer models and rates have been developed for clearance. A significant feature of the model is the identification of radiosensitive target cells in each region for which the doses will be calculated. The database will be much more broadbased; it will cover breathing at various levels of activity; a wide range of aerosol diameters; oro-nasal and mouth breathing; women and children.

In the new model, the following two major regions are postulated:

(a) *Extrathoracic Region*: This corresponds approximately to the previous N-P region, but also includes the oral passage. It consists of :

(i) anterior nasal passage (ET_1); (ii) naso-oropharynx/larynx (ET_2); and (iii) extrathoracic lymph nodes (LN_{ET}).

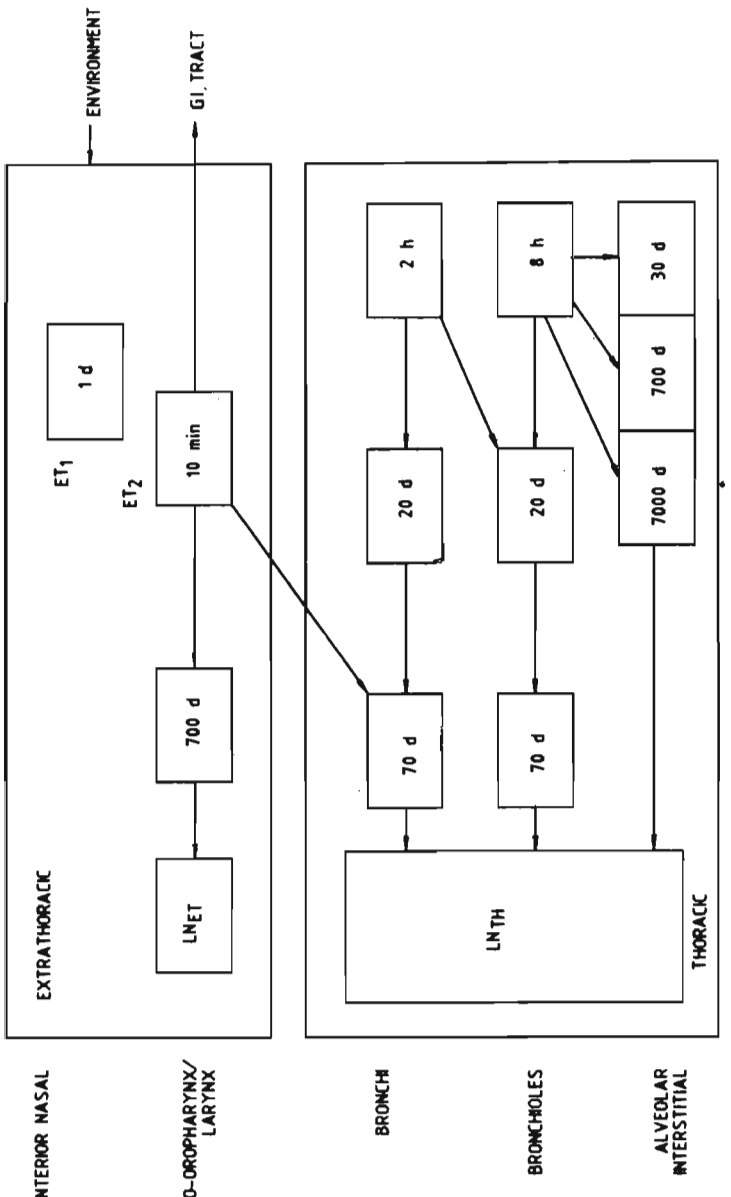

Figure 21.3. Simplified version of proposed new ICRP lung model (reproduced with permission from the National Radiological Protection Board, Chilton).

(b) *Thoracic Region*: This comprises of (i) bronchial (BB) (generations 1 to 8; (ii) tracheobronchial (bb) (generations 9 to 15); (iii) alveolar-interstitial (AI), corresponding to the previous P region; and (iv) thoracic lymph nodes (LN_{TH}). The two regions (i) and (ii) together formed the previous T-B region.

IN ICRP 30, the dose to the N-P region was neglected and the 'mean lung dose' (average dose to T-B + P regions and lymph nodes) was calculated. The mean lung dose approximated the dose to the P region which accounts for most of the mass of the lungs. Only for radon daughters was the TB region considered to be more sensitive per unit mass and taken into account.

In the new model, for each region, target cells with higher radiosensitivity have been identified (basal cells in the ET region; basal and secretory cells in the BB region, and secretory cells in the bb region). 'Regionally-adjusted' doses are obtained for each organ (i.e. the thoracic and extrathoracic airways). The dose calculated to each region is multiplied by a 'relative sensitivity' factor (0.95 for ET_2; 0.8 for BB; 0.15 for bb; 0.05 for ET_1 and AI; 0.001 for LN_{ET} and LN_{TH}). The adjusted doses to the individual organs are summed to obtain the regionally-adjusted dose for each organ, which is then multiplied by the organ weighting factor, W_T, to obtain the contribution to the effective dose. W_T for extrathoracic and thoracic regions are taken to be 0.025 and 0.12 respectively as per ICRP 60.

A compartment model for mechanical transport of particles to the GI tract and lymph nodes (taken to be the same for all materials) has been developed, in addition to the translocation (absorption) of materials into blood (Fig.21.3).

It is assumed that the rate of translocation to blood is the same in all regions. Where specific rates are not available, the following defaults are proposed for the earlier D, W and Y classes respectively:

(a) Fast (F): clearance half-time 10 min.;

(b) Moderate (M): clearance half-times of 3 d for 50% of the total and 100 d for the rest; and

(c) Slow (S): clearance half-time of 7000 d.

Whereas D, W and Y define overall clearance, F, M and S refer only to translocation from blood; mechanical transport rates to the GI tract and lymph nodes are the same for all 3 types, viz. 100 d.

Bailey and Birchall discuss an illustrative case of the implications of the new model for a 1 μm aerosol of ^{239}Pu of types M and S. It is found that for class W material (type M), the committed equivalent doses to red marrow, bone surfaces, liver and gonads are nearly the same in both models; but for lungs the new model gives a regionally adjusted lung dose 5 times that of the mean lung dose of the ICRP 30 model. (This is because of the high relative sensitivity of the BB region which receives the highest dose.) On the other hand, for class Y (type S) material, the lung doses come out to be nearly equal while the doses to red marrow, bone surfaces, liver and gonads as per the new model are twice those of the ICRP 30 model.

The weighted committed equivalent doses ($W_T.H_{50,T}$) and effective doses have also been computed using the ICRP 30 model but with the organ weighting factors recommended by ICRP 60. (W_T for bone surfaces is 0.01 as per ICRP 60 as against 0.03 given by ICRP 26). The weighted committed equivalent doses to all organs are nearly the same for both W and Y classes in the two models (except for the lung dose in W class, where the new model gives 5 times the ICRP 30 value). However, the effective dose commitments are nearly the same in both the models, although there is a shift in weighted organ doses, with irradiation of the respiratory tract making a greater contribution.

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Table 21.1: Approximate expressions for the number of transformations in the various compartments of the lung following inhalation of 1 Bq of activity

(from ICRP 30, Part 1)

Compartment	Number of Transformations	Compartment	Number of transformations
a	$\frac{D_{N-P}\ F_a}{\lambda_a + \lambda_R}$	b	$\frac{D_{N-P}\ F_b}{\lambda_b + \lambda_R}$
c	$\frac{D_{T-B}\ F_c}{\lambda_c + \lambda_R}$	d	$\left\{\left[\frac{D_{T-B}\ F_d}{\lambda_d + \lambda_R} + \frac{D_p}{\lambda_d + \lambda_R}\right] \times \left[\frac{\lambda_f F_f}{\lambda_f + \lambda_R} + \frac{\lambda_g F_g}{\lambda_g + \lambda_R}\right]\right\}$
e	$\frac{D_p\ F_e}{\lambda_e + \lambda_R}$	f	$\frac{D_p\ F_f}{\lambda_f + \lambda_R}$
g	$\frac{D_p\ F_g}{\lambda_g + \lambda_R}$	h	$\frac{D_p\ F_h}{\lambda_h + \lambda_R}$
i	$\frac{D_p F_h \lambda_h F_i}{(\lambda_i + \lambda_R)\ (\lambda_i + \lambda_R)}$	j	$\frac{D_p F_h \lambda_h F_j (1 - e^{-\eta\lambda_R})}{(\lambda_h + \lambda_R)\lambda_R}$

CHAPTER 22

DOSIMETRIC MODEL FOR THE GASTROINTESTINAL TRACT

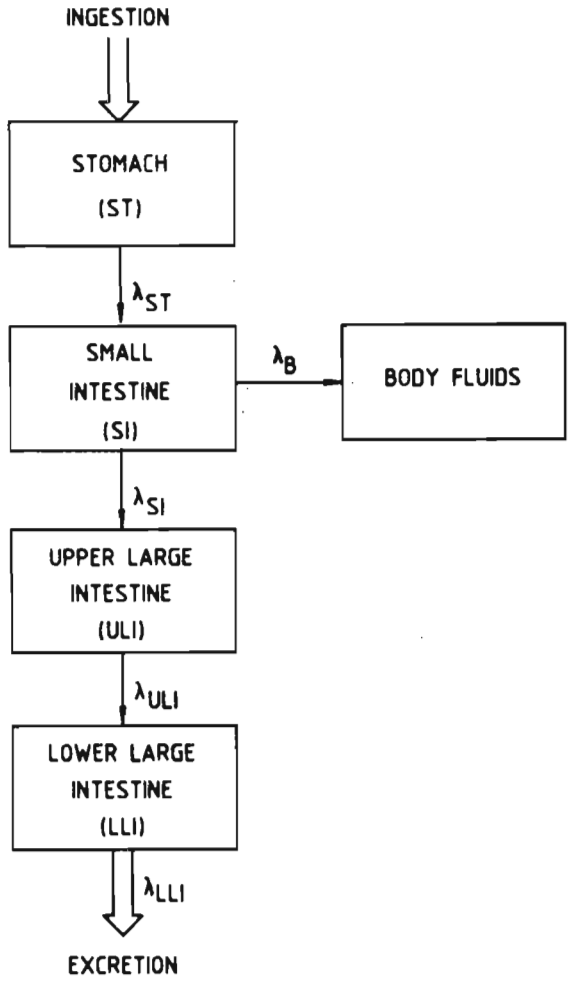
22.1 INTRODUCTION

Methods are described in ICRP 30 for calculating U_S, the number of transformations in source organ over the 50 years following intake and *SEE(T <— S)*, the specific effective energy absorbed in target organ T from radiations originating in source organ S, for sections of the GI tract following ingestion of a radionuclide. These sections of the GI tract, viz. stomach, small intestine, upper large intestine and lower large intestine, are treated as separate target tissues. Organs and tissues of the body other than the GI tract will also be irradiated by that fraction of the radionuclide which enters the body fluids, and also, in some cases, by photon irradiation from material retained in the GI tract. The general methods used to calculate $H_{50,T}$ following entry of a radionuclide into body fluids have been described in Chapter 19. The method used to calculate the activity of an ingested radionuclide or its radioactive daughters, transferred to body fluids from the GI tract, is discussed in what follows.

22.2 DOSIMETRIC MODEL

The dosimetric model is based on the biological model developed by Eve (1966). For purposes of radiological protection the GI tract is taken to consist of the 4 sections shown schematically in Fig.22.1.

Each of these sections is considered as a single compartment and translocation from one compartment to the next is taken to be governed by first order kinetics. We can then set up a set of first order differential equations on the basis of the general methodology discussed in Chapter 19. In Fig.22.1, $\lambda_B q_{SI}(t)$ is the rate of transfer of activity to body fluids from the small intestine, assumed to be the only site of absorption from the GI tract to body fluids. (Account has to be taken, where necessary, of the entry of activity to the GI tract from the respiratory system.) The value of λ_B can be estimated from f_1, the fraction of a stable element reaching body fluids through ingestion using the relation:



SECTION OF GI TRACT	MASS OF WALLS (g)	MASS OF CONTENTS (g)	MEAN RESIDENCE TIME (day)	λ day^{-1}
Stomach (ST)	150	250	1/24	24
Small Intestine (SI)	640	400	4/24	6
Upper Large Intestine (ULI)	210	220	13/24	1.8
Lower Large Intestine (LLI)	160	135	24/24	1

Figure 22.1. Mathematical model to describe the kinetics of radionuclides in the gastrointestinal tract (from ICRP 30).

$$\lambda_B = \frac{f_1 \lambda_{SI}}{1 - f_1}$$

Values of f_1 are given in the metabolic data for the individual elements. Where a value of $f_1 = 1$ is given it is assumed that the radionuclide passes directly from the stomach to body fluids and does not pass through other sections of the GI tract. The activity transferred to body fluids is the integral over 50 years of $\lambda_B q_{SI}(t)$. The method of calculation is described in the Appendix to ICRP 30, Part 1.

22.3 CALCULATIONS OF COMMITTED DOSE EQUIVALENT, H_{50}, TO SECTIONS OF THE GI TRACT

$SEE(T \leftarrow S)$, for any radionuclide i, is given by

$$SEE\,(T \leftarrow S)_i = \sum_i \frac{Y_i E_i A F\,(T \leftarrow S)_i Q_i}{M_T} \qquad \text{MeV g}^{-1} \text{ per transformation}$$

where Y_i is the yield of radiations of type i per transformation of radionuclide j; E_i (in MeV) is the average, or unique, energy of radiation i; $AF(T \leftarrow S)_i$ is the average fraction of energy absorbed in T from radiation i arising in S; Q_i is the quality factor appropriate for radiation i; and M_T (in g) is the mass of the target organ. $H_{50,T}$ is estimated for the walls of each section of the GI tract and it is convenient to consider values of SEE for non-penetrating (np) and penetrating (p) radiations separately.

For both p and np radiations, $H_{50,T}$ is calculated for the mucosal layer of each section of the GI tract. For p radiations the average dose to the walls of the tract is used as a measure of the dose to the mucosal layer. Thus, the value of SEE for any target section of the GI tract and for all emissions from radionuclide j within the tract is given by the following expression:

$$SEE = \sum_{np} \frac{Y_{np} E_{np} Q_{np} AF\,(ML \leftarrow T)_{np}}{M_T^{\mu L}} + \sum_S \sum_p \frac{Y_p E_p Q_p AF\,(W \leftarrow S)_p}{M_T^W}$$

where Y_{np} is the yield of non-penetrating radiations per transformation of the radionuclide; E_{np} (in MeV) is the average or unique energy of the non-penetrating radiation; Q_{np} is 20 for recoil atoms, fission fragments and alpha particles, and 1 for electrons;

$$\frac{AF(M_L \leftarrow T)_{np}}{M_T^{ML}}$$

is the specific absorbed fraction for the mucosal layer of the section of the GI tract under consideration and is taken to be equal to be $0.5(1/M^c{}_T).v$, where $M^c{}_T$ (in g) is the mass of the contents of that section of the GI tract (Fig. 22.1) and v is a factor between 0 and 1 representing the degree to which these radiations penetrate the mucus. The factor 0.5 is introduced because the dose at the surface of the contents will be approximately half that within their volume for np radiations; v is taken to be unity for beta particles, zero for recoil atoms and 0.01 for alpha particles and fission fragments. Although the latter factor is clearly arbitrary, there is experimental evidence that its use is warranted (Sullivan *et al.*, 1960); $M^w{}_T$ (in g) is the mass of the walls of the target section concerned (Fig. 22.1); Y_p is the yield of penetrating radiations per transformation of the radionuclide; E_p (in MeV) is the average or unique energy of the penetrating radiation; Q_p is 1 for photons and 10 for fission neutrons; and $AF(W \leftarrow S)_p$ is the fraction of the energy of a photon or fission neutron emitted in the source organ S which is absorbed in the walls of the target section concerned.

Values of $AF(W \leftarrow S)_p/M^w{}_T$, the specific absorbed fractions for photons, are given in ICRP 23. Corresponding values for fission neutrons have been derived from data by Dillman and Jones (1975) and Ford *et al.* (1977).

For the general case of a number of radionuclides, including the case of an ingested parent together with its daughters produced in the tract, H_{50} is derived by the summation of U_S and *SEE* over all radionuclides and source organs, as described in Chapter 19. In the dosimetric data for each radionuclide presented in the Supplements to ICRP 30, values of U_S are given for the ingested parent and for its radioactive daughters produced in the various sections of the GI tract. *SEE* values are also given for the various sections of the GI tract as target organs with the contents of these sections and other organs and tissues of the body as source organs.

Table 22.1 gives approximate expressions for the number of transformations in the various regions of the GI tract following ingestion of 1 Bq of activity. (The symbols are defined in Fig. 22.1.)

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Table 22.1: Approximate expressions for the number of transformations in the various compartments of the gastrointestinal tract following ingestion of 1 Bq of activity
(from ICRP 30)

Region	Number of transformations
Stomach	$\frac{1}{\lambda_{ST} + \lambda_R}$
Small intestine	$\frac{\lambda_{ST}}{(\lambda_{ST} + \lambda_R)(\lambda_{SI} + \lambda_B + \lambda_R)}$
Upper large intestine	$\frac{\lambda_{ST}\lambda_{SI}}{(\lambda_{ST} + \lambda_R)(\lambda_{SI} + \lambda_B + \lambda_R)(\lambda_{ULI} + \lambda_R)}$
Lower large intestine	$\frac{\lambda_{ST}\lambda_{SI}\lambda_{ULI}}{(\lambda_{ST} + \lambda_R)(\lambda_{ST} + \lambda_B + \lambda_R)(\lambda_{ULI} + \lambda_R)(\lambda_{LLI} + \lambda_R)}$

CHAPTER 23: SECTION 1

DOSIMETRIC MODEL FOR BONE

23(1).1 INTRODUCTION

Bone dosimetry is both important and complex. Historically, the incidence of osteogenic sarcomas among radium dial painters led to interest in bone dosimetry in the early decades of the present century. The widespread use of plutonium in the nuclear industry, as well as its occurrence in fallout from nuclear weapon tests, intensified the importance of bone dosimetry. Several ICRP publications deal with the metabolism of bone-seekers (mainly radium and the transuranics) as well as bone dosimetry. Among them are: ICRP publication 2, 'Report of Committee II on Permissible Dose from Internal Radiation' (1959) (which has been superseded by ICRP publication 30), ICRP publication 11, 'A review of the radiosensitivity of tissues in bone' (1968), ICRP publication 19, 'The metabolism of compounds of plutonium and other actinides' (1972), ICRP publication 20, 'Alkaline earth metabolism in adult man' (1973), ICRP publication 30, 'Limits for intake of radionuclides by workers. Part 1' (1979), and ICRP publication 48, 'The metabolism of plutonium and related elements' (1986). ICRP publication 56, 'Age-dependent doses to members of the public from intake of radionuclides, Part I' (1989) has a section describing a new biokinetic model for actinides.

The contents of ICRP 11 have already been summarized in Chapter 11, Section 3. This Section 1 of Chapter 23 discusses the ICRP 30 methodology for bone dosimetry. Section 2 of this chapter summarizes the contents of ICRP 20, while section 3 summarizes the contents of ICRP 19 and ICRP 48. Section 4 gives a brief account of the salient features of the new bone model for actinides discussed in ICRP 56.

The cells at carcinogenic risk in the skeleton are the haematopoietic stem cells of marrow, and among the osteogenic cells, particularly those on endosteal surfaces, and certain epithelial cells close to bone surfaces. The haematopoietic stem cells in adults are assumed to be randomly distributed predominantly throughout the haematopoietic marrow cells within trabecular bone. Therefore committed equivalent doses to red marrow (RM)

are calculated as the average over the tissue which entirely fills the cavities within trabecular bone. For the osteogenic tissue on endosteal surfaces and epithelium on bone surfaces (BS) the committed equivalent doses are calculated as an average over tissue up to a distance of 10 μm from the relevant bone surfaces. The endosteal area is taken as 12 m^2, half being associated with trabecular bone and half with cortical bone. (The area of epithelium on bone is comparatively smaller.) The mass of the 10 μm thick area covering this 12 m^2 is 120 g. The mass of active red marrow in cavities within the trabecular bone is 1.5 kg, the mass of trabecular bone 1.0 kg, and the mass of cortical bone 4.0 kg. For all radionuclides except gamma emitters, the source tissue S will normally be cortical and trabecular bone (ICRP 11, 20, 23, 26).

ICRP 20 assumes that isotopes of alkaline earths with half-lives greater than 15 days can be considered to be uniformly distributed throughout the volume of bone, while those with half-lives less than 15 days are considered to be distributed on bone surfaces since they are unlikely to be buried far into the volume of bone by bone deposition before they decay.

23(1).2 CALCULATIONS OF COMMITTED EQUIVALENT DOSES, H_{50}, TO CELLS ON BONE SURFACES AND ACTIVE RED MARROW

The general formulae for the calculation of the committed equivalent doses have been discussed in Chapter 19. Bone dosimetry involves estimates of U_S for trabecular and cortical bone, and estimates of $AF(T \leftarrow S)_i$ for all radiations, radionuclides, source organs and target tissues.

Six broad classes can be considered, as discussed in the following sub-section. A radionuclide may belong to more than one class (e.g. beta and gamma emitter), and $AF(T \leftarrow S)_i$ is to be calculated separately for each case.

23(1).2.1 Estimates of Absorbed Fractions in Skeletal Tissues

A summary of values of absorbed fractions for the various cases is given in Table 23(1).1. They are average representative values chosen by ICRP based on conclusions derived from the work of Mays and Sears (1962), Thorne (1977), Spiers and co-workers (Spiers, 1968, 1969, 1974a, b; Whitwell and Spiers, 1976).

23(1).2.1.1 Photon Emitters

S is any organ containing the radionuclide and T is either BS or RM. Values of $AF(T \leftarrow S)/M_T$ are given in ICRP 23 for target tissues skeleton (taken here for BS) and RM.

23(1).2.1.2 Alpha Emitters Uniformly Distributed throughout the Volume of Mineral Bone

It is assumed that the radionuclide is uniformly distributed both in cortical and trabecular bone but not necessarily in equal concentrations. Because of the short range of alphas, surfaces of trabecular bone can be considered essentially infinite and flat for dosimetric purposes.

The main contribution to the dose to bone surface is from the activity in trabecular bone. Since part of the 120 g of tissue on bone surfaces of interest lies on the Haversian canals (diameter ~ 50 μm) and some cross-fire is likely to occur even for alpha particles, there will be an additional small contribution to the dose to bone surfaces from the activity in cortical bone.

The contribution to the RM dose from cortical bone is taken to be zero since active red marrow is considered to be entirely within the marrow cavities of trabecular bone.

23(1).2.1.3 Alpha Emitters on Bone Surfaces

The assumption of uniform spread of the source in an infinitely thin layer over relevant bone surfaces will overestimate the dose to BS and RM because it disregards burial of radioactive deposits by the deposition of new bone mineral. If effects of cross-fire are neglected, the absorbed fraction in BS from cortical bone will be equal to that from trabecular bone.

Considering the surfaces of trabeculae as infinite planes (and assuming them to be sufficiently far apart to make the effects of cross-fire negligible), by simple geometric arguments we can see that the absorbed fraction in RM from the source in trabecular bone is 0.5. As in the case of volume distribution, the absorbed fraction in RM from cortical bone is also taken to be zero.

23(1).2.1.4 Beta Emitters Uniformly Distributed throughout the Volume of Mineral Bone

Spiers and co-workers (Spiers, 1968, 1969, 1974a,b; Whitwell and Spiers, 1976) have given the methodology for calculating the dose rate D_0 to a small tissue-filled cavity in an infinite extent of mineral bone, uniformly contaminated at a level of unit activity per unit mass, as well as D_1 to D_4, the dose rates for various source and target organs, viz. *BS <—TB*, *BS <—CB*, *RM <—TB*, and *RM <—CB*, where TB and CB stand for trabecular bone and cortical bone respectively. We can then calculate the ratios $P_j = D_j/D_0$, where j = 1 to 4; these quantities are independent of the level to which the bone is contaminated. Table 23(1).2 gives the values of P_j for typical radionuclides.

It can be shown that

$$AF(T \leftarrow S)_i = (kM_T P_j)/M_S,$$

where M_S and M_T are the masses of source and target organs, and k is the ratio of stopping powers of the electrons in soft tissue and bone. k is taken to be 1.07 for electrons over a wide range of energies E (Berger and Seltzer, 1966). Average representative values of E for dosimetric purposes are shown in Table 23(1).2.

Since the main contribution to doses in red marrow arises from activity in trabecular bone, we can neglect the contribution to the dose from beta activity in cortical bone, irrespective of the beta energy.

23(1).2.1.5 Beta Emitters on Bone Surfaces

(a) For beta emitters of average energy less than 0.2 MeV, the average range is similar to that of alpha particles, and the same value of absorbed fractions as for alpha particles can be used.

(b) For higher average beta energies, the average range is many times the linear dimensions of a marrow cavity or trabeculum, and the values chosen for the *AF* are shown in Table 23(1).1.

23(1).2.1.6 Fission Fragments and Recoil Atoms

The range of fission fragments in tissue is around 20 μm (Green *et al.*, 1977) and absorbed fraction values for alpha particles can be used.

The energies of recoil atoms are negligible in comparison with the primary emissions and are disregarded for purposes of bone dosimetry.

23(1).2.2 Number of Transformations in Trabecular and Cortical Bone

Except for the alkaline earths for which a metabolic model has been developed in ICRP 20, it is difficult to calculate U values for trabecular bone and cortical bone exactly; nevertheless estimates of these quantities can be made from a knowledge of the U value for mineral bone as a whole.

(a) If a radionuclide is deposited uniformly on all bone surfaces and is removed at the same rate from both cortical and trabecular bone, then, since the surface areas of cortical and trabecular bone are assumed to be equal,

$$U_{\text{Trabecular bone}} = U_{\text{Cortical bone}} = 0.5U_{\text{Mineral bone}}.$$

(b) If a radionuclide redistributes, in a time short compared with both its radioactive half-life and its time of residence in bone, such that it uniformly contaminates all bone mass, then:

$$(U_{\text{Trabecular bone}})/(U_{\text{Cortical bone}}) \sim (M_X)/(M_Z)$$

where M_X is the mass of trabecular bone (1 kg) and M_Z is the mass of cortical bone (4 kg).

For radioisotopes of the alkaline earths, values of U for cortical and trabecular bone can be obtained directly from the retention functions given in ICRP 20. ICRP 30 also discusses the case of radioactive daughters produced in the body from decay of the parent radionuclide.

Knowing the values of U and SEE (the latter calculated from the AF), the committed dose equivalents can be computed separately for the different source and target tissues for photon emitters, alpha and beta emitters, as appropriate, from the formulae given in Chapter 19.

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Table 23(1).1: Summary of recommended absorbed fractions

(from ICRP 30, Part 1)

Source organ	Target organ	Class of radionuclide				
		Alpha emitters		Beta emitters		
		Uniform in volume	On bone surfaces	Uniform in volume	On bone surfaces >0.2 MeV	On bone surfaces < 0.2MeV
TB	BS	0.025(a)	0.25(d)	0.025	0.025	0.25
CB	BS	0.01(b)	0.25(e)	0.015	0.015	0.25
TB	RM	0.05(c)	0.5	0.35	0.5	0.5
CB	RM	0.0	0.0	0.0	0.0	0.0

TB: Trabecular Bone BS: Bone Surface CB: Cortical Bone RM: Red Marrow

(a): Range: 0.018 for 3 MeV Alphas to 0.032 for 8 MeV Alphas; (Mays and Sears, 1962; Thorne, 1977); (b): Range: 0.006 to 0.020; (Thorne, 1976; Spiers, 1974a); (c): Range 0.02 for 3 MeV Alphas to 0.09 for 8 MeV Alphas; (Mays and Sears, 1962; Thorne, 1977); (d): Range 0.43 for 3 MeV Alphas to 0.15 for 8 MeV Alphas; (Mays and Sears, 1962; Thorne, 1977); and (e): Taken as the same for *AF(BS <— TB)*.

Table 23(1).2: Values of the absorbed dose ratio, P_j, for bone surface and red bone marrow from beta-emitting radionuclides uniformly distributed throughout the volume of mineral bone

[from ICRP 30, Part 1 (based on Spiers, 1974a; Whitwell and Spiers, 1976).]

Nuclide	E (MeV)	P_1	P_2	P_3	P_4
^{45}Ca	0.08	0.21	0.42	0.11	0.00
^{90}Sr	0.20	0.21	0.47	0.22	0.02
^{89}Sr	0.55	0.19	0.47	0.26	0.04
^{90}Y*	0.93	0.16	0.45	0.27	0.06

* Assumed to be uniformly distributed throughout the volume of mineral when it is produced from its parent ^{90}Sr.

CHAPTER 23: SECTION 2

ALKALINE EARTH METABOLISM IN ADULT MAN

23(2).1 ICRP 20 MODEL FOR ALKALINE EARTH METABOLISM

ICRP publication 20, 'Alkaline earth metabolism in adult man' (1973) is a report prepared by a Task Group of Committee 2 of ICRP. A new model based on products of power and exponential functions: (i) relates radioisotope retention mechanisms to bone formation, resorption, diffusion, and exchange in compact and cancellous bone; (ii) represents, within 20%, most existing microscopic and macroscopic data for stable or radioactive isotopes; and (iii) facilitates calculation of dose to endosteal or marrow cells following any schedule of radioisotope intake by any route.

The report starts with a summary giving the model, the list of data, results, intended uses, estimate of accuracy, and comparison with existing models. Important parts of the summary are reproduced below.

23(2).1.1 The Model

A new whole body retention function is presented as follows:

$$R = (1-p)\,e^{-mt} + p\varepsilon^{b}\,(t+\varepsilon)^{-b}\left[\beta\,e^{-r\lambda t} + (1-\beta)\,e^{-\sigma r\lambda t}\right]$$

where R is the fraction of the injected activity remaining in the body following a single intravenous injection (not including radioactive decay), ε is a small time (0.3-3 days), related to the turnover of an initial pool, b is power function slope (0.1-0.5), related to diffusion of activity from bone to blood and excretion of part of that activity from the body, λ is the rate of apposition and resorption in compact bone (2.5%/year), σ is the ratio of the turnover rates of cancellous and compact bone (4), β is the fraction of bone volume activity deposited in compact bone (~ 0.5), r is a factor which corrects for redeposition of activity in new bone at sites of resorption long after injection (0.83-0.99), m is the rate constant of a small early exponential in R (0.1-0.8 day^{-1}), p is the fraction of R not in the early exponential (0.6 - 0.8), and t is time (from 0 to any time after injection).

This is a six-parameter model of bodily retention because β and r follow from the other parameter values. The early exponential term is not associated with a particular physiological process. The time derivative and integral of R are used together with additional parameters to generate consistent internal retention functions for bone surface and bone volume, new and old compact and cancellous bone, blood, and soft tissue. The bone functions explicitly separate loss of activity by diffusion (diminution) from that by resorption. All the retention functions are assumed to be age-invariant, so that time integrals may be used to simulate the results of any schedule of radioactive intake into the blood, whether from injection, lung, or diet. The model predictions extend from 15 min to 100 y after intake.

Although there are not nearly enough human data to verify all portions of the eight functions for each of the four alkaline earths (Ca, Sr, Ba and Ra), the calculations of the model are internally consistent to within 1-3% and the model has been found to fit within 20% most of the existing microscopic and macroscopic data for the turnover of bone and the retention of radioactive and non-radioactive isotopes of these elements following either single or continuous intake. However, some of the predictions of the model - particularly those as yet unsupported by data - may be accurate by a factor of 2 only.

The model is useful both for integrating data from different experiments into a single logical structure and for calculating radiation dose to special groups of cells such as osteocytes, endosteal or marrow cells in compact or cancellous bone.

23(2).1.2 Terminology

23(2).1.2.1 Bone

Bone is calcified matrix, comprising organic matrix, bone crystals, an amorphous mineral component (Harper and Posner, 1966), and the water within the organic matrix and within lacunae and canaliculi but *not* the spaces within the blood-carrying Haversian and Volkmann canals and does not include bone marrow or cartilage. Its density is taken as 2.0 g/cm^3 (Robinson, 1964), and its calcium content 0.4 - 0.5 g/cm^3. The total mass of bone in the Standard Man (now called Reference Man) is taken as 5000 g and the total calcium content 1000 g.

This definition of bone corresponds to the anatomical entity, bone, as used by pathologists. It is also appropriate for dosimetry, because lacunae and canaliculi are much smaller than the ranges of alpha or beta particles, whereas Haversian or Volkmann canals often are not smaller than particle ranges.

23(2).1.2.2 Bone Surface

It includes the endosteal and periosteal surfaces of cortical bone, the surfaces of Haversian and Volkmann canals, the surfaces of resorption cavities, and the surfaces of trabeculae, but *not* the surfaces of lacunae and canaliculi. The total bone surface in the Standard Man (Reference Man) is 10 m^2, half in compact bone and the other half in cancellous bone (Lloyd and Hodges, 1971). This definition corresponds to the surface currently postulated as the primary site of osteosarcoma induction (ICRP 11, 1968).

23(2).1.2.3 Bone Volume

Bone volume is *bone* as bounded by bone surface. It includes the volume of lacunae and canaliculi but does *not* include the marrow or the space within the Haversian or Volkmann canals.

23(2).1.2.4 Cancellous Bone

Cancellous bone has an average surface/volume ratio of 120 cm^2/cm^3 (Lloyd and Hodges, 1971) and includes 20% of bone volume, bone mass and bone calcium. Both for cancellous and compact bone, the density is 2.0 g/cm^3, and the calcium content 0.4-0.5 g/cm^3. The whole of vertebrae are associated with cancellous bone.

23(2).1.2.5 Compact Bone

The average surface/volume ratio is 30 cm^2/cm^3 (Lloyd and Hodges, 1971). It includes 80% of bone volume, bone mass and bone calcium. The entire shafts of long bones are associated with compact bone.

23(2).1.2.6 New and Old Bone

New bone refers to all bone after time zero, the time of injection. Old bone refers to all bone existing at time zero, the time of injection.

23(2).1.2.7 Resorption

It is the local reduction of bone volume due to removal (by osteoclasts) of bone mineral and organic matrix from bone surface (Marshall, 1969). Dosimetrically, resorption reduces the volume of bone at risk without changing the activity per gram of remaining bone.

23(2).1.2.8 Diminution

This is any process in bone volume by which alkaline earths transfer from bone to blood without resorption (Marshall, 1969). It is believed to be due to a slow diffusion of ions from the calcified matrix to canaliculi followed by rapid transfer to blood vessels (Marshall and Onkelnix, 1968). Dosimetrically, diminution reduces the activity per gram of bone at risk without decreasing its volume.

23(2).1.2.9 Apposition

It refers to the formation of bone at bone surfaces and involves a local increase in bone volume (by the action of osteoblasts) (Marshall, 1969). We can speak of volume and linear apposition rates, expressed as % per year and μm/day respectively.

23(2).1.2.10 Addition

It is the process by which alkaline earths transfer from blood to bone and remain longer than time *t*. It comprises apposition and augmentation (Marshall, 1969).

23(2).1.2.11 Augmentation

It is the process by which alkaline earths transfer from blood to bone without apposition and remain longer than time *t*. It is believed to be due to rapid transfer of ions from blood up canaliculi followed by slow diffusion into calcified matrix.

23(2).1.2.12 Haversian System (Osteon)

It is the roughly cylindrical volume of bone which surrounds a blood vessel and which is formed by osteoblasts in the spaces excavated by osteoclastic resorption within bone. The process of resorption and subsequent apposition at the same site in compact bone is called remodelling. Haversian canals in man range from 45-65 μm in diameter and have an average length of 240 μm (Jowsey, 1968). They are found in compact but not in cancellous bone.

23(2).1.2.13 Lacunae

These are tiny spaces within mammalian bone which house the osteocytes. In human bone they are about 24 μm long, 8 μm dia., and occupy 2% of bone volume; there are about 25,000 lacunae /mm^3 (Robinson, 1964).

23(2).1.2.14 Canaliculi

These are canals about 0.5 μm dia., separated by 3 μm, which intersect all the lacunae, providing fluid pathways for rapid transport of ions between the matrix and the blood vessels (Robinson, 1964).

23(2).1.2.15 Recycling

It refers to the redeposition in bone of activity that has transferred from bone to blood and back to bone again.

23(2).1.3 List of Data

The periods of observation for the human data considered in developing the model range from 15 min to 87 y after injection and include the following (detailed references to literature are given in ICRP 20):

(a) *Bone*

Number of osteons *vs* age in compact bone (Kerley, 1965); tetracycline data for rates of bone formation (Frost, 1969); fraction of bone surface forming new bone (Jowsey *et al.*, 1965); and surface/volume ratio of compact and cancellous bone (Lloyd and Hodges, 1971).

(b) Detailed data on biokinetics of calcium, strontium, barium, and radium.

23(2).1.4 Results

The results are presented in four ways:

(a) Summary graphs of the whole body and internal retention functions for each alkaline earth from 10^{-2} to 10^{5} days after intravenous injection;

(b) Twenty-six detailed graphs which compare the model with the sets of data;

(c) Tables of the eight retention functions for each alkaline earth from 15 min to 87 y after injection; and

(d) Tables of integrals of the retention function for each alkaline earth isotope (including radioactive decay) to 1 year and to 50 years after injection.

23(2).1.5 Intended Uses

The model is intended to be useful both as a tool for further metabolic research and for calculating radiation doses to special groups of cells in human bone. As a research tool it allows one to work downwards from established data towards mechanisms. It also provides a test of consistency for the individual mechanisms and the fragments of existing data.

Concerning radiation dose, the model should be useful for interpolating or extrapolating from the existing fragmentary data to obtain estimates of exposures to osteocytes, endosteal or marrow cells in compact or cancellous bone following any schedule of intake of an alkaline earth radioisotope. These estimates may be standardized against the known toxicity of radium in humans.

23(2).1.6 Comparison with Existing Models

The new model is consistent both with the results of kinetic analysis and with direct microscopic observations of bone formation. The model fits the ^{90}Sr data for adult body burden from fallout *vs* year as well as the existing models. In addition, it supplies separate predictions for compact

and cancellous bone. The model includes the Observed Ratio model as a special case. The new whole body retention function for radium is within 20% of Norris's power function model (Norris *et al.*, 1955) from 1 day to 25 years after radium injection. However, the new model retention curve falls more rapidly after 25 years so that dose calculations based upon late measurements of body burden will be larger than those calculated from the Norris function (for measurements made 36 years after injection the factor is 1.5; this factor becomes 2 at 63 years after injection).

23(2).2 INTRODUCTION TO THE ICRP 20 MODEL

In order to calculate the radiation dose to various parts of bone tissue from internally deposited radionuclides, one needs to know their pattern of uptake and retention within the skeletal system. Data for humans are scarce, so that existing estimates have been based either on the assumption of a uniform distribution of the radionuclide or an Observed Ratio which relates the activity of newly formed bone to the specific activity of the diet. ICRP 20 has gone more deeply into the subject to construct a quantitative metabolic model.

The new model so far applies only to the bone-volume-seekers, the stable and radioactive isotopes of calcium, strontium, barium and radium, because they are most closely related to the metabolism of bone itself. The pattern of uptake and retention of the bone-surface-seekers, the rare earths and actinides - particularly plutonium - can probably be related to that of the alkaline earths, using surface/volume ratios of bone (Lloyd and Hodges, 1971) and setting the rate of diminution (the removal of activity from bone without resorption) equal to zero. It may be possible to derive the retention functions for activity taken in during adolescence from the present functions through suitable integrals. The present report is limited to the four alkaline earths in normal adult humans.

The model was developed by trial and error subject to three constraints: Accuracy (fit with existing data for man), logic, and simplicity. Most of the parameters can be measured in separate experiments independently of the values of other parameters. Although some principles of compartmental systems have been incorporated, compartmental analysis itself has been avoided; the attempt has been to start with the whole body retention function and work with it towards the mechanisms rather than the other way round. The model has used the minimum number of parameters necessary to fit the data satisfactorily.

In ICRP 20, the historical evolution of the model is traced. Modifications and additions to the Marshall Theory of Alkaline Earth Metabolism (Marshall, 1964) are described. Then follow sections on terminology, definitions of parameters, data, and results.

23(2).3 NON-MATHEMATICAL DESCRIPTION OF THE MODEL

In 1958 Norris, Tyler and Brues (Norris *et al.*, 1958) recognized that the whole body retention of radioactive bone seekers following a single intravenous injection could be represented by

$$R = at^{-b},$$

where R is the whole body retention function, b is a positive fraction (0.1-0.6), t is the time after injection in days ($t > 1$ day), and a is the fraction of injected activity retained at 1 day.

The behaviour of this function, both at short and at long times, made no physiological sense. As t approaches zero, R increases without limit. At long times, the integral of R increases without limit as t increases and so would not predict an eventual equilibration of body and blood under continuous radioisotope intake. Modifications to the formula were then developed to get over the anomalies in the short term and long term behaviour. Further refinements took place culminating in the development of the present model.

23(2).3.1 Postulates of the New Model

The retention functions of the present model are based on the following nine postulates [some of them are carry-overs from the Marshall theory (Marshall, 1964) of alkaline earth metabolism]:

23(2).3.1.1 The Excretion Postulate (1)

The number of radioactive atoms excreted from the body per unit time is proportional to the number in the serum or plasma at that time.

23(2).3.1.2 The Retention Postulate (2)

When a radioisotope has been introduced into the blood at time zero, its subsequent retention in the body is given by the whole body retention function R.

23(2).3.1.3 Age-Invariance Postulate (3)

The functions R and $R_{\text{subscript}}$, which describe the retention of a radioisotope in the body or in part of the body following a single intravenous injection, do not depend upon the age at the time of injection.

(This postulate may not be applicable for ages less than about 24 years.)

23(2).3.1.4 The Equilibrium Postulate (4)

If an alkaline earth radioisotope were introduced into the blood at a constant rate for an endless time, the activity per gram calcium in the body or any part of the body would eventually equilibrate with the activity per gram of calcium in blood serum or plasma.

For calcium, this equilibrium implies equality. However, for other alkaline earths, the equilibrium activities per gram calcium may be somewhat larger than for plasma. For example, there is a preferential uptake of all the three other rare earths in human soft tissue, and of barium and radium in bone (ICRP publication 2, 1960; Tipton, 1968; Maletskos *et al.*, 1969; Liniecki, 1971).

The above postulates are now used to derive a relation between the time integral of retention, R, and the rate of excretory plasma clearance, ηk (g Ca/day); k is the rate of endogenous calcium excretion, urinary plus fecal; η is the excretory discrimination factor for the given alkaline earth relative to calcium. From postulates 1 and 3 it follows that for continuous intake, the activity per gram calcium in the plasma, S, will increase until the equilibrium value $\dot{q}/\eta k$, where $\dot{q}$ is the rate at which activity is introduced into the blood. From postulate 4 it follows that the activity per gram calcium in the body will increase until it reaches an equilibrium value equal to $\upsilon\dot{q}/\eta k$, where υ is a factor which represents the overall effect of discrimination between blood and other parts of the body for a particular alkaline earth (υ= 1 for calcium). Thus

$$\int_0^\infty R\,dt = \upsilon c/\eta k$$

where c is the whole body calcium in grams. This expression relates the rate of excretory plasma clearance ηk to the final exponential rate λ and hence to the turnover rate of compact bone.

23(2).3.1.5 Compact-Cancellous Postulate (5)

In the new model, 80% of bone is classified as compact and 20% as cancellous, these proportions applying to bone volume, to bone mass, and to bone calcium. Each of the two classes of bone is assumed to have its own rate of apposition-resorption. If there is any discrimination among alkaline earths in the transfer from blood to bone, it is assumed to be the same in compact as in cancellous bone.

This postulate is a simplification. The turnover rate of compact bone varies from diaphysis to metaphysis, and the turnover rates of different cancellous bones are different. Human compact bone has a surface/volume ratio of 30 cm^2/cm^3 and cancellous bone has a ratio of 120 cm^2/cm^3 (Lloyd and Hodges, 1971). The ratio of these ratios is 4, and has been built into the model.

A ratio of 4 in surface/volume ratio of the two classes of bone together with the inverse ratio of 4 in their volume implies that there is the same amount of bone surface in compact and cancellous bone (assuming that the densities are the same). This value of 4 is used by ICRP 20 as the ratio of the turnover rates of compact and cancellous bones, implying that the amount of bone formation per unit area of bone surface is roughly the same in both types of bone. (The terms 'compact' and 'cancellous' are used in preference to 'cortical' and 'trabecular' bone respectively).

The assumption of equal discrimination in the two classes of bone, together with postulate 4, is used to determine β, the fraction of bone volume activity deposited in compact bone. Under continuous intake, the activity per gram calcium of compact bone would eventually reach a constant value equal to that attained earlier by cancellous bone. Therefore, the time integrals of the activities per gram calcium in the two classes of bone following a single injection must be equal.

23(2).3.1.6 Diminution-Resorption Postulate (6)

The two processes of alkaline earth removal, diminution and resorption, are associated with individual terms of the local and whole body retention functions. Diminution in both compact and cancellous bone is

represented by the power function, while resorption in each class of bone is represented by its own exponential function. This is the key postulate of the new model.

23(2).3.1.7 New Bone Postulate (7)

Whenever new bone is formed, its activity per gram calcium equals ω times the current activity per gram calcium of the blood plasma, where the discrimination factor ω is unity for calcium or strontium, but may be somewhat greater than unity for barium or radium. This postulate is broadly supported by experimental data.

23(2).3.1.8 Old Bone Postulate (8)

In each class of bone, compact and cancellous, the retention function for activity in old bone is at all times directly proportional to the retention function for new bone.

It implies equal diminution in new and old bone as well as equal probabilities of resorption of new and old bone. The evidence for this postulate is scarce and somewhat mixed.

23(2).3.1.9 Bone Surface Postulate (9)

The rapidly exchangeable alkaline earth activity in bone occurs at bone surfaces and can be represented by a single compartment in direct exchange with blood. The calcium content of this compartment per unit bone surface area is assumed to be the same throughout all bone.

23(2).3.2 Sub-division of Activity within the Model

In addition to the retention function, R, for the whole body, (dimensionless) functions for the fractions of injected activity in different components following a single intravenous injection (not including radioactive decay), denoted by $R_{\text{subscript}}$, are postulated for the different components:

(a) Radioactivity in the body is subdivided into those in blood, soft tissue and bone;

(b) Radioactivity in bone is subdivided into those on bone surface and in bone volume;

(c) Bone volume activity is subdivided into those in compact and cancellous bone;

(d) Bone volume activity in compact and cancellous bone is further subdivided into those in old and new bone; and

(e) Half of bone surface activity is in compact and half in cancellous bone.

Except for blood, all other fractional retention functions are zero at time zero. ICRP 20 then discusses the local retention functions in detail.

23(2).4 HOW TO USE RESULTS TO CALCULATE THE DOSES

Tables of time integral of the effective retention function

$$\int_0 R\, e^{-\lambda' t} dt$$

for each isotope are given (λ' is the radioactive decay constant). The integral values are therefore mean residence times following the introduction of one unit of activity by ingestion or inhalation.

To use these integral values to calculate endosteal or marrow doses, one should first consider the effects of particle range.

For long range beta emitters (such as ^{90}Sr + ^{90}Y), it is probably sufficient to use the total activity of bone as a function of time to calculate the doses to bone marrow or to endosteal surfaces. The exact location of the activity within the bone should not have a significant effect upon the corresponding distribution of dose.

On the other hand, for alpha or short range beta emitters (< 100 μm), one should take into account the effect of hot spot burial accompanying bone formation which greatly reduces the dose to the bone surface or bone marrow from the new bone components of the activity (Marshall, 1962). Therefore, in this case, the significant components of activity are probably old bone (the diffuse component) and bone surfaces. The former is more significant for long-lived isotopes and the latter for short-lived ones. Factors to convert these bone doses to the corresponding doses to small cavities within bone, D_0 , and to calculate the ratio of the average doses to endosteal cells and to marrow cells to a given dose D_0 have been given by Spiers (1968).

ICRP 20 mentions that further evidence should be obtained before the immunity (suggested in ICRP 11) of the osteocytes to radiation carcinogenesis is regarded as established.

23(2).5 COMPARISON OF MODEL WITH DATA FOR NORMAL MAN

23(2).5.1 Bone Data

23(2).5.1.1 Number of Osteons in Human Compact Bone as a Function of Age

Kerley (1965) has reported data for the number of osteons in the outer one-third of the cortex from the midshaft of the femur, tibia and fibula in human bone specimens covering ages from birth to 95 years. He observed a strong correlation between the number of osteons and the age of the subject which he presented as a microscopic method for determining the age. ICRP 20 has re-analyzed Kerley's data to find their correspondence with the postulate of random remodelling. The report has also attempted to derive a value for the turnover rate of normal compact human bone from Kerley's data. The rate of osteon formation comes out to 2-23% per year in a considerable volume of compact bone.

23(2).5.1.2 Tetracycline Data for Rates of Bone Formation in Man

Since tetracycline is taken up on growing bone surfaces and can be identified in bone sections by fluorescence, it can be used for the determination of rates of appositional bone formation and bone turnover. The rates of bone formation in adults are rather variable, but normal values for compact bone are of the order of 2-6% per year. For children less than 1 year old, the value is 85 ± 40% per year, for 1-10 year olds 38 ± 24%, and for 10-20 year olds 21 ± 15% per year (Frost, 1969).

23(2).5.1.3 Microautoradiographic Measurements of Bone Formation Surface in Normal Man

Jowsey *et al.* (1965) have made microradiographic measurements of the fraction of the total bone surface at different sites which pertains to formation surface, that is bone surface at which new bone surface is being formed. Formation surface is distinguished from resting surface or resorption surface by its low mineral density. The mean value comes to 2.7 ± 0.5%, depending upon the site; there appears to be no correlation with age.

23(2).5.2 Data for Calcium, Strontium, Barium and Radium

There is a detailed discussion of the experimental data in animals as well as humans on the biokinetics of the different alkaline earths, both for single and repeated intakes, and a comparison of the data with the computations of the present model.

23(2).6 RESULTS

Values of the parameters defined for the four alkaline earths are shown in Table 23(2).1. ICRP 20 then gives values of the retention functions for each of the four alkaline earths. Values of the per cent of injected dose *vs.* time after a single injection are tabulated for various times from 0.01 to 31,600 days for the different components mentioned earlier. Plots of these functions are also given. As an example a table and a graph for radium are reproduced as Fig.23(2).1 and Table 23(2).2.

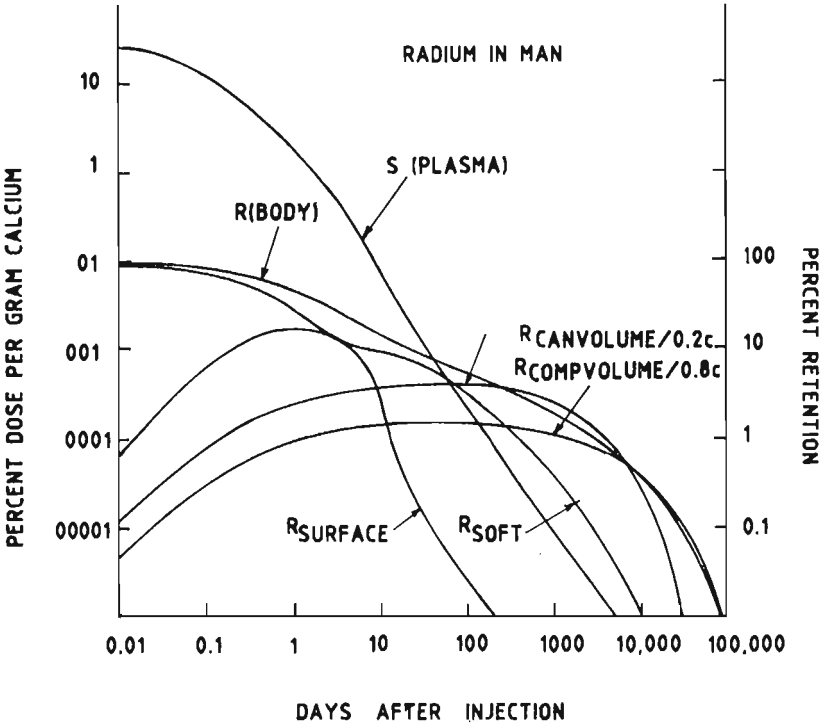

Figure 23(2).1. Summary graph for radium giving percent dose per gram calcium and percent retention in various compartments *vs.* days after injection (from ICRP 20).

Next are given (in ICRP 20) tables of the time integrals of the retention functions in days for 1, 50 and ∞ years after injection (which include the effect of radioactive decay). Table 23(2).3 for radium is given as an illustration.

23(2).7 NEED FOR MORE DATA

The report concludes by pointing out gaps in the data that need to be filled. These include bone and soft tissue data for the heavier alkaline earths; data for the relative activity in new and old bone; data for diminution in new and old bone; continual measurements of ^{90}Sr levels in different human bones; and data for rates of resorption in the radium cases.

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Table 23(2).1: Values of the whole body and tissue retention function parameters for Ca, Sr, Ba and Ra
(from ICRP 20)

Parameter	Units	Calcium	Strontium	Barium	Radium
Independent					
b	Dimensionless	0.10	0.18	0.237	0.415
ε	Day	0.76	0.20	0.007	0.12
λ	%/year	2.5	2.5	4.0	1.5
k	g Ca/day	0.275	0.275	0.275	0.275
η	Dimensionless	1	3.80	37.1	36.5
σ	Dimensionless	4	4	4	4
p	Dimensionless	0.79	0.60	0.62	0.821
m	Day^{-1}	0.10	0.25	0.75	0.4
ω	Dimensionless	1	1	1.52	1.31
c	g Ca	1000	1000	1100	1000
$c_{SURFACE}$	g Ca	4	2	4	12
λ_{SUR}	Day^{-1}	0.3	0.3	0.3	0.3
f_c	Dimensionless	2.29	2.22	2.0	3.0
f_i	Dimensionless	-	0.19-0.21	-	0.15-0.21
v_i	g Ca/day	1	1	1	1

(contd ...)

Parameter	Units	Calcium	Strontium	Barium	Radium
Dependent					
$10\,\eta k$	Litres/day	2.75	10.4	102	100
β	Dimensionless	0.532	0.555	0.564	0.608
r	Dimensionless	0.826	0.949	0.991	0.997
A_S /c	%/year	15.2	15.0	43.5	52.0
INTRSOFT	%	0.403	1.98	6.36	14.4
ν	Dimensionless	1	1.016	1.62	1.50
f_T	Dimensionless	2.01	1.78	1.54	1.94
DIFRATIO	Dimensionless	3.14	2.55	2.17	1.87
t_x	Days	730	190	24	42
t_z	Years	20	16	9	21
$(B/S)_\infty$	Dimensionless	4.86	16.1	84.6	244

(*contd* ...)

Legend (for Table 23(2).1):

The meaning of symbols relating to some of the parameters of the retention function has already been explained in the Summary at the beginning of this Chapter. The other symbols are explained below.

Independent Parameters

k	rate of endogenous Ca excretion (urinary plus fecal)
η	excretory discrimination factor for the given alkaline earth relative to Ca
ω	discriminatory factor for alkaline earth relative to Ca from blood to new bone
c	whole body Ca (also used for total bone Ca)
$c_{SURFACE}$	Ca content of bone surface pool
λ_{SUR}	rate constant of bone surface pool
f_c	ratio of the total activity deposited in compact bone volume to that deposited in new compact bone
f_i	fraction of dietary activity absorbed into the blood
v_i	daily intake of Ca in the diet

Dependent Parameters

$10\,\eta k$	rate of excretory plasma clearance (l/day) when ηk is in (g Ca/day)
A_t	addition rate at time t (mass of Ca per unit time)
INTRSOFT	fraction of alkaline earth in soft tissue under continuous intake
ν	discrimination factor for alkaline earth relative to Ca from blood to body as a whole
f_T	ratio of total activity deposited in cancellous bone volume to that deposited in new cancellous bone
DIFRATIO	ratio of activity per g Ca deposited in the diffuse component of cancellous bone to that deposited in compact bone
t_x	time for equal activities per g Ca in plasma, S, and in body, B
t_z	time for equal activities per g Ca in cancellous and compact bone

Variables

S	activity per g Ca of the blood plasma or serum
B	activity per g Ca of the body as a whole
R	fractional retention of activity in the body following a single intravenous injection
$R_{SUBSCRIPT}$	fractional retention of injected activity in the tissue indicated by the subscript
F	activity per g Ca of the diet
θ	a time-dependent substitution for the power function
t	time following single injection or the beginning of continuous intake

Table 23(2).2: Percent of injected dose in whole body and various tissues vs time after single injection of radium
(from ICRP 20)

Time (days)	Blood	Bone surfaces	Soft tissue	Bone volume				Compact bone	Cancellous bone	Whole body
				New compact	Old Compact	New cancellous	Old cancellous			
0.01	7.80	0.072	89.3	0.0119	0.0237	0.0119	0.0111	0.0716	0.0590	97.2
0.0316	6.30	1.94	83.8	0.0337	0.0670	0.0337	0.0316	1.07	1.04	92.2
0.1	3.81	5.94	70.9	0.0820	0.163	0.0820	0.0770	3.22	3.13	81.0
0.316	1.56	11.6	49.9	0.156	0.312	0.156	0.146	6.27	6.10	63.8
1.0	0.504	15.5	27.3	0.239	0.478	0.239	0.224	8.47	8.21	44.5
3.16	0.139	12.3	11.8	0.319	0.639	0.319	0.299	7.11	6.77	25.8
10.0	0.0198	2.48	9.01	0.372	0.744	0.372	0.348	2.36	1.96	13.3
31.6	0.00318	0.134	6.02	0.390	0.781	0.389	0.365	1.24	0.821	8.08
100	0.000633	0.253	3.04	0.390	0.781	0.386	0.361	1.18	0.760	4.99
316	0.000127	0.00509	1.27	0.362	0.724	0.348	0.326	1.09	1.677	3.04
1000	0.0000266	0.00106	0.393	0.293	0.586	0.259	0.243	0.880	0.503	1.77
3160	0.0000058	0.000231	0.0830	0.194	0.388	0.132	0.123	0.582	0.255	0.921
10000	0.0000011	0.000044	0.0112	0.0970	0.194	0.0282	0.0265	0.291	0.0547	0.356
31600	0.0000001	0.000005	0.00079	0.0252	0.0505	0.00051	0.00048	0.0757	0.00099	0.0780

Table 23(2).3: Time integrals of the whole body and tissue retention functions (including radioactive decay) in days for 1, 50 and ∝ years after single injection of radium

(from ICRP 20)

Isotope (halflife)	Time (years)	Blood	Bone Surface	Soft tissue	Bone volume				Compact bone	Cancellous bone	Whole body
					New compact	Old compact	New cancellous	Old cancellous			
^{226}Ra											
(1602yrs)	1	0.0288	1.16	10.8	1.46	2.92	1.43	1.34	4.96	3.35	18.8
	50	0.0296	1.19	21.2	24.2	48.5	12.8	12.0	73.3	25.4	119.0
	∝	0.0297	1.19	21.5	33.3	66.5	13.1	12.2	100.4	25.9	147.7
^{228}Ra											
(5.75yrs)	1	0.0288	1.15	10.4	1.37	2.74	1.34	1.26	4.69	3.18	18.0
	50	0.0292	1.17	16.2	7.16	14.3	5.72	5.36	22.0	11.7	49.8
	∝	0.0292	1.17	16.2	7.16	14.3	5.72	5.36	22.0	11.7	49.8
^{225}Ra											
(14.8days)	1*	0.0250	0.868	2.14	0.0768	0.154	0.0766	0.0718	0.665	0.582	3.36
^{223}Ra											
(11.4days)	1*	0.0244	0.815	1.83	0.0580	0.116	0.0578	0.0542	0.582	0.520	2.91
^{224}Ra											
(3.64days)	1*	0.0209	0.516	0.933	0.0160	0.0320	0.0160	0.0150	0.306	0.289	1.53
^{227}Ra											
(41.2min)	1*	0.00234	0.00111	0.0312	0.0000154	0.0000307	0.0000154	0.0000144	0.000601	0.000585	0.0348

* Value of integral equals that for infinite time.

CHAPTER 23: SECTION 3

THE METABOLISM OF PLUTONIUM AND OTHER ACTINIDES

23(3).1 INTRODUCTION

ICRP publication 19, 'The metabolism of compounds of plutonium and other actinides' (1972) and ICRP publication 48, 'The metabolism of plutonium and related elements' (1986) are reports prepared by Task Groups of Committee 2 of ICRP.

ICRP 19 dealt mainly with plutonium compounds, both because of the paucity of literature on other actinides, and because it was the most important radionuclide in terms of potential exposures and radiotoxicity. ICRP 48 is intended to complement, rather than replace, ICRP 19, and follows the general pattern of presentation of the latter.

In ICRP 19, with the exception of the study on the behaviour of intravenously injected plutonium citrate in human volunteers (Langham and Carter, 1951; Langham *et al.*, 1980), virtually all the data came from animal studies. In recent years, considerable information has become available, both from studies on a wide range of animal species and from the analysis of human tissues taken at autopsy from normal persons exposed only to $^{239,240}Pu$ from atmospheric nuclear weapon testing, or from persons occupationally exposed to plutonium or americium in the nuclear industry.

ICRP 48 begins with a short overview of the chemistry of the actinides - plutonium, thorium, neptunium, including the trivalent actinides, viz. actinium, americium and curium (all of them being alpha emitters) in mammalian systems, then reviews the behaviour of the elements following entry by inhalation, by ingestion, or through the skin, and subsequently deals with the retention of plutonium and the actinides in liver, bone, gonads and other organs of the human body. Finally, conclusions are presented, dealing not only with occupational exposure, but also the possible exposure of the general population, including infants and fetuses, in the event of releases of these elements from nuclear installations.

23(3).2 THE CHEMISTRY OF PLUTONIUM AND RELATED ELEMENTS

The biological behaviour of plutonium and, usually to a lesser extent, that of the other actinides, may be influenced markedly by the physico-chemical form in which the element enters the body. It is not possible to predict the exact form in which actinides will enter the body under all likely exposure situations. Some of the more common types of compounds are: metal; oxides; mixed oxides (plutonium-uranium; plutonium-potassium; plutonium-sodium); chloride, oxychloride; carbonate; nitrate; nitride; oxalate; tributyl phosphate (TBP) complex; tri-iso-butyl phosphate (TIBP) complex; cermets; complexes with soluble organic substances in foodstuffs, e.g. citrate, phytate, lactate, proteins; and insoluble species deposited in meat and vegetables or suspended in water.

The term 'monomeric' refers to preparations in which any particulates formed are below 0.01 μm in diameter and 'polymeric' refers to those with particle diameters 0.01 μm to 1 μm or larger. In the milieu of the body monomeric plutonium becomes converted into at least the minimally polymeric form. The colloidal and particulate character of the actinide compounds in tissue provides the main basis for difference in the physiological behaviour of these polyvalent nuclides as compared with the divalent alkaline earths.

From the point of view of industrial radiation protection the 'oxides' form what is probably the most important class of compounds. They may be in pure form or in non-stoichiometric mixtures with other oxides and they may have been formed at either very high (above 1500 °C) or relatively low, including ambient, temperatures. The composition and formation temperature of the 'oxides' can markedly influence their solubility and, thus, their biological behaviour. In mixed oxides the plutonium is usually accompanied by a greater mass of uranium, sodium or other metal oxide which is more soluble than the plutonium oxide itself. *In vivo*, this bulk matrix is relatively rapidly solubilized, leaving the plutonium in the form of very small aggregates, < 1 nm in diameter, which may not dissolve completely, but appear to enter the systemic circulation and to be deposited in tissues or excreted in particulate form (Cooper *et al.*, 1979).

The actinides in the fallout from nuclear weapon testing in the atmosphere consist predominantly of plutonium with traces of americium, curium and neptunium. The fallout plutonium consists, in part, of 'oxides' which were formed at very high temperatures in the nuclear explosion. However, perhaps as much as two-thirds of the approximately 6 tons of ^{239}Pu

deposited on the earth's surface was produced from volatilized ^{238}U via a (n,γ) reaction and subsequent decay of the ^{239}U through ^{239}Np to ^{239}Pu (Joseph *et al.*, 1971). This material was formed as single atoms and was not subjected to high temperatures (Watters *et al.*, 1980). This type of plutonium oxide appears to be more soluble than if the oxide had been produced at high temperatures.

The exact chemical form ('speciation') of the actinides in various components of food is not known, but may vary according to the source of plutonium and the specific food or water source. In most cases, the plutonium may be associated with other actinides such as americium and curium (which arise by neutron capture, or other nuclear reactions occurring as a result of the fission of uranium or plutonium, or by subsequent radioactive decay).

In the actinide series, the ionic radii of the elements, for a given oxidation state, decrease with increasing atomic number from Ac(III), r = 0.111 nm, to Cf(III), r = 0.094 nm (Katz and Seaborg, 1957). This 'actinide contraction' is related to the filling of the 5f electron shell as the nuclear charge increases, and produces a characteristic trend in certain chemical properties. For example, as the atomic number increases, there is a small progressive increase in the acidity of the cations, in the tendency to form covalent bonds, and in the stability of some complexes.

Neptunium, plutonium, americium and curium exhibit multiple oxidation states from +3 to +7 for Pu and +2 to +6 for Np, Am and Cm. With Pu, all 4 oxidation states, from +3 to +6, may co-exist in equilibrium in acidic aqueous solutions. In mammalian body fluids Pu(IV) is assumed to be the predominant state, mainly because most ligands stabilize this state (Raymond and Smith, 1981). Np(V) state appears to predominate at pH values up to 7. For Am, Cm and the higher transplutonium elements the +3 oxidation state is the most stable in solution.

The competing phenomena of hydrolysis and complex formation play important roles in determining the biological behaviour of these elements. Because of their high ionic charge, the tetravalent actinides, Th and Pu, show a strong tendency to hydrolysis, leading to the formation of polymers or particles at pH > 2. The trivalent transplutonium elements, Am to Fm, hydrolyze to a much lesser degree. Np(V) ion shows virtually no tendency to hydrolyze below a pH of 7.

Hydrolytic reactions may be modified by a number of factors, such as the concentration of the metal in the solution, the pH, the temperature and the presence of other anions or cations. In mammalian systems at about pH 7, it is unlikely that uncomplexed Th, Pu or transplutonium ions can exist except at extremely low concentrations. (Polymerization and precipitation can be prevented by the presence of high concentrations of complexing anions like citrate.) Thus if free ions enter the body they will hydrolyze rapidly to form polymeric species or interact with body constituents. In the GI tract hydrolysis of soluble actinide salts to form insoluble products accounts for their extremely low absorption. Similarly, when soluble salts penetrate the skin or enter a wound, the hydrolytic and complexation reactions result in the deposition of most of the material as an insoluble mass at the site of entry from which translocation to other tissues is slow.

Following administration of a strong chelating agent like DTPA after contamination with an actinide, some of the material already deposited in the tissues may react to form a stable low molecular weight chelate which is rapidly excreted from the body in the urine or feces. This is the basis of chelation therapy for internal decontamination.

Transferrin is the predominant transport protein in the plasma for Pu and Th, and to a lesser extent for other elements under consideration. The metals seem to bind to the same sites as those which bind iron and involve largely the same binding ligands (Harris *et al.*, 1981).

The binding of actinides to other proteins including ferritin, bone sialoprotein and salivary proteins has been reported. Actinide complexes with small molecules, especially citrate and other carboxylic acids which are ubiquitous in tissues and body fluids, also play an important role *in vivo*. Plutonium (and to a smaller extent some other actinides) may be excreted as a citrate complex which has been ultra-filtered from the blood (Popplewell *et al.*, 1975).

The observed differences in the deposition and retention patterns of plutonium and other actinides may be related to differences in the chemical binding of the elements within the tissues. Plutonium retention in the liver shows wide species variations. Liver lysosomes are the principal intracellular deposition sites for most actinides.

23(3).3 ENTRY OF ACTINIDES BY INHALATION

Inhalation is the most significant route of occupational exposure. The ICRP 30 lung model developed for general application in radiological

protection (described in Chapter 21) cannot reflect the unique pulmonary behaviour of every inhaled radioactive compound.

Compounds of plutonium and other actinides and rare earths largely fall into class Y or class W of the ICRP lung model. Carbides, oxides, hydroxides and fluorides belong to class Y, while nitrates, halides other than fluoride, and carbonate fall into class W. Those compounds stable enough to remain soluble under physiological conditions all belong to class D. Examples of class D compounds are plutonium tri-n-butyl phosphate (Pu-TBP), which is of importance to occupational exposure conditions, EDTA and DTPA complexes. Only plutonium is discussed in some detail in this chapter.

23(3).3.1 Plutonium Compounds

In addition to the extensive animal data, some human data are available, based on studies of populations chronically exposed to fallout and contaminated workers. It appears that humans behave more like dogs than like rats.

In general, $^{239}PuO_2$ behaves like a class Y compound. Micrometre-sized particles of $^{239}PuO_2$ and mixed oxides of plutonium and uranium are cleared in 2, or 3, phases. In the first few days after exposure, mechanical transport along the airways is the dominant process. For tens of days thereafter, clearance is a combination of mechanical transport and absorption. Longer-term clearance involves half-times of several hundred days (but there may be wide variations between individuals of a given species, probably related to serious lung damage).

Provided lung function is normal, it is proposed that, in man, mechanical transport of insoluble ^{239}Pu particles should be represented as proceeding at about 0.1% per day. At long times after exposure, clearance may become more dependent upon the translocation of the plutonium to blood and to lung-associated lymph nodes, than on mechanical transport.

The amount of ^{239}Pu reaching the blood is influenced by the crystalline structure of the inhaled oxide, the shape and size of the deposited particles, the degree of mixing of the plutonium with other elements, and the solubility of the material in the phagolysosomes of alveolar macrophages. The rate of translocation of a given compound to blood appears to be reasonably constant among different species. For oxides fired below a temperature of 1000°C, a transport rate of 0.02-0.1% per day is proposed,

depending on the particle size of the inhaled aerosol; $^{239}PuO_2$ and $^{239}PuO_2$: UO_2 fired at higher temperatures exhibit an even slower rate.

The transfer of $^{239}PuO_2$ to lung-associated lymph nodes is assumed to be 0.035% per day for a class Y compound; this is based on studies on beagles exposed to high levels. Human experience at lower levels of inhalation of material indicates an order of magnitude reduction in the amount reaching the nodes.

^{239}Pu in nanometre-sized oxide aerosols are more rapidly dispersed than the ^{239}Pu associated with micrometre-sized $^{239}PuO_2$ aerosols. These small sized particles are rapidly translocated (> 1% per day) across the alveolar capillary-endothelial interface, corresponding to a class W compound, although a small class Y component is usually present.

Autoradiographic studies of animals exposed to a polydisperse $^{239}PuO_2$ aerosol inhalation show that a few months after exposure there is a uniform distribution of the radioactivity in the lungs; a year after exposure the distribution is non-uniform, with aggregation in peripheral and subpleural tissues. After about 6 years, a small volume of lung tissue contains most of the activity and clustering occurs in areas of fibrosis (McShane *et al.*, 1980).

Translocation to extra-pulmonary tissues is the dominant process involved in the clearance of plutonium compounds other than the oxide. On contact with lung fluids, a soluble salt such as plutonium nitrate rapidly hydrolyzes and polymeric plutonium is phagocytosed by alveolar macrophages. Colloidal phospholipid complexes are similarly phagocytosed. The release of plutonium from the macrophages then depends upon the rates of dissolution from the complex and the polymer. Soluble plutonium salts conform generally to the properties of a class W compound (but with considerable variations from the value of 0.5% per day used in the model).

Lung pathology and cigarette smoking decrease the rate of clearance in animals.

There are some limited human data on inhaled oxides of plutonium, viz. populations chronically exposed to fallout, and contaminated workers. The computed estimates of plutonium tissue content in populations (based on radioactivity measurements in air, food and water, and application of the ICRP dosimetric model) have been compared with measured contents in an extensive autopsy survey in US (McInroy *et al.*, 1979). Plutonium was measurable in lung, liver and bone. There was reasonable agreement

between computed and measured values for lung. Computed values were 3 times lower for skeleton and somewhat lower for liver than measured values (McInroy *et al.*, 1981).

23(3).3.2 Conclusions

Predicting the clearance pattern of actinides from human lungs is uncertain. Studies on human volunteers indicate that actinide clearance from the lungs will occur in 2 components, with half-times of 20-30 days and 500 days respectively (Bailey *et al.*, 1982). There is no rapid clearance of insoluble particles from the pulmonary parenchyma. The rate of clearance of particles deposited in the smaller bronchial airways may be substantially less than that predicted by the lung model.

Animal studies suffer from the limitation that the animals were generally exposed to amounts large enough to produce radiation damage which might affect the clearance pattern. Even when smaller amounts are administered, there are marked inter- and intra-species variations in the mechanical transport rate. The rate of translocation of actinides from lungs to blood is species-independent (Cuddihy *et al.*, 1979).

The precise composition of many of the potentially-respirable actinide compounds is not known. The shape, size, mass and crystalline structure of the particles, the specific activity of the radionuclide, and presence of other elements are factors that may influence the *in vivo* solubility of actinide particles in lung fluids. The presence of naturally occurring complexing agents in lung fluids and macrophages also influences the availability of the actinide and its ability to reach the blood.

Plutonium oxide inhaled as micrometre-sized $^{239}PuO_2$ particles translocates to blood with half-times of several hundreds of days (class Y compound), although the rate varies with particle size, temperature of formation, and presence of other elements. Micrometre-sized $^{238}PuO_2$ particles translocate to blood 10 times more readily than similar sized $^{239}PuO_2$ particles, and show properties closer to class W compounds.

Actinides inhaled as nanometre-sized $^{239}PuO_2$, other soluble plutonium compounds, all americium and curium compounds, or soluble compounds of einsteinium and californium, translocate to blood with half-time of several tens of days (class W compounds); but the rate varies according to the mass of material inhaled, and the competing rates of hydrolysis, polymerization and complexation occurring in lung fluids.

23(3).4 ENTRY OF ACTINIDE ELEMENTS VIA THE GASTRO-INTESTINAL TRACT

When any material enters the gastro-intestinal tract, a fraction of the material will pass through the cells of the mucosa into the blood stream; this fraction of the ingested material which is transferred to the blood is called the 'fractional absorption' (f_1). Immediately after its entry into the blood stream, a part of the absorbed material will be excreted in the urine and feces (via the bile). Thus, when the retention in the total body, minus the GI tract, is measured at some time after ingestion, the measured 'fractional retention' will be smaller than the fractional absorption by the amount excreted.

On the basis of the then available data, ICRP 19 suggested a value of 3×10^{-5} for the fractional absorption of the more easily soluble compounds of plutonium, and a value of 10^{-6} for highly insoluble compounds such as PuO_2. Subsequently, ICRP 30 modified these values to 10^{-4} and 10^{-5} respectively. For all neptunium compounds, a value of 10^{-2} is currently recommended, whereas for all compounds of americium, curium and californium the value is 5×10^{-4}.

ICRP 48 gives a number of tables on the absorption and retention of plutonium and other actinides. In a few instances, the experimental conditions used are of little relevance to likely accidental or environmental intake situations in humans. Wide variations in the absorption of plutonium after ingestion of the same compound have been reported, indicating that the actual chemical and/or physiological conditions in the GI tract at the time of absorption varied considerably; perhaps there could have been differences in the true chemical composition of the solutions administered. The actinide concentration, the pH, and the presence of inorganic or organic complexing anions would have influenced the proportion of soluble and colloidal or particulate species, especially in solutions of plutonium in dilute nitric acid. The presence of food residues in the GI tract may also influence the absorption. The reported wide variations following administration of nominally similar compounds to animals indicate that considerable fluctuation should be anticipated in humans too.

23(3).4.1 Plutonium Absorption in Adult Animals

Absorption of plutonium from the GI tract may be influenced by the mass and chemical form of the ingested plutonium, as well as by dietary and other factors. The extensive data on different animal species studied

under various conditions (including high and low mass intakes and fed or fasting states) are summarized in Table 23(3).1.

At high mass levels of the metal, the citrate is more readily absorbed than the nitrate, and both are absorbed much more readily than the insoluble oxides; at lower administered masses, such as may be encountered under environmental conditions, absorption may be an order of magnitude greater than at high mass intakes, and there is no difference between the absorption of the citrate and the more readily hydrolyzable nitrate or carbonate (Larsen *et al.*, 1981a; Sullivan *et al.*, 1983a).

There is no convincing evidence to suggest that the valency state of the ingested material markedly influences the absorption.

The results of studies on the role of the mass of the ingested plutonium on the fractional absorption from the GI tract are equivocal. Absorption following ingestion of amounts comparable to those likely in accidental or environmental exposure situations may be more than tenfold greater than that following ingestion in the milligram range; further, at small ingestion levels, the influence of the chemical form on the absorption disappears. The presence of food residues in the GI tract can reduce absorption. Fasting increased absorption in 3 of the 5 animal species examined; it would be prudent to assume that a similar effect may occur in humans.

Deficiencies of calcium, vitamin D or zinc, diseases, drugs, concomitant external radiation (as may happen in a nuclear accident or nuclear war) could influence absorption, but these have not been studied in detail. When plutonium is administered as a complex with DTPA, the absorption is increased, but most of the absorbed complex is excreted rapidly in the urine (Ballou *et al.*, 1978).

The mechanism of absorption of actinides from the GI tract is not well understood. Extensive changes in plutonium speciation will occur during transit from mouth to duodenum (where the main absorption is considered to take place) as a result of mixing with saliva, then with acid gastric juices, and finally with neutral or alkaline duodenal juices. Thus for soluble plutonium the concentration of complexing ligands or of substances capable of forming insoluble compounds with plutonium may play a role in the absorption (Taylor *et al.*, 1986).

No direct studies on plutonium absorption in humans have been reported. Analysis of fallout plutonium content of bones suggests a value of

8×10^{-4} for fractional absorption; the general agreement of this value with animal data is encouraging (Mussalo-Rauhamaa *et al.*, 1984).

23(3).4.2 Plutonium Absorption in Neonatal Animals

Enhanced absorption (by a factor of 100-1000) has been observed in several species during the first few days or weeks of life, falling down to adult values by the time of weaning. This is considered to be related to the needs of the neonate to acquire antibodies by absorption of these macromolecules from the maternal colostrum. A similar high absorption should be assumed to occur in humans.

There is a high (temporary) retention of plutonium in the intestinal villi of the neonate (Sullivan and Gorham, 1983). It appears that the plutonium is taken into the mucosal cells and not bound only on the cell surface. The majority of this plutonium is not transferred to the blood, but is returned to the intestinal lumen by the normal sloughing of the mucosal cells and excreted in the feces (Sullivan *et al.*, 1984).

23(3).4.3 Absorption of Thorium

Animal data give values in the range of 5×10^{-5} to 6×10^{-3} for the fractional absorption. Neonate absorption may be 100-fold greater (Sullivan *et al.*, 1983b).

Human studies following oral administration of relatively large (milligram) quantities of thorium nitrate from a 'mock' luminous paint (^{234}Th sulphate) in elderly persons show a fractional absorption of 6×10^{-4}. Thorium can, with caution, be regarded as a plutonium analogue (Maletskos *et al.*, 1969).

23(3).4.4 General Discussion and Conclusions on Fractional Absorption Values to be Used for Radiation Protection Purposes

In the absence of adequate direct information in humans, it is necessary to extrapolate from animal studies. The derivation of a single value of fractional absorption for human situations must take account of all those physiological and dietary factors, including the chemical form, that may influence absorption. If the chemical and physical characteristics of the ingested material were known accurately (which is unlikely, especially in relation to members of the public) it might be practicable to derive separate values for each specific compound.

Comparison of the data for plutonium (and of the less extensive data for other actinides) shows some evidence of species differences. However, there is no clear evidence to indicate that any one of the animal species is a particularly appropriate model for humans.

For the various species studied, for plutonium nitrate at dose levels less than 5 μg/kg body weight, the majority of the values for fractional absorption following oral administration lie between 2 and 12 times the current (ICRP 30) value of 1×10^{-4} for soluble plutonium.

The majority of the values for citrate and bicarbonate/carbonate solutions lie in the range 3×10^{-4} to 4×10^{-3}. An intake of 5 μg/kg is equivalent to a total intake of 800 kBq by an adult and is large in relation to the probable industrial accidents and very large with respect to environmental intakes by the general population.

For plutonium oxides (for which less information is available) the fractional absorption values range from 3×10^{-8} to 1.7×10^{-4}. The current (ICRP 30) recommended value for insoluble compounds is 10^{-5}. The recommendation of a separate value of f_1 for all plutonium oxides seems unjustified.

Taking account of the many factors that influence absorption plus the fact that the majority of the absorption values for low mass intakes of plutonium lie above the current ICRP-recommended value of 10^{-4}, it would appear that some revision of this value is desirable. Since the animal data suggest a value larger than 10^{-4} but not greater than 10^{-3}, the latter would appear to give a sufficient margin of safety for radiation protection purposes in all situations where the intake cannot be described precisely. For occupational exposures, smaller values might be justified, where the chemical and physical state of the ingested material can be confidently established. The current value of 10^{-4} for plutonium nitrate and 10^{-5} for insoluble oxides could still be used.

At large mass intakes of neptunium (> 5 mg/kg body mass) the reported absorption or retention accords quite well with the current ICRP-recommended value of 10^{-2}. In most human exposure situations, the mass of neptunium ingested is unlikely to exceed 1 mg/kg, and an f_1 value of 10^{-3} appears reasonable for both workers and the general population.

The relatively limited data for americium, curium, californium and einsteinium indicate that the fractional absorption or retention of both soluble and insoluble compounds lies in the range 6×10^{-5} to 3×10^{-4},

which accords well with the current ICRP-recommended value of 5×10^{-4}. However, the influence of physiological and dietary factors on the absorption of these trivalent actinides has been little studied, and, in view of the fact that such factors can increase plutonium absorption, it would be prudent to assume the same f_1 value for these elements as for plutonium, viz. 10^{-3}.

In neonates, absorption may be 1 to 3 orders of magnitude higher than for adults. A value of 10^{-2} for the first year of life may be appropriate.

As discussed earlier, there is enhanced retention of ingested plutonium in the GI mucosa of neonates; the majority of this plutonium is not transferred to blood, but is returned to the intestinal lumen within a few days by normal sloughing of the mucosal cells and then excreted. It would be prudent to assume that some retention (~ 50% of the ingested dose) for a few days may occur in human infants during the first few weeks of life.

23(3).5 THE PENETRATION OF PLUTONIUM AND OTHER ACTINIDES THROUGH THE INTACT SKIN

Work with actinides can present some risk of contamination of the worker's skin on unprotected areas of the body. ICRP 19, because of paucity of data, did not recommend numerical values for percutaneous absorption. At present we have substantial data both from animal and human studies. Pig skin is a particularly appropriate model for the human skin in view of similar anatomical characteristics including surface density of hair follicles.

For occupational plutonium exposure, the sizes of the plutonium particles that would contaminate the skin would be mostly below 1.3 μm (Khodyreva *et al.*, 1975). On rubber gloves the particle sizes were higher, 0.5-20 μm. During maintenance work on a nuclear plant the surface density of the particles was found to be 15-100 cm^{-2}.

There appears to be enhanced absorption when the actinides are in organic solvents or when there is epidermal injury (as when the skin is burnt by acid solutions) (Lagerquist *et al.*, 1969; Ilyin *et al.*, 1982). These results emphasize the necessity for prompt decontamination treatment.

Passive diffusion plays a major role in skin penetration. Most of the plutonium is bound to the insoluble proteins. Americium behaves simi-

larly to plutonium. The mechanisms of Pu and Am loss from the skin include: (i) transfer into the microcirculation and then into the blood and lymphatic systems; (ii) transfer on to the skin surface with the secretions of the sudoriferous and sebaceous glands; and (iii) loss into the environment during desquamation.

Complexing agents are quite effective in decontaminating the skin although they can also enhance absorption into the body. It is therefore important that the complexing or chelating agent should not be metabolizable.

23(3).5.1 Conclusions Relevant to Radiation Protection

Animal studies and limited human experience suggest that for the intact skin the absorption of plutonium from dilute aqueous acid solution probably does not exceed 0.01% during the first hour after contamination. Absorption from organic solutions may be more rapid (~ 0.04% in 15 min.). Skin damage caused by strong acids can lead to increased absorption through the skin. The absorption of americium and neptunium seems to be similar to that of plutonium.

23(3).6 DISTRIBUTION AND RETENTION OF SYSTEMICALLY ABSORBED ACTINIDES

This section discusses the distribution and retention of actinides within the body once they have gained access to the blood stream. Particular attention is given to the liver and skeleton, the two principal sites of systemic deposition and retention. Attention is also given to the gonads, and cross-placental transport and deposition in the fetus. Since the publication of ICRP 19 extensive human and animal data have become available.

23(3).6.1 Human Data on Actinide Distribution and Retention

There are 3 sources of human data: (i) a small number of experimental injection cases dating from the earliest years of plutonium availability; (ii) a larger number of occupationally exposed cases that have come to autopsy; and (iii) large number of autopsies on persons exposed to fallout from nuclear weapon tests.

23(3).6.2 Experimental Injection Studies on Humans

Analysis of 7 cases (Durbin, 1976) gave mean partition among liver, skeleton, other tissues and excreta, of 30%, 50%, 10% and 10% respectively of the total plutonium burden. The vertebrae, ribs and sternum contained 2-4 times higher than average skeletal concentrations, while concentration in appendicular bones was 2-8 times lower; within a given bone, concentrations in trabecular regions were 2-5 times higher than in cortical regions (Larsen *et al.*, 1981b). After about 20 years following injection, initial surface deposits were entirely absent and the nuclide was distributed throughout the bone volume. There was, however, evidence of a secondary deposit on the endosteal surfaces of cortical bone, attributed to the recycling of plutonium released to blood in the remodelling of bone (Schlenker and Oltman, 1981). Among other tissues, 7% of the injected activity was found on the average in muscle, 1.7% in skin and 1.0% in lung at autopsy. The concentration in gonads was very small (0.04% in ovaries and 0.01% in testes), lower than that for fallout plutonium.

Recent re-analysis of the excretion data for these cases gives half-times of whole body retention of 40-100 years (Leggett *et al.*, 1984; Moss, 1985).

23(3).6.3 Occupational Exposure Studies

A number of workers in the nuclear industry with histories of actinide exposure have come to autopsy. ICRP 48 has critically reviewed the data. It is assumed that 80% of the initial plutonium deposit is retained at autopsy in liver plus skeleton. The partition between liver and skeleton is extremely variable. Mean values were: liver - 32% and skeleton-48%.

23(3).6.4 Fallout Exposure Studies

More than 900 autopsies have been carried out (McInroy *et al.*, 1979; Fox *et al.*, 1980). Weighted means for the percent distribution of total systemic deposit were: liver - 45%; skeleton - 35%; ovaries - 0.07%; testes - 0.13%. There was an increase in liver concentration correlated with age at death and a correlated decrease in skeletal content - suggestive of transfer from skeleton to liver or enhanced skeletal deposition at earlier ages. Concentration in the placenta is about half the average total body plutonium concentration (Mussalo *et al.*, 1980). There are large variations in the partition between liver and skeleton in the different samples.

The fallout data differ from the injection and occupational exposure cases in that exposure to fallout has occurred, and continues to occur, at a varying rate extending over 30 years. Analysis of the data provides some support for the ICRP lung model (45% deposition and 40 year half-time in liver; 45% deposition and 100 year half-time in skeleton).

23(3).6.5 Data on Actinide Distribution and Retention

Human data relating to actinides other than plutonium, their microdistribution within the critical organs, as well as cross-placental transfer are rare. ICRP 48 reviews the available human and animal data.

The ICRP 19 estimate of 40 years half-life for actinides in liver does not have experimental justification; similarly, the estimate of 100 years for retention half-time of plutonium in bone seems to be somewhat on the higher side. For americium, a skeletal half-life of 30 years is suggested in humans. Retention may be more a function of bone turnover rates than of the specific chemical properties of the metal; a retention half-time of the order of the normal life-span of the animal may be generally descriptive of this process (Taylor, 1983).

For radiation protection purposes we now calculate doses separately for bone marrow and for cells lining the endosteal bone surfaces. It is assumed in ICRP 30 that actinides are uniformly deposited on the surfaces of trabecular and cortical bone. Plutonium is a bone surface seeker and contrasts with the more uniform distribution of alkaline earths. We now know that actinides are not retained indefinitely on bone surfaces as assumed in the ICRP 30 bone model, but with time they approach a volume type distribution. If this trend is taken into account, the 50-year committed dose to bone surfaces would be about 6 times lower than the value given by the ICRP 30 model (Priest and Birchall, 1985). However, the dose to marrow would be reduced by a factor of only 3 (because of irradiation from plutonium contained in the marrow macrophages). This would lead to increases by a factor of 1.5 to 3 in the relevant ALI values for plutonium.

23(3).6.6 Gonads

ICRP 30 employed a deposition fraction of 3.5×10^{-4} for testes and 1.1×10^{-4} for ovaries. There is a large variability in testes deposition between species; in all cases, the concentration in testes is less than the average in the body. The concentration in the ovary relative to total body

is substantially higher than in testes. The elimination of actinides from gonads seems to be very little.

23(3).6.7 Cross-placental Transport

The subject is of interest in occupational exposure because of the employment of women who might be pregnant. Cross-placental transfer of plutonium in humans has been demonstrated qualitatively. Although data are not very conclusive, there seems to be no clear tendency for marked concentration of plutonium in the fetus; placenta and fetal membranes collect the plutonium that might otherwise have reached the fetus. Fetal concentration may be 0.1-1% of injected activity. For other actinides data are less extensive but indicate that they may be less transportable than plutonium.

23(3).7 GENERAL CONCLUSIONS

The general conclusions from ICRP 19 and ICRP 48 are summarized in the following.

23(3).7.1 Inhaled Actinides

Recent information on the behaviour of inhaled plutonium and other actinide compounds, in animals or in humans, is not always consistent with the assumptions of the current ICRP Lung Model.

23(3).7.2 Effect of Chemical Form

The rate of translocation of plutonium and other actinides, from the primary deposition site in lung, in muscle or in wounds, is influenced by the chemical and physical form of the deposited nuclide.

23(3).7.3 Absorption from the Gastro-intestinal Tract

Absorption is influenced by the mass ingested, by fasting, by incorporation into foodstuffs, by complexing anions such as citrate and DTPA, and by a variety of other factors. The animal data for what appears to be the same compound often show wide fluctuations, which suggests that the absorption may be critically dependent on the chemical microenvironment at the site of absorption.

The fractional absorption (f_1) values listed in Table 23(3).2 are regarded as the best estimates for purposes of radiation protection. The proposed value of $f_1 = 10^{-3}$ for unknown or mixed compounds of plutonium and other actinides is considered to provide an adequate margin of safety for radiation protection purposes. For comparison, the f_1 values used in the assessment of the ALIs for workers given in ICRP 30 are also listed in the last column of Table 23(3).2.

The use of the cautious value of 10^{-3} may not be appropriate in all situations; if a different value more suitable to the specific situation can be justified, it should be employed.

Plutonium absorption from the GI tract may be increased by at least an order of magnitude in the human neonate. This increased absorption appears likely to decrease very rapidly during the first few days or weeks of life. For the first year of life, a value of f_1 of 10^{-2} is suggested, with the adult value of 10^{-3} being applied to all succeeding years.

23(3).7.4 Retention in Liver and Bone

Liver and bone are the principal deposition sites and account for 80% of the plutonium which reaches the blood stream. The partition of plutonium between liver and skeleton varies widely in individual cases, but the most likely average deposition is considered to be 50% in the skeleton and 30% in the liver. This distribution appears to apply for americium and curium also. However, the variability of the deposition of plutonium, americium and curium between individuals is such that, for radiation protection purposes, the ICRP 30 model of equal distribution between skeleton and liver (45% in each) remains a satisfactory assumption. Animal data indicate that for the actinides of higher atomic number, i.e. californium, berkelium and einsteinium, an inital distribution of 65% in skeleton and 25% in liver would seem more appropriate; for neptunium the corresponding values would be 75% and 15%.

The partition of actinides between liver and skeleton in children and neonates is likely to be different from that observed in adults because of differences in metabolism.

For the gonads, the currently assumed values of 0.035% in testes and 0.011% in ovaries seem to be appropriate, although they give an impression of (unjustified) precision.

The retention half-times of 40 years in liver and 100 years in skeleton recommended for plutonium in ICRP 19 and employed in ICRP 30 are too long. Values of 20 and 50 years for liver and skeleton, respectively, seem more reasonable.

Perhaps retention half-time for americium is shorter than for plutonium; but human data are limited. There is no compelling argument for assuming that retention times for neptunium, americium or the higher actinides are substantially different from those of plutonium.

The assumption in ICRP 30 of an infinite retention half-time for actinides in testes and ovaries is a cautious one, consistent with animal data, although non-human primate studies suggest a relatively short half-time.

23(3).7.5 Embryo and Fetus

There is no strong evidence to show that actinides concentrate preferentially in the embryo or fetus, and such deposition as may occur will be diluted rapidly by growth. Control procedures that protect the mother should be fully protective of her fetus, even assuming some greater radiosensitivity of the fetus.

23(3).7.6 Microdistribution in the Skeleton

The present ICRP 30 assumption with regard to the microdistribution of actinides in the skeleton, and the assumption of uniform distribution in liver and gonads, are inconsistent with the limited human data and with the substantial body of animal data. The simplifying assumptions presently recommended by ICRP 30 would seem to overestimate the risk, and therefore, can be viewed as a cautious approach.

23(3).7.7 Effect of the Proposed Changes in f_1 and Retention Half-time in Liver and Bone on ALIs

For isotopes of plutonium of long physical half-life, the ALI for ingestion of unknown or mixed compounds will need to be decreased by almost a factor of 10. However, the ALIs for ingestion of long-lived isotopes of plutonium as nitrates or oxides, as well as those for the ingestion of isotopes of short physical half-life, where the dose to the intestinal mucosa is of overriding importance, will need little change. Similarly, the effect on ALIs for inhaled plutonium isotopes will also be small, since, in

this situation, transfer from GI tract to blood is very small in comparison with transfer from lung to blood. Values of ALI for ingested americium, curium and californium will be much less affected, since the proposed change in f_1 values is only by a factor of 2. For ^{237}Np, the ALI by ingestion will need to be increased by about an order of magnitude.

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Table 23(3).1: Summarized interspecies comparison of the fractional absorption of orally administered plutonium compounds
(from ICRP 48)

Compound	Species	Range of observed values	Fractional absorption x 10^4	
			Non-weighted median	Number of reported studies
Nitrate	Rat	0.1-12	1.0	29
	Mouse	0.6-17	0.9	7
	Hamster	0.1-2	0.2	8
	Guinea pig	2.7-5.5	4.4	4
	Beagle	2.3 & 3.5	2.9	2
	Pig	0.05-3.7	1.8	4
	Rabbit	0.88 & 0.94	0.9	2
Citrate	Rat	1.6-41	20.0	16
	Mouse	0.7-27	2.7	5
	Hamster	0.3-1	0.6	3
	Beagle	3.1-8.3	6.4	3
	Rabbit	2	2.0	1
Biologically incorporated plutonium	Rat	2.5-40	11.0	8
	Hamster	1	1.0	1
	Guinea pig	4.6 & 11	7.0	2
	Rabbit	0.4	0.4	1
Oxides, incl. polydisperse, monodisperse, mixed Na-Pu oxides	Rat	0.02-1.7	0.5	6
	Hamster	0.004	0.004	1
	Pig	0.0003-0.002	0.0009	3
	Cow	0.2 & 0.2	0.2	2

Table 23(3).2: Comparison of f_1 values : ICRP 48 and ICRP 30
(from ICRP 48)

Element	Type of exposure, compound	$f_1 \times 10^4$ ICRP 48	ICRP 30
	Occupational exposure		
	oxides, excluding "polydisperse" oxides	0.1	0.1
Pu	nitrates	1	1
	other compounds or unknown mixtures	10	1
	Population exposure (via food chains)		
	all compounds	10	
Np	Occupational and population exposure, all compounds	10	100
Am	Occupational and population exposure,		
Cm	all compounds	10	5
Cf			

CHAPTER 23: SECTION 4

REVISED BONE MODEL FOR ACTINIDES

23(4).1 INTRODUCTION

ICRP publication 56, 'Age-dependent doses to members of the public from intakes of radionuclides: Part 1 (1989) has a section describing a new biokinetic model for actinides.

Components of the actinide model given in ICRP 48 are mathematical expressions that were not intended to represent actual physiological processes involved in the retention and translocation of actinides in the body. In contrast to the assumptions of ICRP 48, there is much evidence indicating that a substantial portion of actinides deposited in bone surfaces gradually becomes volume distributed due to bone restructuring. Another portion is removed to bone marrow or to blood and recycled to a large extent to bone surfaces and liver. Most activity originally deposited in liver appears to be removed to blood and recycled systemically rather than being excreted directly.

23(4).2 AGE-SPECIFIC MODELS FOR PLUTONIUM, NEPTUNIUM AND AMERICIUM

Two physiologically based age-specific models for plutonium and one for americium have been developed (Leggett *et al.*, 1984; Leggett and Eckerman, 1984; Leggett and Warren, 1987; Priest, 1987). Both the plutonium models (Leggett *et al.* and Priest) are based on similar physiological considerations, do not differ greatly with regard to the assumed movements of radionuclides at different ages between bone surfaces, red marrow and liver, and produce similar dose estimates; they have, however, different subdivisions of the skeleton. The simpler model of Leggett has been adopted in ICRP 56.

23(4).3 PLUTONIUM MODEL

Figure 23(4).1 gives a simplified version of the compartments and directions of movements in the plutonium model of Leggett. (The simpli-

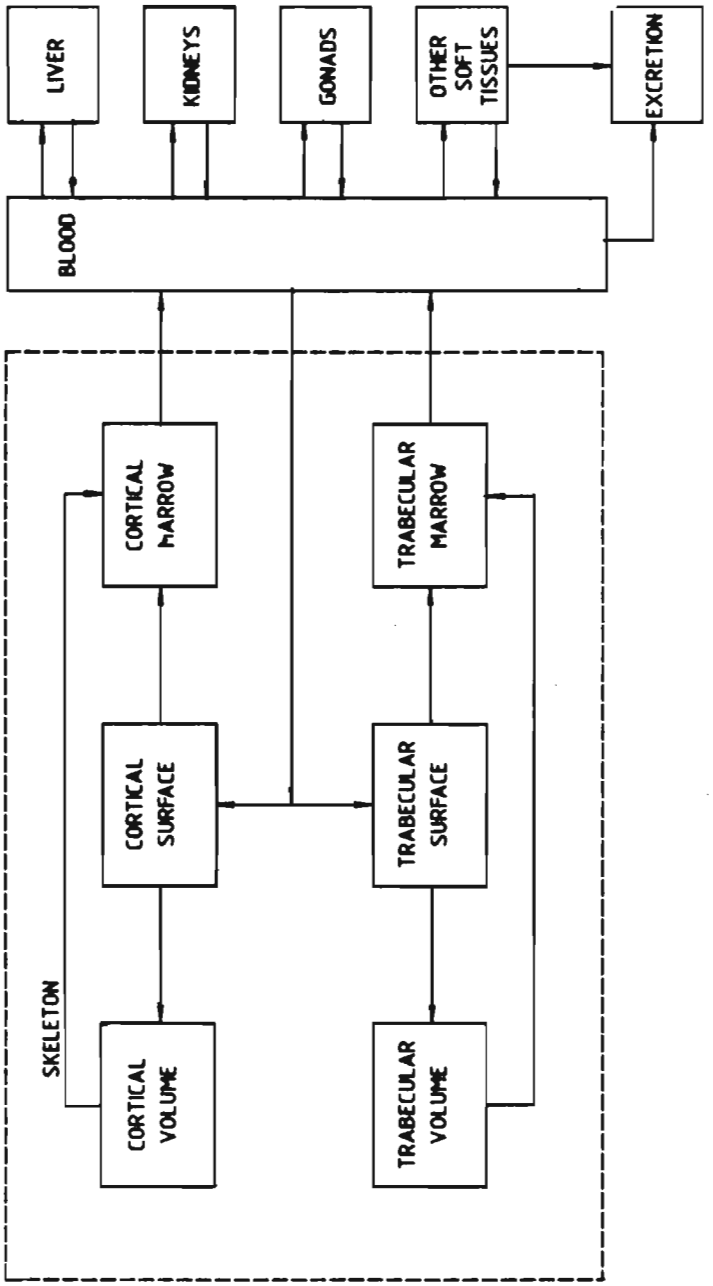

Figure 23(4).1. Simplified version of the compartments and directions of movement in the plutonium model of Leggett (from ICRP 56).

fications consist in considering the liver as a single pool of activity that is lost only to blood; and treatment of the excretion, kidney, and 'other soft tissue' compartments is simplified by giving less attention to specific pathways by which actinides are lost to excretion. Pathways from plasma to bone marrow for insoluble, colloidal, or polymeric material that has reached the blood are also not considered.) ICRP 56 gives tables of age-specific values for all the parameters shown in Fig.23(4).1 for ages of 3 months, 1, 5, 10 and 15 years as well as adults.

23(4).3.1 Initial Distribution of Plutonium

It is assumed for all the three actinides considered here that the GI absorption fractions are 0.01 for the 3-month old and 0.001 for all other ages. It is also assumed that skeletal uptake is 70% of the amount not promptly excreted for newborn, and 50% for adults. For 1-15 years of age, 60% is assumed. At all ages, the sum of the skeletal and hepatic fractions is assumed to be 80% of the amount not promptly excreted.

23(4).3.2 Uptake and Translocation by the Skeleton

Plutonium taken up from blood is deposited initially in cortical and trabecular bone surfaces, with highest deposition at sites with highly vascularized, red (haematopoietic) marrow and lowest at sites of yellow (fatty) marrow (pathways K and L) (Wronski *et al.*, 1980). In the adult, nearly all of the red marrow is in trabecular bone (Cristy, 1981). In children cortical bone is also active. It is assumed that 60% goes to trabecular bone and 40% to cortical bone in adults, while the corresponding figures are 50 and 50 for children.

Bone surfaces labelled with plutonium may remain unchanged, or they may be buried by formation of new bone (pathways A, C), or resorbed by osteoclasts (pathways B, D) (Jee, 1972a,b). If bone formation and resorption occurred on opposite phases of a bone segment (as in children), then the removal rate of plutonium from bone surface can be taken to be the sum of the resorption rate λ_1 (movement to bone marrow) and the f tion rate λ_2 (movement to bone volume) (Enlow, 1963). But if tion represented only the replacement of resorbed bone at the sar tion (Frost, 1976), then the removal rate would be approximately $\lambda_$ plutonium would be buried in volume only by (a) depositing in unminera osteoid and moving to the mineralized surface under the osteoid, or (b depositing and remaining in resorbed areas and eventually being covered

by bone. An intermediate scenario between (a) and (b) is assumed for both cortical and trabecular bone for adults; the burial rate in volume is taken to be $0.5\lambda_2$, and the removal rate from bone surface $\lambda_1 + 0.5\lambda_2$. In children λ_1 may be smaller than λ_2, while in older adults λ_1 may be slightly larger than λ_2. The conservative assumption is made here that $\lambda_1 = \lambda_2$.

Plutonium resorbed by osteoclasts may be released and concentrated by macrophages, mainly in marrow cavities (Jee, 1972a). A conservative value of 90 days is assumed for the half-time of retention of plutonium in macrophages at all ages.

Although the Leggett model assumes that bone remodelling is biased towards older bone, ICRP 56 assumes conservatively that modelling is a random process. The rate of removal from bone volume at a given age is assumed to be equal to the bone resorption at that age (pathways E, F). It is assumed that all activity removed from bone marrow is systemically recycled (pathways I, J). This is more likely than local redeposition on to bone surfaces (pathways G, H), especially in highly vascularized growing bone.

23(4).3.3 Plutonium in Liver

The liver is assumed to represent a uniformly mixed pool that exchanges activity only with blood, with a half-life of 10 years at all ages. Since activity is recycled between liver and skeleton, the net half-time in liver will be of the order of 40 years for some periods after exposure, depending on the age at exposure.

23(4).3.4 Plutonium in Kidneys, Other Soft Tissues and Excretion

Of the remaining unexcreted activity, 0.5% is assigned to kidneys, and 19.5% to 'other soft tissues', with removal half-times of 500 days (Leggett, 1985; Durbin, 1972). Activity removed from kidneys is assumed to be returned to blood, with urinary excretion being accounted for in other parts of the model. About 31% of activity leaving 'other soft tissue' goes directly to excretion, and 4% of plutonium reaching blood (including recycled plutonium) is rapidly excreted. The remaining 69% is returned to blood. Whole body retention would decline more rapidly in children because of more rapid movement of activity from skeleton to blood and subsequent removal of a portion of activity from blood to excretion.

23(4).3.5 Plutonium in Gonadal Tissues

Following ICRP 48, it is assumed that 0.035% of absorbed activity is taken up by testes and 0.011% by ovaries in adults. For non-adult males, it is assumed that fractional uptake from blood per gram of testes is two times greater than in adults. The fractional uptake per gram of ovaries is taken to be the same for all ages. The removal half-time from gonads to blood is assumed to be 10 years for all ages.

23(4).3.6 Recycling of Activity

Plutonium reaching the blood stream after removal from any tissue is assumed to trace the same pathways as the initial deposit in blood. It is assumed that plutonium leaves blood with an effective half-time of 0.85 days.

23(4).4 MODIFICATIONS TO THE PLUTONIUM MODEL FOR APPLICATION TO AMERICIUM

The salient features are summarized in the following:

(a) Amounts of unexcreted americium going to liver plus skeleton is 80% for all ages, but the partition between liver and skeleton varies with age: 50:30 for adults, 10:70 for newborn, and 30:50 for 1-15 year olds (Lloyd *et al.*, 1972; Durbin and Schmidt, 1985; Griffith *et al.*, 1983).

(b) Urinary excretion is greater for americium than for plutonium (Lloyd *et al.*, 1978a,b). A fraction 0.07 of americium in blood goes directly to excretion.

(c) In the first few years after exposure, apparent half-time for outflow from liver is 2-8 years (Griffith *et al.*, 1983; Durbin and Schmidt, 1985). At times remote from exposure, there is a greater feedback from skeleton to liver, leading to a larger apparent half-time (Leggett, 1989).

(d) 2% of unexcreted americium is deposited in kidneys, from which it is removed with a half-time of 100 days. Rate of removal from 'other soft tissue', as well as the parameter values for gonads are the same as for plutonium. The removal rate from blood is two orders of magnitude higher for americium than for plutonium (Turner and Taylor, 1968).

23(4).5 MODIFICATIONS OF THE MODEL FOR APPLICATION TO NEPTUNIUM

The salient features are summarized as follows:

(a) For the adult, 50% of absorbed neptunium is deposited in the skeleton, 10% in liver, 2% in kidneys, 3% in 'other soft tissue', remaining being excreted immediately in urine. The corresponding values for other ages are:

Newborn: 80%, 3%, 2%, 3%;

1-15 years of age: 65%, 5%, 2%, 3%.

(b) Neptunium is deposited on bone surfaces and formation of aggregates in marrow following remodelling is evident (Nenot *et al.*, 1972; NCRP, 1988). Neptunium bone data resemble those of americium and strontium more than of plutonium (Durbin *et al.*, 1986). The initial partition of neptunium between trabecular and cortical bone is assumed to be intermediate between those for americium and strontium (55:45 for adults; 40:60 for 15 year olds; and 35:65 for 0-10 year olds). Rate of movement among bone surfaces, bone volume and bone marrow due to bone restructuring, and the rate of removal from marrow to blood, are assumed to be the same as for plutonium.

(c) Loss of neptunium from liver to blood is assumed to occur with a half-life of only 2 years for all ages. At times far removed from the acute exposure, this may increase to 15 years.

(d) Parameter values for gonads are the same as for plutonium. Removal half-times from kidneys and 'other soft tissue' are the same as for americium. Removal half-time from blood is taken to be 0.25 days (ICRP 30, ICRP 48).

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CHAPTER 24

DOSIMETRIC MODEL FOR SUBMERSION IN A RADIOACTIVE CLOUD

24.1 INTRODUCTION

When a person is submerged in a radioactive gas, the skin and other organs may be irradiated both by external irradiation and by internal irradiation from gas absorbed into body tissues. The respiratory system and other organs may also be irradiated by gas contained in the lungs.

24.2 RELATIVE MAGNITUDES OF DOSE-EQUIVALENT RATES FROM EXTERNAL AND INTERNAL RADIATION

Consider a person submerged in a radioactive cloud of infinite extent and of volume concentration C Bq m^{-3}. Let the dose-equivalent rate to any tissue from external radiation be $\dot{H}_E$, from internal irradiation by absorbed gas be $\dot{H}_A$, and to the lung from contained gas be $\dot{H}_L$.

Then $\dot{H}_E$, in a small element of tissue in a person submerged in a radioactive cloud of infinite extent, is given by

$$\dot{H}_E = C\,s\,k\,g_E/\rho_A \quad \text{Sv h}^{-1}$$

where ρ_A, the density of air, is about 1300 g m^{-3}; s (in Sv h^{-1}) is the dose-equivalent rate in a small element of any medium of infinite extent uniformly contaminated at a concentration of 1 Bq g^{-1}; k, which is usually close to unity, is the mass stopping power of radiations in tissue relative to their mass stopping power in air; and g_E is a geometrical factor to allow for shielding by overlying tissues.

The value of g_E is always zero for beta emissions from tritium and for all alpha particle emissions, since these are unable to reach any of the sensitive tissues of the body, including the lenses of the eyes and the basal layers of the epidermis, which, for the purposes of dosimetry, are taken to be at depths of 3 mm and 70 μm respectively (ICRP 26). For most beta

emissions and for low energy photons, g_E is about 0.5 for tissues near the surface of the body and tends to zero for deep-lying tissues. For very penetrating photons, g_E approaches unity for all tissues of the body.

After prolonged exposure to the cloud an equilibrium is reached between the concentrations of the gas in air and tissue. Under these conditions it may be shown that the concentration of gas in tissue C_T is given by:

$$C_T = \delta C / \rho_T \quad \text{Bq g}^{-1}$$

where ρ_T, the density of tissue, is about 10^6 g m^{-3}, and δ is the solubility of the gas in tissue expressed as the volume of gas in equilibrium with unit volume of tissue at normal atmospheric pressure.

This solubility coefficient increases with the atomic weight of the gas; for example, in water at body temperature its value varies from about 0.02 for hydrogen to about 0.1 for xenon (Kaye and Laby, 1956). These values may be increased by a factor of 3-20 in adipose tissue (Lawrence *et al.*, 1946). Thus the dose-equivalent rate in tissue from absorbed gas, $\dot{H}_A$, is given by:

$$\dot{H}_A = s\ C g_A / \rho_T \quad \text{Sv h}^{-1}$$

where g_A is a geometric factor determined by the dimensions of a person and the range of the radiations concerned. For alpha and beta emissions and also for low energy photons g_A will be approximately unity for tissues at the centre of the body and 0.5 for surface tissues. For more energetic photons, g_A is much less than 1 for all tissues and decreases with increasing photon energy.

The dose-equivalent rate in the lung from contained gas, $\dot{H}_L$, is given by:

$$\dot{H}_L = sCV_L g_L / M_L \quad \text{Sv h}^{-1}$$

where V_L, the average volume of air contained in the lungs, is about 3×10^{-3} m^3; M_L, the mass of the lungs, is taken to be 1000 g; g_L is a geometrical factor which, for alpha and beta emissions and for low energy photons, is approximately unity. The value of g_L decreases with increasing photon energy.

24.2.1 Tritium

For tritium $\dot{H}_E$ is zero for all relevant tissues of the body because of the short range of tritium beta emissions in tissue. The ratio of dose-equivalent rate in any tissue from absorbed gas to that in the lung from contained gas is, from the above equations, given by:

$$(\dot{H}_A)/(\dot{H}_L) = (\delta g_A M_L)/(V_L g_L \rho_T)$$

Since $\rho_T \sim 10^6$ g m^{-3} and $M_L/V_L = 10^6/3$ g m^{-3}, while g_A and g_L are both approximately unity for tritium beta emissions, the expression reduces to:

$$(\dot{H}_A)/(\dot{H}_L) \sim (\delta/3)$$

For tritium δ is about 0.02 for aqueous tissues and 0.05 for adipose tissues (Lawrence *et al.*, 1946). Thus the dose-equivalent rate in lung from the tritium gas contained within it will be 60 to 150 times that in any tissue from absorbed gas. Therefore, submersion in tritium gas is limited solely by consideration of dose-equivalent rate in the lung. However, the limit on exposure to tritiated water is very much less than that for elemental tritium and in most cases in practice exposure to tritiated water will be the limiting factor.

24.2.2 Radon and Thoron

Limits for exposure to radon and thoron are dealt with in ICRP 34 (see Chapter 42).

24.2.3 The Noble Gases

All the radioisotopes of the noble gases argon, krypton and xenon, emit either photons or beta particles of considerable energy. Thus, for tissues near the surface of the body, including the skin, g_E will be about 0.5. We then get:

$$\frac{\dot{H}_E}{\dot{H}_L} = \frac{M_L k g_E}{V_L g_L \rho_A} \geq \frac{130}{g_L}$$

and since g_L cannot be greater than unity, $\dot{H}_E$ is more than 130 times $\dot{H}_L$. Similarly,

$$\frac{\dot{H}_E}{\dot{H}_A} = \frac{\rho_T k g_E}{\rho_A \delta g_A}$$

Since $\delta \leqslant 2$ (Lawrence *et al.*, 1946), $\rho_T / \rho_A \sim 800$, and $g_A \leqslant 1$, $\dot{H}_E$ is more than 200 times $\dot{H}_A$.

Thus it is clear that for exposure by submersion in radioisotopes of the noble gases, external irradiation will be of such overriding importance that it alone need be considered, and the doses from absorbed gas and gas contained in the lung can be disregarded.

24.3 DOSE EQUIVALENT RATES IN BODY TISSUES FROM SUBMERSION

24.3.1 Photon Emitters

From the data on energy spectra from sources of mono-energetic photons in an infinite extent of air (Dillman, 1971), the dose equivalent rate $\dot{H}$ (Sv/h) to organs and tissues may be calculated for a Reference Man situated within a semi-infinite cloud bounded by the floor on which he stands (Poston and Snyder, 1974). The dose rate in the lens is assumed to be the same as that in the skin, taken to extend from 0-2 mm depth (ICRP 23).

For a cloud of finite dimensions as in a room, the dose rate in skin or internal organs can be approximately taken to be:

$$2\dot{H} \left[1 - \exp\left(-\mu_A \rho_A r \right) \right]$$

where $\dot{H}$ is the dose rate estimated for a semi-infinite cloud and the factor 2 is used because for most room sizes the floor no longer limits irradiation by the cloud to a 2π geometry, at least for the head of a standing worker; μ_A is the mass energy absorption coefficient in air; ρ_A is the density of air; and r is the effective radius of the room. $[1-\exp(\mu_A \rho_A r)]$ is the factor by which the first interaction dose from an infinite cloud is reduced, to allow for the fact that there are no sources of photons outside the room.

In ICRP 30 computations have been made for the dose rates for submersion in a semi-infinite cloud and in rooms of volume 1000 m^3, 500 m^3 and 100 m^3.

24.3.2 Electrons and Beta Emitters

The dose rate in the skin at a depth of 70 μm and in the lens at a depth of 3 mm from a semi-infinite cloud of electrons is computed by integrating the point kernel of Berger (1971, 1974).

24.4 DERIVED AIR CONCENTRATION (DAC) FOR SUBMERSION

As discussed earlier, exposure to elemental tritium in air in any year is limited by consideration of stochastic effects in the lung as follows:

$$W_{Lung}\ \dot{H}_{Lung} \int C(t)\ dt \leq 0.05 \qquad \text{Sv}$$

where W_{Lung} is the weighting factor for lung; $\dot{H}_{Lung}$ [in {(Sv/h)/(Bq/m^3)}] is the dose equivalent rate to lung from exposure to unit concentration of tritium in air (i.e. 1 Bq/m^3), and $C(t)$ (in Bq/m^3) is the concentration of elemental tritium in air at any time t and the limits on integration are over a working year.

Exposure to an inert radioactive gas in any year is limited by consideration of external radiation of the body as follows:

$$W_T \dot{H}_T \int C(t)\, dt \leq 0.05 \qquad \text{Sv}$$

and

$$\dot{H}_T \int C(t)\, dt \leq 0.5 \qquad \text{Sv}$$

and

$$\dot{H}_{Lens} \int C(t)\, dt \leq 0.3 \qquad \text{Sv}$$

where $\dot{H}_T$ [in {(Sv/h)/(Bq/m^3)}] is the dose equivalent rate in any tissue T; and $\dot{H}_{Lens}$ is the corresponding value for the lens of the eye, resulting from submersion of Reference Man in unit concentration of the inert gas in air (Bq/m^3); $C(t)$ (in Bq/m^3) is the concentration of the inert radioactive gas in air at any time t and the limits on integration are over a working year.

For convenience, ICRP recommends values of DAC which are 1/2000th of the greatest value of $\int C(t)\, dt$ which satisfies the appropriate equation given above for tritium or an inert radioactive gas. When the DAC is determined by non-stochastic effects, the organ or tissue concerned (usually the skin) is named below the value of DAC in the tabulations and a greater value of DAC (determined by stochastic effects) is shown in pa-

rentheses. This value of stochastic limit DAC is useful when considering the limitation of exposure from several sources.

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CHAPTER 25

DOSIMETRIC DATA AND ALI VALUES FOR SOME IMPORTANT RADIONUCLIDES

The following tables give dosimetric data and ALI values for some radionuclides of importance. They include:

(i) All the radionuclides for which data are given in ICRP 54. These were selected for their potential importance in occupational exposure within the nuclear industry, in research, and in medical procedures;

(ii) All the radionuclides for which data are given in ICRP 56. These are among the most radiologically significant radionuclides that might be released to the environment due to various human activities, mainly connected with the different phases of the nuclear fuel cycle. ICRP 56 deals with only Part 1 of the total list of such radionuclides;

(iii) Certain other important radionuclides.

Table 25.1 gives the dose rates for submersion in a semi-infinite cloud of radioactive vapours and gases for some important radionuclides. Effective dose rates $\dot{E}$, expressed in [(Sv/h)/(Bq/m^3)], the organ or tissue, T, getting the highest dose, and the dose rate to that organ, $\dot{H}_T$, expressed in [(Sv/h)/(Bq/m^3)] are tabulated. The data are taken from the Supplements to ICRP 30.

Table 25.2 gives the f_1 values, committed effective doses, $E(50)$, committed equivalent doses to the organ or tissue T receiving the highest dose, $H_T(50)$, both expressed in (Sv/Bq), and the annual limit on intake, (ALI), expressed in Bq, for ingestion. Table 25.3 lists similar values for inhalation of vapours, class D, class W, and class Y materials.

The f_1 and ALI values are from ICRP 61. There is no change in f_1 values between ICRP 30 and ICRP 61. ICRP 30 and its Supplements do not give directly the effective doses (except in ICRP 30, Part 4, dealing with the transuranics). The Supplements, however, give the committed equivalent doses as well as the weighted committed equivalent doses to all the organs that get a substantial dose. By adding the weighted committed equivalent doses one can arrive at the effective dose commitments.

The organ dose values will not change as a result of any changes in dose limits brought out in ICRP 60. The effective doses may change somewhat, because of the changes in tissue weighting factor, W_T, between ICRP 26 and ICRP 60. However, as mentioned in ICRP 61, the effective dose is a robust quantity and the variations will not be substantial. ALI values will be revised substantially downwards, mainly because of the downward revision of the annual stochastic dose limits from 50 mSv to 20 mSv; one can say, as a first approximation, that the ALI values (which are mostly determined by stochastic effects) will be 2.5 times smaller as per the new ICRP 60 recommendations and as given in ICRP 61, as compared to the ICRP 30 values based on ICRP 26 recommendations.

Where the ALI is determined by the non-stochastic dose limits to a given organ, an asterik is put after the ALI value in all the tables.

Brief Analysis of ALI Values Given in ICRP 61

Table 25.4 gives a summary analysis of the ALI values reported in ICRP 61. The lowest values for inhalation are: 20 Bq for ^{227}Ac, class D; 10 Bq for ^{250}Cm, class W; and 60 Bq for ^{229}Th, class Y. For ingestion, the lowest value is 2000 Bq for ^{250}Cm. The highest values for both inhalation and ingestion are in the range of 10^{10} Bq.

Metabolic Data for Selected Elements and their Radionuclides

Table 25.5 gives the metabolic data for the selected elements and their radionuclides for which dosimetric data have been given in the above tables. The information has been summarized from ICRP 30, ICRP 54 and ICRP 56.

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Table 25.1: Dose rates for submersion in a semi-infinite cloud of radioactive vapours and gases for some important radionuclides
(based on ICRP 30)

Radionuclide	$\dot{E}$ (Sv/h)/(Bq/m^3)	Organ/ tissue T	$\dot{H}_T$ (Sv/h)/(Bq/m^3)
Elemental tritium	1.2×10^{-15}	Lung	9.9×10^{-15}
^{41}Ar	2.2×10^{-10}	Skin	3.9×10^{-10}
^{85}Kr	4.6×10^{-13}	Skin	4.7×10^{-11}
^{133}Xe	6.1×10^{-12}	Skin	1.9×10^{-11}

$\dot{E}$: effective dose rate for submersion in unit concentration of the radionuclide in air, T: organ or tissue getting the highest dose rate, and $\dot{H}_T$: equivalent dose rate to T.

Table 25.2: f_1 values, committed effective doses $E(50)$, committed equivalent doses to organs or tissues receiving the highest dose $H_T(50)$, and ALI values for ingestion of some important radionuclides

Radionuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
12.4 y ^{3}H Tritiated water	1.0	1.7×10^{-11}	All organs get same dose	All organs get same dose	1×10^{9}
5730 y ^{14}C Organic compounds	1.0	5.6×10^{-10}	All organs get same dose	All organs get same dose	4×10^{7}
2.6 y ^{22}Na	1.0	3.2×10^{-9}	Bone surface	5.5×10^{-9}	7×10^{6}
15.0 h ^{24}Na	1.0	3.9×10^{-10}	Stomach wall	1.2×10^{-9}	5×10^{7}
14.3 d ^{32}P	0.8	2.1×10^{-9}	Red marrow	8.1×10^{-9}	8×10^{6}
87.4 d ^{35}S	0.8	1.2×10^{-10}	LLI wall	5.7×10^{-10}	1×10^{8}
	0.1	1.8×10^{-10}	LLI wall	2.2×10^{-9}	7×10^{7}
27.7 d ^{51}Cr	0.1	3.6×10^{-11}	LLI wall	2.5×10^{-10}	5×10^{8}
	0.01	3.5×10^{-11}	LLI wall	2.7×10^{-10}	4×10^{8}
312.5 d ^{54}Mn	0.1	7.3×10^{-10}	LLI wall	2.2×10^{-9}	3×10^{7}
2.7 y ^{55}Fe	0.1	1.6×10^{-10}	Spleen	5.6×10^{-10}	1×10^{8}

(*contd ...*)

Radio-nuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
44.5 d					
^{59}Fe	0.1	1.8×10^{-9}	LLI wall	8.4×10^{-9}	1×10^{7}
270.9 d					
^{57}Co	0.05	1.8×10^{-10}	LLI wall	1.3×10^{-9}	9×10^{7}
	0.3	3.1×10^{-10}	LLI wall	1.1×10^{-9}	6×10^{7}
70.8 d					
^{58}Co	0.05	7.8×10^{-10}	LLI wall	4.0×10^{-9}	2×10^{7}
	0.3	9.4×10^{-10}	LLI wall	3.3×10^{-9}	2×10^{7}
5.3 y					
^{60}Co	0.05	2.7×10^{-9}	LLI wall	1.1×10^{-8}	7×10^{6}
	0.3	7.0×10^{-9}	LLI wall	1.4×10^{-8}	3×10^{6}
243.9 d					
^{65}Zn	0.5	3.9×10^{-9}	LLI wall	5.0×10^{-9}	5×10^{6}
78.3 h					
^{67}Ga	0.001	1.9×10^{-10}	LLI wall	1.6×10^{-9}	8×10^{7}
119.8 d					
^{75}Se	0.8	2.6×10^{-9}	Kidneys	7.2×10^{-9}	9×10^{6}
	0.05	4.6×10^{-10}	LLI wall	1.8×10^{-9}	4×10^{7}
64.8 d					
^{85}Sr	0.3	5.3×10^{-10}	LLI wall	1.5×10^{-9}	4×10^{7}
	0.01	3.7×10^{-10}	LLI wall	1.8×10^{-9}	5×10^{7}

(*contd ...*)

Radio-nuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
50.5 d ^{89}Sr	0.3	2.2×10^{-9}	LLI wall	2.1×10^{-8}	6×10^{6}
	0.01	2.3×10^{-9}	LLI wall	2.9×10^{-8}	6×10^{5}*
29.1 y ^{90}Sr	0.3	3.6×10^{-8}	Bone surface	4.2×10^{-7}	6×10^{5}*
	0.01	3.2×10^{-9}	LLI wall	2.6×10^{-8}	5×10^{6}
64.0 d ^{95}Zr	0.002	9.2×10^{-10}	LLI wall	7.8×10^{-9}	2×10^{7}
35.2 d ^{95}Nb	0.01	6.1×10^{-10}	LLI wall	4.0×10^{-9}	3×10^{7}
66.0 h ^{99}Mo	0.8	8.2×10^{-10}	LLI wall	3.1×10^{-9}	3×10^{7}
	0.05	1.2×10^{-9}	LLI wall	1.4×10^{-8}	1×10^{7}*
6.0 h ^{99m}Tc	0.8	1.6×10^{-11}	Thyroid	8.5×10^{-11}	1×10^{9}
39.3 d ^{103}Ru	0.05	7.3×10^{-10}	LLI wall	6.5×10^{-9}	2×10^{7}
368.2 d ^{106}Ru	0.05	5.8×10^{-9}	LLI wall	7.1×10^{-8}	2×10^{7}
249.9 d ^{110m}Ag	0.05	2.9×10^{-9}	LLI wall	1.1×10^{-8}	7×10^{6}
2.8 d ^{111}In	0.02	3.0×10^{-10}	LLI wall	2.0×10^{-9}	5×10^{7}
1.7 h ^{113m}In	0.02	2.7×10^{-11}	SI wall, Stomach wall	1.3×10^{-10}	9×10^{8}

(*contd* ...)

Radio-nuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
115.1 d ^{113}Sn	0.02	7.4×10^{-10}	LLI wall	7.9×10^{-9}	2×10^{7}*
60.2 d ^{124}Sb	0.1	2.4×10^{-9}	LLI wall	2.1×10^{-8}	6×10^{6}
	0.01	2.5×10^{-9}	LLI wall	2.3×10^{-8}	6×10^{6}
2.8 y ^{125}Sb	0.1	6.6×10^{-10}	LLI wall	5.8×10^{-9}	2×10^{7}
	0.01	7.0×10^{-10}	LLI wall	6.3×10^{-9}	2×10^{7}
13.2 h ^{123}I	1.0	1.3×10^{-10}	Thyroid	4.4×10^{-9}	9×10^{7}*
60.1 d ^{125}I	1.0	1.0×10^{-8}	Thyroid	3.4×10^{-7}	1×10^{6}*
1.6×10^{7} y ^{129}I	1.0	7.4×10^{-8}	Thyroid	2.5×10^{-6}	2×10^{5}*
8.0 d ^{131}I	1.0	1.4×10^{-8}	Thyroid	4.8×10^{-7}	8×10^{5}*
2.1 y ^{134}Cs	1.0	2.0×10^{-8}	Soft tissue	2.3×10^{-8}	1×10^{6}
30.0 y ^{137}Cs	1.0	1.4×10^{-8}	Soft tissue	1.5×10^{-8}	1×10^{5}
12.7 d ^{140}Ba	0.1	2.3×10^{-9}	LLI wall	2.6×10^{-8}	6×10^{6}*

(*contd ...*)

Radio-nuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
284.3 d ^{144}Ce	$3x10^{-4}$	$5.3x10^{-9}$	LLI wall	$6.6x10^{-8}$	$2x10^{6}$*
13.4 d ^{143}Pr	$3x10^{-4}$	$1.2x10^{-9}$	LLI wall	$1.5x10^{-8}$	$1x10^{7}$*
2.6 y ^{147}Pm	$3x10^{-4}$	$2.5x10^{-10}$	LLI wall	$3.2x10^{-9}$	$5x10^{7}$*
32.0 d ^{169}Yb	$3x10^{-4}$	$7.6x10^{-10}$	LLI wall	$7.1x10^{-9}$	$2x10^{7}$
74.0 d ^{192}Ir	0.01	$1.4x10^{-9}$	LLI wall	$1.3x10^{-8}$	$1x10^{7}$
2.7 d ^{198}Au	0.1	$1.2x10^{-9}$	LLI wall	$1.1x10^{-8}$	$1x10^{7}$
64.1 h ^{197}Hg					
Organic compounds	1.0	$1.4x10^{-10}$	Kidneys	$9.6x10^{-10}$	$2x10^{8}$
	0.4	$2.0x10^{-10}$	LLI wall	$1.5x10^{-9}$	$9x10^{7}$
Inorganic compounds	0.02	$2.4x10^{-10}$	LLI wall	$2.4x10^{-9}$	$6x10^{7}$
46.6 d ^{203}Hg					
Organic compounds	1.0	$2.7x10^{-9}$	Kidneys	$1.9x10^{-8}$	$1x10^{7}$
	0.4	$1.5x10^{-9}$	Kidneys	$7.5x10^{-9}$	$2x10^{7}$
Inorganic compounds	0.02	$5.7x10^{-10}$	LLI wall	$5.5x10^{-9}$	$3x10^{7}$

(*contd* ...)

Radio-nuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
3.0 d ^{201}Tl	1.0	7.8×10^{-11}	Kidneys	2.7×10^{-10}	3×10^{8}
138.4 d ^{210}Po	0.1	4.4×10^{-7}	Spleen	4.4×10^{-6}	9×10^{4}
1600 y ^{226}Ra	0.2	3.1×10^{-7}	Bone surface	6.8×10^{-6}	9×10^{4}*
5.8 y ^{228}Ra	0.2	3.3×10^{-7}	Bone surface	5.8×10^{-6}	9×10^{4}*
1.9 y ^{228}Th	2×10^{-4}	1.0×10^{-7}	Bone surface	2.4×10^{-6}	3×10^{5}*
1.4×10^{10} y ^{232}Th	2×10^{-4}	7.4×10^{-7}	Bone surface	1.9×10^{-5}	5×10^{4}*
1.6×10^{5} y ^{233}U	0.05	7.2×10^{-8}	Bone surface	1.2×10^{-6}	7×10^{5}*
	0.002	6.8×10^{-9}	LLI wall	5.0×10^{-8}	3×10^{6}
2.5×10^{5} y ^{234}U	0.05	7.1×10^{-8}	Bone surface	1.1×10^{-6}	7×10^{5}*
	0.002	6.7×10^{-9}	LLI wall	4.9×10^{-8}	3×10^{6}
7.0×10^{8} y ^{235}U	0.05	6.8×10^{-8}	Bone surface	1.0×10^{-6}	7×10^{5}*
	0.002	6.8×10^{-9}	LLI wall	5.3×10^{-8}	3×10^{6}
4.5×10^{9} y ^{238}U	0.05	6.3×10^{-8}	Bone surface	1.0×10^{-6}	8×10^{5}*
	0.002	6.1×10^{-9}	LLI wall	4.6×10^{-8}	3×10^{6}

(*contd* ...)

Radio-nuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
2.1×10^6 y ^{237}Np	0.001	1.1×10^{-6}	Bone surface	2.7×10^{-5}	3×10^4*
2.4 d ^{239}Np	0.001	8.0×10^{-10}	LLI wall	8.7×10^{-9}	2×10^7*
87.7 y ^{238}Pu	1×10^{-3}	8.5×10^{-7}	Bone surface	1.6×10^{-5}	4×10^4*
	1×10^{-4}	8.5×10^{-8}	Bone surface	1.6×10^{-6}	3×10^5*
	1×10^{-5}	1.3×10^{-8}	Bone surface	1.6×10^{-7}	2×10^6*
2.4×10^4 y ^{239}Pu	1×10^{-3}	9.6×10^{-7}	Bone surface	1.8×10^{-5}	4×10^4*
	1×10^{-4}	9.6×10^{-8}	Bone surface	1.8×10^{-6}	3×10^5*
	1×10^{-5}	1.4×10^{-8}	Bone surface	1.8×10^{-7}	2×10^6*
6.5×10^3 y ^{240}Pu	1×10^{-3}	9.6×10^{-7}	Bone surface	1.8×10^{-5}	4×10^4*
	1×10^{-4}	9.6×10^{-8}	Bone surface	1.8×10^{-6}	3×10^5*
	1×10^{-5}	1.4×10^{-8}	Bone surface	1.8×10^{-7}	2×10^6*
14.4 y ^{241}Pu	1×10^{-3}	1.8×10^{-8}	Bone surface	3.5×10^{-7}	2×10^6*
	1×10^{-4}	1.8×10^{-9}	Bone surface	3.5×10^{-8}	2×10^7*
	1×10^{-5}	2.0×10^{-10}	Bone surface	3.5×10^{-9}	2×10^8*
432.2 y ^{241}Am	0.001	9.7×10^{-7}	Bone surface	1.8×10^{-5}	3×10^4*

(*contd ...*)

Radio-nuclide	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
162.8 d ^{242}Cm	0.001	2.8×10^{-8}	Bone surface	4.5×10^{-7}	9×10^{5}*
18.1 y ^{244}Cm	0.001	5.4×10^{-7}	Bone surface	9.8×10^{-6}	6×10^{4}*
2.6 y ^{252}Cf	0.001	2.7×10^{-7}	Bone surface	5.8×10^{-6}	1×10^{5}*

* ALI determined by deterministic dose.
ALI and f_1 values from ICRP 61; $E(50)$ and $H_T(50)$ values from ICRP 30.

Table 25.3: Values of f_1, committed effective doses $E(50)$, committed equivalent doses to organs or tissues receiving the highest dose $H_T(50)$, and ALI values for inhalation of different classes of some important radionuclides

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
12.4 y ^{3}H						
Tritiated water	V	1.0	1.7×10^{-11}	All organs get same dose		1×10^{9}
5730 y ^{14}C						
Organic compounds	V	1.0	5.6×10^{-10}	All organs get same dose		4×10^{7}
^{14}C monoxide	V	1.0	7.8×10^{-13}	All organs get same dose		3×10^{10}
^{14}C dioxide	V	1.0	6.4×10^{-12}	All organs get same dose		3×10^{9}
2.6 y ^{22}Na	D	1.0	2.2×10^{-9}	Bone surface	3.5×10^{-9}	1×10^{7}
15.0 h ^{24}Na	D	1.0	2.6×10^{-10}	Lungs	1.2×10^{-9}	6×10^{7}
14.3 d ^{32}P	D	0.8	1.5×10^{-9}	Red marrow	6.0×10^{-9}	1×10^{7}
	W	0.8	3.6×10^{-9}	Lungs	2.6×10^{-8}	5×10^{6}
87.4 d ^{35}S	D	0.8	7.7×10^{-11}	Lungs	2.0×10^{-10}	2×10^{8}
	W	0.8	6.1×10^{-10}	Lungs	5.1×10^{-9}	3×10^{7}
	V	1.0	9.5×10^{-11}	All organs get same dose		
27.7 d ^{51}Cr	D	0.1	2.9×10^{-11}	LLI wall	5.9×10^{-11}	7×10^{8}

(*contd ...*)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
27.7 d ^{51}Cr	W	0.1	5.7×10^{-11}	Lungs	3.8×10^{-10}	3×10^{8}
	Y	0.1	7.1×10^{-11}	Lungs	5.3×10^{-10}	2×10^{8}
312.5 d ^{54}Mn	D	0.1	1.5×10^{-9}	Liver	4.6×10^{-9}	2×10^{7}
	W	0.1	1.7×10^{-9}	Lungs	6.7×10^{-9}	1×10^{7}
2.7 y ^{55}Fe	D	0.1	6.9×10^{-10}	Spleen	2.8×10^{-9}	3×10^{7}
	W	0.1	3.3×10^{-10}	Lungs	1.1×10^{-9}	6×10^{7}
44.5 d ^{59}Fe	D	0.1	4.0×10^{-9}	Spleen	8.3×10^{-9}	5×10^{6}
	W	0.1	2.7×10^{-9}	Lungs	1.4×10^{-8}	6×10^{6}
270.9 d ^{57}Co	W	0.05	4.9×10^{-10}	Lungs	4.1×10^{-9}	3×10^{7}
	Y	0.05	2.0×10^{-9}	Lungs	1.7×10^{-8}	8×10^{6}
70.8 d ^{58}Co	W	0.05	1.2×10^{-9}	Lungs	7.9×10^{-9}	1×10^{7}
	Y	0.05	1.9×10^{-9}	Lungs	1.6×10^{-8}	7×10^{6}
5.3 y ^{60}Co	W	0.05	8.0×10^{-9}	Lungs	3.6×10^{-8}	2×10^{6}
	Y	0.05	4.1×10^{-8}	Lungs	3.4×10^{-7}	4×10^{5}
243.9 d ^{65}Zn	Y	0.5	5.0×10^{-9}	Lungs	2.1×10^{-8}	4×10^{6}
78.3 h ^{67}Ga	D	0.001	9.5×10^{-11}	Bone Surface	4.0×10^{-10}	2×10^{8}
	W	0.001	1.3×10^{-10}	LLI wall	5.7×10^{-10}	1×10^{8}

(*contd ...*)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
119.8 d ^{75}Se	D	0.8	1.9×10^{-9}	Kidneys	5.4×10^{-9}	1×10^{7}
	W	0.8	2.2×10^{-9}	Lungs	5.4×10^{-9}	1×10^{7}
64.8 d ^{85}Sr	D	0.3	5.2×10^{-10}	Bone surface	1.0×10^{-9}	4×10^{7}
	Y	0.01	8.6×10^{-10}	Lungs	7.2×10^{-9}	1×10^{7}
50.5 d ^{89}Sr	D	0.3	1.6×10^{-9}	Bone surface	8.4×10^{-9}	1×10^{7}*
	Y	0.01	1.0×10^{-8}	Lungs	8.4×10^{-8}	2×10^{6}
29.1 y ^{90}Sr	D	0.3	6.2×10^{-8}	Bone surface	7.3×10^{-7}	4×10^{5}*
	Y	0.01	3.4×10^{-7}	Lungs	2.9×10^{-6}	5×10^{4}
64.0 d ^{95}Zr	D	0.002	5.2×10^{-9}	Bone surface	1.0×10^{-7}	5×10^{6}*
	W	0.002	3.5×10^{-9}	Bone surface	2.2×10^{-8}	5×10^{6}
	Y	0.002	4.9×10^{-9}	Lungs	4.1×10^{-8}	3×10^{6}
35.2 d ^{95}Nb	W	0.01	1.0×10^{-9}	Lungs	5.5×10^{-9}	2×10^{7}
	Y	0.01	1.2×10^{-9}	Lungs	8.3×10^{-9}	1×10^{7}
66.0 h ^{99}Mo	D	0.8	5.2×10^{-10}	Liver	1.9×10^{-9}	5×10^{7}
	W	0.05	9.9×10^{-10}	LLI wall	5.5×10^{-9}	2×10^{7}
6.0 h ^{99m}Tc	D	0.8	8.7×10^{-12}	Thyroid	5.0×10^{-11}	2×10^{9}
	W	0.8	5.7×10^{-12}	Lungs	3.1×10^{-11}	2×10^{9}

(*contd ...*)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
39.3 d ^{103}Ru	D	0.05	8.0×10^{-10}	LLI wall	1.7×10^{-9}	2×10^{7}
	W	0.05	1.4×10^{-9}	Lungs	9.9×10^{-9}	8×10^{6}
	Y	0.05	2.1×10^{-9}	Lungs	1.6×10^{-8}	1×10^{6}
368.2 d ^{106}Ru	D	0.05	1.5×10^{-8}	LLI wall	2.5×10^{-8}	1×10^{6}
	W	0.05	2.5×10^{-8}	Lungs	2.1×10^{-7}	6×10^{5}
	Y	0.05	1.2×10^{-7}	Lungs	1.0×10^{-6}	2×10^{5}
249.9 d ^{110m}Ag	D	0.05	1.0×10^{-8}	Liver	8.1×10^{-8}	2×10^{6}
	W	0.05	7.1×10^{-9}	Lungs	3.2×10^{-8}	3×10^{6}
	Y	0.05	1.4×10^{-8}	Lungs	1.2×10^{-7}	1×10^{6}
2.8 d ^{111}In	D	0.02	2.1×10^{-10}	Kidneys	4.6×10^{-10}	1×10^{8}
	W	0.02	2.0×10^{-10}	LLI wall	7.1×10^{-10}	9×10^{7}
1.7 h ^{113m}In	D	0.02	9.2×10^{-12}	Lungs	5.0×10^{-11}	2×10^{9}
	W	0.02	7.0×10^{-12}	Lungs	5.8×10^{-11}	2×10^{9}
115.1 d ^{113}Sn	D	0.02	1.1×10^{-9}	Bone surface	5.0×10^{-9}	2×10^{7}
	W	0.02	2.4×10^{-9}	Lungs	1.8×10^{-8}	7×10^{6}
60.2 d ^{124}Sb	D	0.1	1.5×10^{-9}	LLI wall	4.0×10^{-9}	1×10^{7}
	W	0.01	5.7×10^{-9}	Lungs	4.1×10^{-8}	3×10^{6}

(*contd ...*)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
2.8 y ^{125}Sb	D	0.1	5.7×10^{-10}	Bone surface	2.7×10^{-9}	4×10^{7}
	W	0.01	2.6×10^{-9}	Lungs	2.2×10^{-8}	6×10^{6}
13.2 h ^{123}I	D	1.0	7.5×10^{-11}	Thyroid	2.2×10^{-9}	2×10^{8}*
60.1 d ^{125}I	D	1.0	6.5×10^{-9}	Thyroid	2.2×10^{-7}	2×10^{6}*
1.6×10^{7} y ^{129}I	D	1.0	4.7×10^{-8}	Thyroid	1.6×10^{-6}	3×10^{5}*
8.0 d ^{131}I	D	1.0	8.8×10^{-9}	Thyroid	2.9×10^{-7}	1×10^{6}*
2.1 y ^{134}Cs	D	1.0	1.3×10^{-8}	Soft tissue	1.5×10^{-8}	2×10^{6}
30.0 y ^{137}Cs	D	1.0	8.7×10^{-9}	Soft tissue	9.5×10^{-9}	2×10^{5}
12.7 d ^{140}Ba	D	0.1	9.7×10^{-10}	LLI wall	4.4×10^{-9}	2×10^{7}
284.3 d ^{144}Ce	W	3×10^{-4}	5.3×10^{-8}	Liver	2.5×10^{-7}	5×10^{5}
	Y	3×10^{-4}	9.5×10^{-8}	Lungs	7.9×10^{-7}	2×10^{5}
13.6 d ^{143}Pr	W	3×10^{-4}	1.7×10^{-9}	Lungs	1.1×10^{-8}	9×10^{6}
	Y	3×10^{-4}	2.0×10^{-9}	Lungs	1.3×10^{-8}	8×10^{6}

(*contd ...*)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
2.6 y ^{147}Pm	W	$3x10^{-4}$	$6.9x10^{-9}$	Bone surface	$1.0x10^{-7}$	$4x10^{6}$*
	Y	$3x10^{-4}$	$9.3x10^{-9}$	Lungs	$7.7x10^{-8}$	$2x10^{6}$
32.0 d ^{169}Yb	W	$3x10^{-4}$	$1.6x10^{-9}$	Lungs	$9.3x10^{-9}$	$1x10^{7}$
	Y	$3x10^{-4}$	$1.9x10^{-9}$	Lungs	$1.4x10^{-8}$	$9x10^{6}$
74.0 d ^{192}Ir	D	0.01	$4.9x10^{-9}$	Kidneys, liver	$1.7x10^{-8}$	$6x10^{6}$
	W	0.01	$3.5x10^{-9}$	Lungs	$2.5x10^{-8}$	$4x10^{6}$
	Y	0.01	$6.3x10^{-9}$	Lungs	$5.2x10^{-8}$	$3x10^{6}$
2.7 d ^{198}Au	D	0.1	$8.9x10^{-10}$	Bladder wall	$1.1x10^{-8}$	$5x10^{7}$
	W	0.1	$8.8x10^{-10}$	LLI wall	$3.7x10^{-9}$	$2x10^{7}$
	Y	0.1	$8.6x10^{-10}$	LLI wall	$4.3x10^{-9}$	$2x10^{7}$
64.1 h ^{197}Hg						
Organic compounds	D	1.0	$1.0x10^{-10}$	Kidneys	$5.6x10^{-10}$	$3x10^{8}$
Inorganic compounds	D	0.02	$1.2x10^{-10}$	Kidneys	$4.1x10^{-10}$	$2x10^{8}$
	W	0.02	$1.6x10^{-10}$	LLI wall	$8.3x10^{-10}$	$1x10^{8}$
	V	1.0	$1.5x10^{-10}$	Lung	$1.1x10^{-9}$	$1x10^{8}$

(*contd* ...)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
46.6 d ^{203}Hg						
Organic compounds	D	1.0	1.7×10^{-9}	Kidneys	1.2×10^{-8}	2×10^{7}
Inorganic compounds	D	0.02	1.1×10^{-9}	Kidneys	6.8×10^{-9}	2×10^{7}
	W	0.02	1.3×10^{-9}	Lungs	8.8×10^{-9}	1×10^{7}
	V	1.0	1.7×10^{-9}	Kidneys	9.6×10^{-9}	1×10^{7}
3.0 d ^{201}Tl	D	1.0	6.1×10^{-11}	Lungs	1.7×10^{-10}	4×10^{8}
138.4 d ^{210}Po	D	0.1	2.2×10^{-6}	Spleen	2.2×10^{-5}	2×10^{4}
	W	0.1	2.1×10^{-6}	Lungs	1.3×10^{-5}	2×10^{4}
1600 y ^{226}Ra	W	0.2	2.1×10^{-6}	Lungs	1.6×10^{-5}	9000
5.8 y ^{228}Ra	W	0.2	1.2×10^{-6}	Lungs	7.2×10^{-6}	2×10^{4}
1.9 y ^{228}Th	W	2×10^{-4}	6.5×10^{-5}	Bone surface	1.4×10^{-3}	500*
	Y	2×10^{-4}	8.3×10^{-5}	Lungs	6.9×10^{-4}	200
1.4×10^{10} y ^{232}Th	W	2×10^{-4}	4.4×10^{-4}	Bone surface	1.1×10^{-2}	90*
	Y	2×10^{-4}	3.1×10^{-4}	Bone surface	5.0×10^{-3}	90*

(*contd* ...)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
1.6×10^5 y ^{233}U	D	0.05	7.4×10^{-7}	Bone surface	1.1×10^{-5}	8×10^4*
	W	0.05	1.9×10^{-6}	Lungs	1.6×10^{-5}	1×10^4
	Y	0.002	3.6×10^{-5}	Lungs	3.0×10^{-4}	500
2.5×10^5 y ^{234}U	D	0.05	7.2×10^{-7}	Bone surface	1.1×10^{-5}	8×10^4*
	W	0.05	1.9×10^{-6}	Lungs	1.6×10^{-5}	1×10^4
	Y	0.002	3.6×10^{-5}	Lungs	3.0×10^{-4}	600
7.0×10^8 y ^{235}U	D	0.05	6.6×10^{-7}	Bone surface	1.0×10^{-5}	8×10^4*
	W	0.05	1.8×10^{-6}	Lungs	1.5×10^{-5}	1×10^4
	Y	0.002	3.3×10^{-5}	Lungs	2.8×10^{-4}	600
4.5×10^9 y ^{238}U	D	0.05	6.4×10^{-7}	Bone surface	9.8×10^{-6}	9×10^4*
	W	0.05	1.7×10^{-6}	Lungs	1.4×10^{-5}	1×10^4
	Y	0.002	3.2×10^{-5}	Lungs	2.7×10^{-4}	600
2.1×10^6 y ^{237}Np	W	0.001	1.3×10^{-4}	Bone surface	3.3×10^{-3}	300*
2.4 d ^{239}Np	W	0.001	6.0×10^{-10}	LLI wall	2.9×10^{-9}	3×10^7

(*contd ...*)

Radio-nuclide	Class	f_1	$E(50)$ (Sv/Bq)	Organ/ tissue T	$H_T(50)$ (Sv/Bq)	ALI (Bq)
87.7 d ^{238}Pu	W	0.001	1.0×10^{-4}	Bone surface	1.9×10^{-3}	300*
	Y	1×10^{-5}	7.5×10^{-5}	Bone surface	7.2×10^{-4}	300
2.4×10^{4} y ^{239}Pu	W	0.001	1.1×10^{-4}	Bone surface	2.1×10^{-3}	300*
	Y	1×10^{-5}	8.1×10^{-5}	Bone surface	8.2×10^{-4}	300*
6.5×10^{3} y ^{240}Pu	W	0.001	1.1×10^{-4}	Bone surface	2.1×10^{-3}	300*
	Y	1×10^{-5}	8.1×10^{-5}	Bone surface	8.2×10^{-4}	300*
14.4 y ^{241}Pu	W	0.001	2.3×10^{-6}	Bone surface	4.2×10^{-5}	2×10^{4}
	Y	1×10^{-5}	1.3×10^{-6}	Bone surface	1.8×10^{-5}	2×10^{4}
432.2 y ^{241}Am	W	0.001	1.2×10^{-4}	Bone surface	2.2×10^{-3}	300*
162.8 d ^{242}Cm	W	0.001	4.6×10^{-6}	Bone surface	4.9×10^{-5}	6000*
18.1 y ^{244}Cm	W	0.001	6.4×10^{-5}	Bone surface	1.2×10^{-3}	500*
2.6 y ^{252}Cf	W	0.001	3.6×10^{-5}	Bone surface	6.9×10^{-4}	900*
	Y	0.001	4.0×10^{-5}	Lungs	3.0×10^{-4}	500

* ALI determined by deterministic dose.
Class, f_1, and ALI values from ICRP 61; $E(50)$ and $H_T(50)$ values from ICRP 30.

Table 25.4: Analysis of ALI values as given in ICRP 61
(No. of tabulations in each category)

ALI range (Bq)	Inhalation class				Ingestion
	V	D	W	Y	
$1\text{-}9 \times 10^{1}$	-	1	5	3	-
$1\text{-}9 \times 10^{2}$	-	2	24	20	-
$1\text{-}9 \times 10^{3}$	-	-	15	4	3
$1\text{-}9 \times 10^{4}$	-	17	17	7	26
$1\text{-}9 \times 10^{5}$	1	15	32	19	43
$1\text{-}9 \times 10^{6}$	-	42	89	50	108
$1\text{-}9 \times 10^{7}$	8	118	163	71	329
$1\text{-}9 \times 10^{8}$	7	162	134	60	296
$1\text{-}9 \times 10^{9}$	5	85	112	47	44
$1\text{-}9 \times 10^{10}$	2	3	8	3	6
Total	23	445	599	284	855

Table 25.5: Metabolic data* for selected elements

[*Author's Note* : The information presented here has been collated from ICRP 30, ICRP 54 and ICRP 56.]

Uniform distribution of the radionuclide in any organ or tissue is assumed. For brevity, when tissue is mentioned, it means organ or tissue. Values of f_1, the fraction of a stable element reaching body fluids after its entry into the GI tract (from ingestion or inhalation) have been given in Tables 25.2 and 25.3. The fractions of the inhaled or ingested activity reaching any tissue and the retention half-time in that tissue are indicated in brackets. Values given here are those used by ICRP in its dosimetric models; for more detailed information the data given in ICRP 30 may be consulted.

Methods of internal monitoring for certain important radionuclides are indicated. Mention is made at appropriate places wherever the methods are not sensitive enough for monitoring at levels below or around the ALI.

Tritium

Tritium may be released into the environment as tritium gas, tritiated water, and organically bound tritium (OBT). In the environment tritium gas is converted into tritiated water.

Tritiated water, taken into the body by inhalation, ingestion or absorption through the skin is assumed to be completely and rapidly mixed with total body water. The rate of turnover of body water varies among individuals and depends upon factors such as fluid intake rate and ambient temperature. Retention can be adequately described by a single exponential with a half-life of 10 days (range 4-18 days). An increase of fluid intake reduces the biological half-life by a factor of 2-3 (NCRP, 1980).

About 1-5% of the tritium in tritiated water in the body becomes incorporated into non-volatile organic compounds (Takeda and Kasida, 1979) with an elimination half-time of 40 days in the adult, corresponding to that of carbon. OBT contributes about 10% to the total dose.

A major part of tritium intake by members of the public will occur by ingestion of food into which tritium has been incorporated into both plant and animal components, and can be taken as completely absorbed (f_1 = 1.0). It is assumed that of the OBT that enters the blood, 50% behaves similarly to tritiated water while the remaining 50% enters into bonds with carbon and follows the general metabolic behaviour of carbon.

When tritium labelled organic compounds are ingested, a considerable fraction may be broken down in the GI tract and also catabolized after they have crossed the gut to produce tritiated water. Many organic compounds of tritium are not very volatile and the probability of their being inhaled as vapours is very small.

Except for tritiated thymidine which is taken by the nuclei of cells synthesizing DNA, tritium compounds are usually distributed throughout soft tissues. ALI values for organic compounds might differ considerably from those for tritiated water, and the value for tritiated thymidine might be as much as 50 times lower.

The biological retention half-times of both the tritiated water and the OBT get reduced with decreasing age; the values are 3 days and 8 days respectively for the 3-month old. DAC for exposure to elemental tritium in air is limited by the dose to lung and is 4 orders of magnitude higher than for tritiated water. Internal monitoring of ^{3}H is by liquid scintillation counting of urine samples.

Carbon

The fractional absorption of dietary carbon from GI tract to blood is very high, while for non-dietary forms it is very variable. However, some organic compounds in food may be less completely absorbed.

For the general public, ^{14}C in foodstuffs is the most important source of entry into the body. Complete absorption into body fluids is assumed at all ages. Nucleic acids represent a relatively small but radiosensitive component into which ^{14}C may be incorporated after intake of labelled precursors. Higher biosynthetic rates of nucleic acids are associated with more rapid cell division (embryonic and neonatal development; puberty; specific tissues in adults). Most organic compounds are not very volatile. Metabolism of some ^{14}C labelled compounds is age-dependent. Dietary carbon has a retention half-time of 40 days in the adult. It decreases to 8 days in the 3-month old.

Carbon monoxide, when inhaled, has a relative solubility in body tissues. The main doses are from CO bound to haemoglobin, or, to a lesser extent, to other iron-haem compounds such as cytochrome oxidase. When CO is inhaled, 0.4 is instantaneously bound to haemoglobin and uniformly distributed in all tissues (200 min). The degree of exercise may profoundly modify the CO level in blood.

The solubility of carbon dioxide in water is 24 times greater than that of oxygen. All carbon dioxide entering the respiratory system is translocated to blood where it exists mainly as the bicarbonate. It is distributed in all tissues (0.18 - 5 d; 0.81 - 60 min; 0.01 - 40 d).

Sodium

Inhalation class: D

The biological half-life of sodium is strongly influenced by the level of sodium in the diet, decreasing from 335 days for a daily intake of 0.25 g to 5 d for an intake of 40 g. Of the sodium leaving the transfer compartment, 0.3 is translocated to bone (0.29 - 10 d; 0.01 - 500 d), and 0.7 to other tissues (10 d).

Isotopes of sodium in bone are distributed throughout mineral bone, bone marrow, skeletal cartilage and periarticular tissues.

Phosphorus

Inhalation class: D - all compounds except some phosphates; W: some phosphates.

Phosphorus entering the transfer compartment is retained there with a half-life of 0.5 d. Of this phosphorus, 0.15 is excreted directly, 0.15 goes to extracellular fluids (2 d), 0.40 to soft tissue (19 d), 0.20 to bone (permanently retained). 90% is excreted via urine.

Isotopes like ^{32}P with a half-life of less than 15 d are taken to be retained on bone surfaces. Internal monitoring for ^{32}P is by beta counting of urine.

Sulphur

Inhalation class: D or W - sulphates, sulphides; W - elemental sulphur.

Inhaled sulphur is instantaneously transferred to the transfer compartment; from then on, its metabolism is the same as that of sulphur entering the transfer compartment following ingestion or inhalation of any other inorganic compound.

Of sulphur entering the transfer compartment 0.20 is distributed in all tissues (0.15 - 20 d; 0.05 - 2000 d), and 0.80 is excreted. Some organic compounds of sulphur incorporated in aminoacids become incorporated into various metabolites and retained for a long time.

Chromium

Inhalation class: D - all except the following; W - halides, nitrates; Y - oxides, hydroxides.

The retention of chromium in the body is dependent upon its chemical form. Of the chromium entering the transfer compartment (retention half-life 0.5 d), 0.03 is excreted directly, 0.05 goes to bone (1000 d), 0.65 is distributed in other tissues (0.40 - 6 d; 0.25 - 80 d). Sodium chromate has a marked affinity for erythrocytes. Internal monitoring for ^{51}Cr is by whole body counting.

Manganese

Inhalation class: D - all except the following; W - oxides, hydroxides, halides, nitrates.

Of manganese entering the transfer compartment, 0.35 goes to bone (40 d) where it is distributed over the bone surfaces, 0.25 to liver (0.10 - 4 d; 0.15 - 40 d), 0.40 is distributed in other tissues (0.20 - 4 d; 0.20 - 40 d). Internal monitoring for ^{54}Mn is by whole body counting.

Iron

Inhalation class: D - all common compounds except the following: W - oxides, hydroxides, halides.

Absorption of iron from the GI tract depends upon a number of factors, viz. the amount of iron in the diet and its chemical form, the body's need for that iron, and the presence or absence of interfering substances. Absorption of ferrous salts is considered to be somewhat greater than that of ferric salts.

In adult man 70% of total iron is bound in haemoglobin and most of the rest is associated with the iron storage compounds ferritin and hemosiderin in the reticuloendothelial system. Of the iron leaving the transfer compartment, 0.08 goes to liver, 0.01 to spleen, and the rest to the remaining tissues (2000 d). Internal monitoring for ^{59}Fe is by whole body counting.

Cobalt

Inhalation class: W - compounds other than the following; Y - oxides, hydroxides, halides, nitrates.

Of cobalt entering the transfer compartment, 0.5 is directly excreted, 0.05 goes to liver, 0.45 to other tissues. Retention half-times in all tissues: 0.6 - 6 d; 0.2 - 60 d; 0.2 - 800 d; 70% of the excretion is via urine. Internal monitoring for all the cobalt isotopes discussed here is either by whole body counting or gamma spectrometry of urine samples.

Zinc

Uptake to blood depends on fasting state and dietary zinc levels.

Inhalation class: D - sulphates; W - halides, phosphates, sulphides; Y - oxides, hydroxides.

Of the zinc leaving the transfer compartment 0.2 goes to bone (400 d) and rest to other tissues (0.25 - 20 d; 0.55 - 400 d). Because of its long half-life, ^{65}Zn is assumed to be distributed throughout the volume of mineral bone.

Gallium

Inhalation class: D - all except the following; Y - oxides, hydroxides, carbides, halides, nitrates.

Of the gallium leaving the transfer compartment, 0.3 goes to bone, 0.09 to liver, 0.01 to spleen and 0.6 to other tissues. Elimination from tissue: 0.3-1 d; 0.7 - 50 d. ^{67}Ga is distributed on bone surfaces.

Selenium

Inhalation class: D - all except the following; W - oxides, hydroxides, carbides.

Of the selenium leaving the transfer compartment, 0.15 goes to liver, 0.05 to kidneys, 0.01 to spleen, 0.005 to pancreas, and 0.685 to other tissues. Elimination from tissues: 0.1 - 3 d; 0.4 - 30 d; 0.5 - 150 d.

Strontium

Inhalation class: D - all except $SrTiO_3$; Y - $SrTiO_3$.

Studies of fallout ^{90}Sr have provided a large quantity of data on the age-specific behaviour of strontium. Fasting, low dietary calcium levels, magnesium, phosphorus, milk diets and vitamins may increase gastro-intestinal absorption of strontium. The f_1 values increase from 0.3 for the adult to 0.60 for the 3-month old. A comprehensive model for the retention of strontium in adults has been developed in ICRP 20. The ratio of urinary to fecal excretion is 4.

Because of their long half-lives (> 15 d) all the isotopes of strontium considered here are taken to be distributed throughout the volume of mineral bone. The fraction of the amount of strontium absorbed in blood which deposits on bone (mainly cortical) surfaces increases from 0.15 for the adult to 0.61 for the 3-month old. Removal rate from cortical bone surface is 10 times faster for the 3-month old than for adults.

Internal monitoring for ^{85}Sr is by whole body counting or gamma ray spectrometry of urine samples. ^{89}Sr and ^{90}Sr are monitored by beta counting of urine after chemical separation. For routine monitoring of class Y compounds of ^{90}Sr, the above method of detection is not adequate enough.

Zirconium

Inhalation class: D - all except the following; W - oxides, hydroxides, halides, nitrates; Y - carbide.

Of the zirconium entering the transfer compartment, 0.5 is translocated to bone (8000 d) and 0.5 to other tissues (7 d). Zirconium is not a constituent of mineral bone; it is assumed to be distributed on bone surfaces. All the biokinetic parameters are assumed to be age-independent (except that for the 3-month old the f_1 value is twice that at any other age).

Detection is by whole body counting. Gamma spectrometry of urine samples with sodium iodide detectors will not resolve the photopeaks of ^{95}Zr and its daughter ^{95}Nb; interpretation requires a knowledge of the time of intake and assumptions about the behaviour of ^{95}Nb produced in the body.

Niobium

Inhalation class: W - all except the following; Y - oxides, hydroxides.

Of the niobium entering the transfer compartment, 0.71, 0.018, 0.01, 0.002 and 0.26 are assumed to go to mineral bone, spleen, testes, and other tissues respectively. Retention half-times of niobium entering any one of the

organs are: 0.5 - 6 d; 0.5 - 200 d. ^{95}Nb is assumed to be distributed over bone surfaces.

The f_1 value for the child is taken to be twice that for all other ages. Deposition in skeleton increases from 40% in adults to 60% in the 3-month old. Retention half-times are taken to be age-independent.

Molybdenum

Inhalation class: D - all except the following; Y - oxides, hydroxides, MoS_2.

Of molybdenum entering the transfer compartment, fractions 0.3, 0.15, 0.05 and 0.5 are deposited in liver, mineral bone, kidneys, and other tissues respectively. For all tissues retention half-times are: 0.1 - 1 d; 0.9 - 500 d. ^{99}Mo is taken to be distributed over bone surface.

Technetium

In the case of uptake to blood, the f_1 value of 0.8 is unduly large for certain radiopharmaceuticals, e.g. ^{99m}Tc-S-colloid.

Inhalation class: D - all except the following; W - oxides, hydroxides, halides, nitrates.

In man, technetium administered intravenously as pertechnetate is concentrated in the thyroid, GI tract and liver. Of the technetium leaving the transfer compartment (0.02 d), 0.04 is translocated to thyroid (McAfee *et al.*, 1964). Fractions 0.1 and 0.03 go to stomach wall and liver respectively, the remaining to other tissues. Of the technetium translocated to organs other than the thyroid, retention is as follows: 0.75 - 1.6 d; 0.20 - 3.7 d; 0.05 - 22 d.

The above model is inappropriate for ^{99m}Tc labelled radiopharmaceuticals other than pertechnetate. For details, reference may be made to ICRP 53 (1987).

Ruthenium

Inhalation class: D - all except the following; W - halides; Y- oxides, hydroxides.

Of the ruthenium entering the transfer compartment (0.3 d), 0.15 is excreted directly, 0.7 is distributed in all tissues (0.35 - 8 d; 0.30 - 35 d; 0.20 - 1000 d). 80% is excreted via urine. The f_1 value for the child is taken to be twice that for all other ages. Retention half-times are taken to be age-independent. Internal monitoring for ^{106}Ru is by whole body counting or by gamma spectrometry of urine sample.

Silver

Inhalation class: D - all except the following; W - nitrates, sulphides; Y - oxides, hydroxides.

Of the silver leaving the transfer compartment, 0.8 goes to liver and 0.2 to other tissues. Retention pattern in all tissues: 0.1 - 3.5 d; 0.9 - 50 d. Internal monitoring for ^{110m}Ag is by whole body counting.

Indium

Inhalation class: D - all except the following; Y - oxides, hydroxides, halides, nitrates.

Of the indium leaving the transfer compartment , fractions 0.3, 0.03, 0.07, 0.01 and 0.42 go respectively to red bone marrow, liver, kidneys, spleen, and other tissues. Indium is assumed to be retained indefinitely in all tissues.

Tin

Inhalation class: D - all except the following; Y - sulphides, oxides, hydroxides, halides, nitrates, stannic phosphate.

Of the tin leaving the transfer compartment, 0.5 is directly excreted, 0.35 goes to mineral bone and 0.15 to other tissues. For all tissues retention pattern is: 0.2 - 4 d; 0.2 - 25 d; 0.6 - 400 d. ^{113}Sn is distributed in mineral bone.

Antimony

Inhalation class: D - all except the following; W - oxides, hydroxides, halides, sulphides, sulphates, nitrates.

Of the antimony leaving the transfer compartment, 0.2 is directly excreted (0.25 d), 0.2 goes to mineral bone, 0.1 to liver and 0.5 to rest of the tissues. Retention pattern in all tissues: 0.95 - 5 d; 0.05 - 100 d. Antimony is uniformly distributed on bone surfaces. ^{124}Sb and ^{125}Sb can be monitored by whole body counting.

Iodine

Inhalation class: D

Of the iodine entering the transfer compartment, 0.7 is directly excreted and 0.3 translocated to thyroid, from where it is lost in the form of organic iodine (80 d). Organic iodine is uniformly distributed among all tissues (12 d). 0.1 of the organic iodine goes directly to fecal excretion and 0.9 is returned to the transfer compartment as inorganic iodine. The effective half-life of iodine in thyroid is 90 to 120 d.

In countries with a very low intake of stable iodine the accumulation of radioiodine by the thyroid is increased. The low levels of dietary iodine will, however, have resulted in a compensatory increase in the mass of the thyroid, with the result that the concentration of radioiodine in the thyroid will be similar to that using the standard model (Henrichs *et al.*, 1983).

Uptake of radioiodine by the thyroid is enhanced for the first few weeks after birth, but thereafter changes very little with increasing age. However, the turnover rate of iodine decreases from a biological half-life of 80 days in adults to 11 days in the 3-month old. The 'apparent half-times' in thyroid (obtained from thyroid monitoring) will be different from the above values as a result of recycling. They are reduced from about 90 days for the adult to 15 days for the 3-month old.

Internal monitoring for ^{125}I, ^{129}I and ^{131}I can easily be done by *in vivo* thyroid counting. In addition, urine sample analysis can be done by liquid scintillation counting for ^{125}I, beta counting for ^{129}I and by gamma spectrometry for ^{131}I.

Cesium

Inhalation class: D

Complete GI absorption can be assumed at all ages. Cesium is uniformly distributed throughout the body (with a somewhat greater concentration in muscle). Of the cesium entering the transfer compartment, 0.1 goes to one tissue compartment (2 d) and 0.9 to a second tissue compartment (110 d); 80% of excreted cesium is in the urine. In the 3-month old, retention can be taken to be represented by a single exponential with a retention half-time of 16 days.

Internal monitoring for both ^{134}Cs and ^{137}Cs can be done by whole body counting or by gamma spectrometry of urine samples.

Barium

Inhalation class: D

For metabolic behaviour, reference may be made to ICRP 20. ^{140}Ba is taken to be distributed on bone surfaces.

Cerium

Inhalation class: W - all except the following; Y - oxides, hydroxides, fluorides.

Of the cerium entering the transfer compartment, 0.6 is translocated to liver, 0.2 to bone and 0.15 to other tissues. Retention half-time in all tissues is 3500 d. Excretion is 90% via feces and 10% via urine. The chemistry of cerium resembles that of actinides more closely than that of alkaline earths; cerium is taken to be uniformly distributed on bone surfaces.

GI absorption in the 3-month old is taken to be ten times that in any other age. The skeletal uptake increases to 70% in the 3-month old, while the liver uptake decreases to 10%. Internal monitoring for ^{144}Ce is mainly by whole body counting, but the method is not sensitive enough. Urine analysis is not recommended.

Praseodymium

Inhalation class: W - all except the following: Y - oxides, hydroxides, carbides, fluorides.

Of the praseodymium leaving the transfer compartment, 0.6 goes to liver (10 d), 0.25 to bone (3500 d), 0.05 to kidneys, and 0.1 directly excreted. ^{143}Pr is uniformly distributed over bone surface.

Promethium

Inhalation class: W - all except the following: Y - oxides, hydroxides, carbides, fluorides.

Of the promethium leaving the transfer compartment, 0.1 is directly excreted, 0.45 goes to liver (3500 d), and 0.45 to bone (3500 d). ^{147}Pm is uniformly distributed over bone surfaces.

Ytterbium

Inhalation class: W - all except the following: Y - oxides, hydroxides, fluorides.

Of the ytterbium leaving the transfer compartment, fraction 0.5 is translocated to mineral bone (3500 d), 0.03 to liver (3500 d), 0.02 to kidneys (10 d), 0.005 to spleen (3500 d), the rest being directly excreted. The metabolism of chelates differs considerably from that discussed above. ^{169}Yb is taken to be distributed over bone surfaces.

Gold

Inhalation class: D - all except the following: W - halides, nitrates; Y - oxides, hydroxides.

Gold entering the transfer compartment is instantaneously uniformly distributed in all organs and tissues (3 d). Concentration of radioactive isotopes of gold in urine contained in bladder is 10 times that in any other tissue.

Mercury

(a) Vapour Form

Of the vapour entering the lungs, 70% is deposited there and subsequently transferred to blood with a biological half-life of 1-7 d. Its subsequent metabolism is identical to that of inorganic compounds.

(b) Inorganic Compounds

Inhalation class: D - sulphates; W - oxides, hydroxides, halides, nitrates, sulphides.

After intake as an inorganic compound or as the metal, mercury leaving the transfer compartment is distributed as follows: 0.08 goes to kidneys (concentration 15 times that in the rest of body) and 0.92 to other tissues. Retention half-times in all tissues: 0.95 - 40 d; 0.05 - 10,000 d.

(c) Organic Compounds

Inhalation class: D

Of the mercury leaving the transfer compartment, 0.08 goes to kidneys, 0.2 to brain and 0.72 to the rest of the tissues. Retention pattern for all tissues: 0.95 - 80 d; 0.05 - 10,000 d. Neohydrin is more rapidly eliminated than other organic compounds. Internal monitoring for ^{203}Hg is by whole body counting.

Thallium

Inhalation class: D

Thallium entering the transfer compartment is instantaneously translocated to various organs and tissues. Of this thallium, 0.03 goes to kidneys and 0.97 to other tissues (10 d).

Polonium

Inhalation class: D - all except the following: W - oxides, hydroxides, nitrates.

Of the polonium entering the transfer compartment, fractions 0.1, 0.1, 0.1 and 0.7 go respectively to liver, kidney, spleen and other tissues. For all tissues retention half-time is 50 d.

Radium

Inhalation class: W

ICRP 20 has described a comprehensive model. Briefly, fractions 0.54, 0.29 0.11, 0.04, 0.04 and 0.02 of ingested radium are retained with half-times of 0.4 d, 5 d, 60 d, 700 d and 5000 d respectively; 95% of the excretion is via feces and 5% via urine.

^{222}Rn produced from ^{226}Ra in soft tissue escapes from the body without decaying. The fraction of ^{222}Rn retained in mineral bone is a function of the time for which its parent ^{226}Ra has been resident in that tissue, varying from 6% at 1 d to 40% in 27 years (Mays *et al.*, 1958). The average fraction of ^{222}Rn retained in mineral bone, evaluated over the 50 years following intake, is about 0.3 (Norris *et al.*, 1955). Other, shorter-lived, isotopes of radon are assumed to remain totally with their parents.

Isotopes of radium with half-lives greater than 15 days (^{226}Ra, ^{228}Ra) are assumed to be distributed throughout the volume of mineral bone, while ^{223}Ra, ^{224}Ra, ^{225}Ra and ^{227}Ra are assumed to be distributed on bone surfaces.

Internal monitoring for ^{226}Ra and ^{228}Ra is by whole body counting, or by radiochemical separation and alpha spectrometry of urine samples. The latter is more sensitive. Sensitivity of whole body counting is not adequate for routine monitoring unless high efficiency, low background monitors are used.

Thorium

Inhalation class: W - all except the following: Y - oxides, hydroxides.

The biological half-life of the transfer compartment is 0.5 d. Of the thorium entering the transfer compartment fractions 0.7 (8000 d), 0.04 (700 d), 0.16 (700 d) go respectively to bone, liver and other tissues; 0.1 is directly excreted completely via urine. Thorium is predominantly deposited upon endosteal surfaces.

Internal monitoring for both ^{228}Th and ^{232}Th is by radiochemical separation and alpha spectrometry of urine samples. The technique does not have adequate sensitivity to detect intakes around the Investigation Level (IL). Personal air samplers should be used to determine intakes.

Uranium

Inhalation class: D - UF_6, UO_2F_2, $UO_2(NO_3)_2$; W - UO_3, UF_4, UCl_4; Y - UO_2, U_3O_8.

Industrial UO_3, and in some cases UF_4, may behave more like a class D material, while UO_2 aerosols from fuel elements may be nearer to Y class. Of the uranium entering the transfer compartment, 0.54 is directly excreted, while fractions 0.2 (20 d) and 0.023 (5000 d) go to mineral bone, 0.12 to kidneys (6 d), and 0.12 goes to other tissues (6 d). All excretion is via urine.

Shorter-lived isotopes like ^{237}U, ^{239}U and ^{240}U are taken to be distributed over bone surfaces, while the longer-lived ones like ^{233}U, ^{234}U, ^{235}U, and ^{238}U are assumed to be uniformly distributed throughout the volume of mineral bone.

Chemical toxicity: Intakes of the more transportable uranium compounds are limited by considerations of chemical toxicity rather than radiation dose. For continuous occupational exposures, the American Conference of Government Industrial Hygienists (ACGIH, 1983) recommends a threshold value of concentration of 0.2 mg m^{-3}. For a standard breathing rate of 1.2 m^3 h^{-1} this corresponds to an average daily intake of 2 mg. For natural uranium of classes D and W, the limits for chemical toxicity are more restrictive, while for class Y compounds of natural uranium and all classes of highly enriched uranium, radiological considerations are more restrictive.

Internal monitoring for ^{234}U, ^{235}U and ^{238}U can be done by alpha spectrometry (or mass spectrometry) of urine; total uranium can be chemically monitored by fluorimetry of urine samples. These may be supplemented by thorax counting using dual phosphor detectors, or, better, an array of germanium detectors, and by use of personal air sampling.

Many operations involve a diversity of chemical forms of uranium and a range of enrichments. Monitoring programmes must be designed for the materials handled.

Neptunium

Inhalation class: W

Of the neptunium leaving the transfer compartment, 0.75 translocates to bone (50 y), and 0.15 to liver (20 y); the remainder of the neptunium leaving the transfer compartment is cleared with a half-life of 0.25 days. 0.035% goes to testes and 0.011% to ovaries, where it is permanently retained. Like the actinides, neptunium is assumed to be uniformly distributed over the endosteal bone surfaces.

The fractional GI absorption values are the same as for plutonium at any given age. Relative deposition in skeleton is greater and in liver smaller in younger age groups.

Plutonium

ICRP 19 (1972), ICRP 48 (1986) and ICRP 56 (1989) have reviewed in detail the metabolism of plutonium and other transuranics. Data on the absorption of ingested plutonium do not permit determination of f_1 at better than an order of magnitude unless the chemical and physical state of the ingested material can be established. A 3-level classification is adopted for the f_1 values: oxides - 10^{-5}; nitrates - 10^{-4}; others - 10^{-3}.

Inhalation class: W - all except oxides; Y - oxides.

Of the plutonium entering the transfer compartment, 0.45 is translocated to bone (50 y), and 0.45 to liver (20 y); 0.035% goes to testes and 0.011% to ovaries where it is retained permanently. Plutonium is uniformly distributed over bone surfaces (primarily endosteal surfaces). Plutonium is eliminated via feces and urine, the relative ratio depending on time after intake.

The rate of elimination of the absorbed fraction can be increased by intravenous administration of DTPA (urinary excretion can be increased 50 times). Value of GI absorption for the first year of life is taken to be 10 times that for any other age. Relative deposition of plutonium in the skeleton is greater and in liver smaller in younger age groups.

Internal monitoring is by *in vivo* lung counting or alpha spectrometry of urine or fecal samples. Monitoring methods for plutonium and other transuranics are not sensitive enough. Routine monitoring should include additionally personal air sampling.

Americium

Inhalation class: W

The distribution, retention and excretion functions are the same as for plutonium. Chelated forms are more biologically mobile than other chemical forms. Americium is taken to be uniformly distributed on bone surfaces.

Values for GI tract absorption at any age are the same as for plutonium. Relative deposition of americium is greater in skeleton and smaller in liver in younger age groups. Internal monitoring is by *in vivo* thorax counting and by alpha spectrometry of urine or fecal samples. None of the methods is adequately sensitive; fecal analysis is the most sensitive of the three.

Curium

Inhalation class: W

Distribution, retention and excretion functions are the same as for plutonium. All curium isotopes are taken to be uniformly distributed on bone surfaces. Techniques for internal monitoring and their limitations are the same as for americium.

Californium

Inhalation class: W - all except the following; Y - oxides, hydroxides.

Of the californium entering the transfer compartment, 0.65 goes to bone (50 y), 0.25 to liver, and 0.10 is excreted directly; 0.035% goes to testes and 0.011% to ovaries where it is permanently retained. ^{252}Cf is taken to be distributed over bone surfaces. Internal monitoring techniques and their limitations are the same as for americium.

CHAPTER 26

AGE-DEPENDENT DOSES TO MEMBERS OF THE PUBLIC FROM INTAKE OF SOME IMPORTANT RADIONUCLIDES

26.1 INTRODUCTION

ICRP publication 56, 'Age-dependent doses to members of the public from intake of radionuclides, Part 1' (1989) is a report of a Task Group of Committee 2 of ICRP. This effort was to incorporate age-dependent physical models and appropriate biokinetic information. It deals with 19 radionuclides of 12 elements. The Task Group is also to select a second category of radionuclides for assessment on a somewhat longer time scale.

ICRP 30 has used dosimetric models and biokinetic data to establish secondary limits (ALI) for the control of intakes of radionuclides by workers. ICRP does not recommend the use of these biokinetic data for calculating doses and limits on intake of radionuclides for members of the public. In its Statement of the 1983 meeting in Washington, USA, ICRP assessed those factors which, from the viewpoint of age-dependence, influence the limits on intake for members of the public and organ dose coefficients from which these limits are derived.

For this purpose it is necessary to understand the effect of age on the biokinetics of radionuclides and on anatomical and physiological data. Information is also needed on the transfer of radionuclides to the embryo and fetus following maternal intake. The necessity for internationally accepted dose coefficients for members of the public became particularly evident after the Chernobyl reactor accident.

Consideration has been limited to the most radiologically significant radionuclides that might be released to the environment due to various human activities. These include the various phases of the nuclear fuel cycle, and naturally occurring radionuclides, concentrations of which may be modified by human activities.

ICRP 56 describes biokinetic data and the dosimetric models used for these calculations. Dose coefficients for intakes by inhalation are based on the current ICRP Lung Model which does not include age-dependent parameters.

Dosimetric data for the embryo and fetus will be presented in a later ICRP report, after the necessary models have been developed. For most radionuclides the dose to the embryo can be approximated by the dose to the uterus. The dose to the fetus is dependent upon the activity present in both fetal and maternal tissues. For most radionuclides the dose to fetal tissues will be similar to or less than the dose to the corresponding maternal tissues.

26.2 DOSIMETRIC MODELS

For occupational workers the appropriate period for the integration of equivalent dose is taken as a working life-time of 50 years. For members of the public, the committed equivalent doses are calculated in ICRP 56 from the age at intake to age 70 years.

The basic methodology for dosimetry followed here is that of ICRP 30. Dose coefficients per unit intake are presented for 6 different ages. During the first year of life, considerable changes occur in the absorption from the GI tract and in body size. To provide a single value reasonably representative of this period, the intake is considered to occur at 3 months of age, when the body mass is taken to be 6 kg. SEE values are computed for the individuals of various ages considered in the anthropomorphic phantom series of Cristy and Eckerman (1987), as given in Table 26.1. Values at other ages are obtained by a linear extrapolation procedure based upon the reciprocal of the total body mass. Total body mass as a function of age is given in ICRP 23. The SEE values at age 20 years are considered to apply throughout the remainder of life. Dose coefficients are presented for ages 3 months, 1 year, 5 years, 10 years, 15 years and the adult. These values can be taken to be valid for the following age ranges:

3 months:	infants from 0 to 12 months of age
1 year:	from 1 year to 2 years
5 years:	more than 2 years to 7 years
10 years:	more than 7 years to 12 years
15 years:	more than 12 years to 17 years
adult:	more than 17 years

The kinetic energy of alpha particles, beta particles, and photons of energy less than 10 keV are considered to be absorbed fully within the source organ (i.e. $AF = 1$, when source and target are the same organ). The exceptions are the GI tract (the content of the tract is the source region and the wall is the target); the bladder (source is within the contents and the wall is the target), and the endosteal and active marrow of the skeleton.

For the GI tract and the bladder the absorbed fractions were computed as suggested by ICRP 30 using the masses given in Table 26.1. For alpha and beta particles within the skeleton, ICRP 30 data were used for all ages. (The assumption of an $AF = 1$ may lead to an overestimate of the absorbed dose within some small organs of the infant.)

Age-dependent specific absorbed fractions for photons have been tabulated by Cristy and Eckerman (1987) based on radiation transport calculations in a series of anthropomorphic phantoms representing individuals of various ages.

26.3 BIOKINETIC MODELS

26.3.1 GI Absorption

The ICRP 30 GI tract model is used for retention time parameters in various sections of the GI tract. The transit times of material through the GI tract is age-dependent, being smaller for children (Corazziari *et al.*, 1985). The use of the adult parameters for children will overestimate doses to different parts of the GI tract for long-lived radionuclides. For nuclides with physical half-lives similar to the residence time in the stomach, the overestimate of the residence time in the stomach of children could result in a slight underestimate of the activity absorbed from the small intestine and thus of the absorbed dose to other organs.

26.3.2 Absorption in Infants

The absorption of radionuclides tends to be greater in the newborn. Where no data are available, the following approach was adopted. For fractional absorption values between 0.01 and 0.5 in the adult, an increase by a factor of 2 is assumed for the first year of life. For elements with fractional absorption less than 0.01 (e.g. actinides), a value 10 times the adult value is assumed.

The increased GI absorption observed in the immediate postnatal period is associated with high levels of intestinal retention in some mammalian species (Inaba *et al.*, 1984; Sullivan *et al.*, 1987; Fritsch *et al.*, 1988). It seems reasonable to assume that radionuclide retention of actinides in the newborn will closely resemble the values observed in primates and guinea pigs.

26.3.3 Different Models Used

26.3.3.1 Respiratory Tract Model

The ICRP 30 Lung Model has been used.

26.3.3.2 Transfer Compartment Model

The ICRP 30 model has been used.

26.3.3.3 Bone Model for Actinides

Due to the limitations of the ICRP bone model for actinides (ICRP 48), it was felt necessary to develop a better, physiologically based, age-specific model. Two models can be particularly mentioned, viz. those of Leggett *et al.* and Priest (Leggett *et al.*, 1984; Leggett and Eckerman, 1984; Leggett and Warren, 1987; Priest, 1987). ICRP 56 has used a simplified version of the Leggett model for the purpose of dosimetry. This model has been described in Chapter 23, Section 4.

In Table 25.5 giving the metabolic data for the relevant elements, a brief indication has also been given of the age-dependence of the biokinetic parameters for the radionuclides considered in ICRP 56.

26.4 RESULTS OF DOSIMETRIC COMPUTATIONS GIVEN IN ICRP 56

ICRP 56 gives committed equivalent doses to 21 organs (adrenals, bladder wall, bone surfaces, brain, breast, four separate constituents of the GI tract, viz., stomach wall, small intestine wall, upper large intestine wall and lower large intestine wall, kidneys, liver, lungs, ovaries, pancreas, red marrow, skin, spleen, testes, thymus, thyroid and uterus) as well as the effective doses for different important radionuclides for the age groups mentioned earlier.

Table 26.2 gives the committed effective doses for the case of ingestion. The adult values are tabulated first. A comparison with Table 25.2 giving the committed effective doses for occupational workers shows that the differences between the two are only minor. Next are tabulated the ratios of the committed effective dose for age y to that for the adult, for the age groups given in ICRP 56.

If total body mass were the only parameter that determines the age variation of the dose per unit intake, the ratio of the committed effective dose at age y to that for the adult should be equal to the inverse of the ratio of body mass at age y to adult body mass. The corresponding values are 11.7, 7.1, 3.7, 2.2 and 1.3 respectively for 3 months (body mass 6 kg), 1 y, 5 y, 10 y, and 15 y. Significant variations from the above values are seen. This is because the ratios of organ weights at any age to the adult value are not equal to the corresponding ratios of total body masses, due to differential organ growth as a function of age. A further factor that contributes to the difference is the variation in biokinetic behaviour with age. Variations in distances between source and target organs will also make an age-dependent difference in the contribution to the dose from photons. It may be worth noting that for cesium isotopes the committed effective dose per unit intake decreases from the value for the newborn as age increases to about 10 years, and then again increases at around 15 years.

Table 26.3 gives, for the various radionuclides, the organ that receives the highest committed equivalent dose as well as the ratio of that dose to the committed effective dose for the case of ingestion. Such tabulations are given for the two extreme cases of age range, viz. adult and 3 month old. It may be seen that, by and large, the organ that receives the highest dose is the same in both cases. Tables 26.4 and 26.5 give corresponding data for inhalation, similar to the data given for ingestion in Tables 26.2 and 26.3 respectively.

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Table 26.1: Reference organ masses used for calculating SEE
(from ICRP 56)

Organ	Mass of organ (g)					
	Newborn	1 year	5 year	10 year	15 year	ICRP Reference Man
	6 kg	9.8 kg	19 kg	32 kg	55 kg	70 kg
Adrenals	5.83	3.52	5.27	7.22	10.5	14.0
Brain	325	884	1260	1360	1410	1400
Breast	0.107	0.732	1.51	2.60	360	360
Gall bladder wall	0.408	0.910	3.73	7.28	9.27	10.0
GI tract						
LLI wall*	7.96	20.6	41.4	70.0	127	160
SI wall*	32.6	84.9	169	286	516	640
Stomach wall	6.41	21.8	49.1	85.1	118	150
ULI wall*	10.5	27.8	55.2	93.4	168	210
Heart wall	25.4	50.6	92.8	151	241	330
Kidneys	22.9	62.9	116	173	248	310
Liver	121	292	584	887	1400	1800
Lungs	50.6	143	290	453	651	1000
Ovaries	0.328	0.714	1.73	3.13	11.0	11.0
Pancreas	2.80	10.3	23.6	30.0	64.9	100

(contd ...)

Organ	Mass of organ (g)					
	Newborn	1 year	5 year	10 year	15 year	ICRP Reference Man
	6 kg	9.8 kg	19 kg	32 kg	55 kg	70 kg
Skeleton						
Active Marrow	47	150	320	610	1050	1500
Endosteal Tissue	15.0	26.0	37.0	68.0	120	120
Skin	118	271	538	888	2150	2600
Spleen	9.11	25.5	48.3	77.4	123	180
Testes	0.843	1.21	1.63	1.89	15.5	35.0
Thymus	11.3	22.9	29.6	31.4	28.4	20.0
Thyroid	1.29	1.78	3.45	7.93	12.4	20.0
Urinary Bladder Wall	2.88	7.70	14.5	23.2	35.9	45.0
Uterus	3.85	1.45	2.70	4.16	80.0	80.0

* LLI - lower large intestine; ULI - upper large intestine; SI - small intestine

Table 26.2: Age-dependent committed effective doses (E) (from age of intake to age 70 years) for various age groups for members of the public from ingestion of some important radionuclides
(based on ICRP 56)

Radio-nuclide	E Adult (Sv/Bq)	$E_{age\ y}/E_{adult}$				
		3 months	1 y	5 y	10 y	15 y
^{3}H Triti-ated water	1.6×10^{-11}	3.4	2.6	1.6	1.2	1.0
^{3}H OBT	4.0×10^{-11}	2.5	2.8	1.7	1.4	1.0
^{14}C	5.6×10^{-10}	2.3	2.7	1.7	1.4	1.0
^{90}Sr	3.5×10^{-8}	3.7	2.6	1.2	1.2	1.9
^{95}Zr	1.1×10^{-9}	9.1	6.0	3.3	2.0	1.3
^{95}Nb	6.8×10^{-10}	7.6	5.4	3.1	1.9	1.3
^{103}Ru	8.1×10^{-10}	9.5	6.3	3.3	2.1	1.2
^{106}Ru	7.5×10^{-9}	11.9	7.1	3.3	2.1	1.2
^{129}I	6.4×10^{-8}	1.7	2.0	1.6	1.7	1.3
^{131}I	1.3×10^{-8}	8.5	8.5	4.8	2.5	1.6
^{132}I	1.7×10^{-10}	10.6	8.8	4.5	2.2	1.5
^{134}Cs	1.9×10^{-8}	1.3	0.8	0.7	0.7	1.1
^{137}Cs	1.3×10^{-8}	1.5	0.8	0.7	0.8	1.1
^{144}Ce	5.8×10^{-9}	13.8	7.4	3.6	2.2	1.2

(contd...)

Radio-nuclide	E Adult (Sv/Bq)	$E_{age\ y}/E_{adult}$				
		3 months	1 y	5 y	10 y	15 y
^{237}Np	$4.5x10^{-7}$	12.2	1.1	1.0	0.9	1.0
^{239}Np	$8.7x10^{-10}$	11.0	7.2	3.7	2.2	1.3
^{238}Pu	$8.8x10^{-7}$	14.7	1.4	1.1	1.0	1.0
^{239}Pu	$9.7x10^{-7}$	14.4	1.4	1.1	1.0	1.0
^{241}Pu	$1.9x10^{-8}$	11.6	1.2	1.1	1.1	1.1
^{241}Am	$8.9x10^{-7}$	13.5	1.3	1.1	1.0	1.0

Table 26.3: Doses to organs getting highest committed equivalent dose (expressed as a multiple of the committed effective dose) for adults and 3 month olds for members of the public from ingestion of some important radionuclides
(based on ICRP 56)

Radio-nuclide	Adult		3 months	
	Organ	*H/E*	Organ	*H/E*
^{3}H Tritiated water	All organs get same dose			
^{3}H OBT	All organs get same dose			
^{14}C	All organs get same dose			
^{90}Sr	Bone surface	10.9	Red marrow	5.5
^{95}Zr	LLI wall	7.1	LLI wall	7.8
^{95}Nb	LLI wall	5.9	LLI wall	6.7
^{103}Ru	LLI wall	8.1	LLI wall	8.4
^{106}Ru	LLI wall	9.4	LLI wall	8.8
^{129}I	Thyroid	32.8	Thyroid	33.6
^{131}I	Thyroid	33.8	Thyroid	33.6
^{132}I	Thyroid	20.6	Thyroid	22.8
^{134}Cs	Soft tissue	1.2	Soft tissue	1.1
^{137}Cs	Bone surface	1.2	Red marrow	1.4

(*contd* ...)

Radio-nuclide	Adult		3 months	
	Organ	*H/E*	Organ	*H/E*
^{144}Ce	LLI wall	11.4	LLI wall	9.5
^{237}Np	Bone surface	26.7	Bone surface	18.2
^{239}Np	LLI wall	10.1	LLI wall	10.2
^{238}Pu	Bone surface	19.3	Bone surface	12.3
^{239}Pu	Bone surface	18.6	Bone surface	12.9
^{241}Pu	Bone surface	19.5	Bone surface	15.0
^{241}Am	Bone surface	22.5	Bone surface	16.7

H : committed equivalent dose to organ; E : committed effective dose.

Table 26.4: Age-dependent committed effective doses (E) (from age of intake to age 70 years) for various age groups for members of the public from inhalation of some important radionuclides
(based on ICRP 56)

Radio-nuclide	Class	Adult E (Sv/Bq)	$E_{age\ y}/E_{adult}$				
			3 months	1 y	5 y	10 y	15 y
^{3}H Tritiated water	V	$1.6x10^{-11}$	3.4	2.6	1.6	1.2	1.0
^{3}H OBT	V	$4.0x10^{-11}$	2.5	2.8	1.7	1.4	1.0
^{90}Sr	D	$6.0x10^{-8}$	2.0	1.7	1.1	1.0	1.5
^{95}Zr	D	$7.3x10^{-9}$	6.0	4.7	2.9	1.8	1.2
	W	$4.7x10^{-9}$	7.4	5.3	3.0	1.9	1.3
	Y	$6.7x10^{-9}$	7.5	5.5	3.0	1.9	1.4
^{95}Nb	D	$1.4x10^{-9}$	6.4	4.6	2.8	1.6	1.2
	W	$1.4x10^{-9}$	7.1	5.1	2.9	1.9	1.3
	Y	$1.7x10^{-9}$	7.1	5.1	2.9	2.9	1.4
^{103}Ru	D	$8.1x10^{-10}$	8.3	5.7	3.1	2.0	1.2
	W	$1.8x10^{-9}$	8.9	6.1	3.2	2.2	1.4
	Y	$2.5x10^{-9}$	8.8	6.0	3.2	2.1	1.2
^{106}Ru	D	$1.6x10^{-8}$	8.8	6.1	3.3	1.9	1.2
	W	$3.2x10^{-8}$	9.7	6.6	3.4	2.2	1.4
	Y	$1.3x10^{-7}$	7.5	5.8	3.2	2.1	1.4
^{129}I	D	$4.0x10^{-8}$	1.8	2.1	1.7	1.8	1.3

(*Contd...*)

Radio-nuclide	Class	Adult E (Sv/Bq)	$E_{age\ y}/E_{adult}$				
			3 months	1 y	5 y	10 y	15 y
^{131}I	D	8.2×10^{-9}	8.4	8.1	4.8	2.4	1.6
^{132}I	D	9.9×10^{-11}	10.1	8.1	4.2	2.2	1.5
^{134}Cs	D	1.2×10^{-8}	1.4	0.8	0.7	0.8	1.1
^{137}Cs	D	8.6×10^{-9}	1.5	0.9	0.7	0.8	1.0
^{144}Ce	W	4.6×10^{-8}	9.1	6.3	3.5	2.7	1.4
	Y	1.0×10^{-7}	7.9	6.0	3.3	2.1	1.4
^{237}Np	W	5.5×10^{-5}	1.5	1.2	1.1	0.9	1.1
^{239}Np	W	6.4×10^{-10}	10.6	6.7	3.4	2.2	1.4
^{238}Pu	W	1.1×10^{-4}	1.6	1.5	1.2	1.0	1.0
	Y	7.9×10^{-5}	2.9	2.5	1.8	1.4	1.1
^{239}Pu	W	1.2×10^{-4}	1.6	1.4	1.2	1.0	1.0
	Y	8.4×10^{-5}	2.9	2.4	1.9	1.3	1.1
^{241}Pu	W	2.3×10^{-6}	1.2	1.2	1.1	1.0	1.0
	Y	1.4×10^{-6}	1.6	1.5	1.3	1.1	1.1
^{241}Am	W	1.1×10^{-4}	1.5	1.4	1.2	1.0	1.0

Table 26.5: Doses to organs getting highest committed equivalent dose (expressed as a multiple of the committed effective dose) for adults and 3 month olds for members of the public from inhalation of some important radionuclides

(based on ICRP 56)

Radionuclide	Class	Adult		3 months	
		Organ	*H/E*	Organ	*H/E*
^{3}H Tritiated water	V	All organs get same dose			
^{3}H OBT	V	All organs get same dose			
^{14}C	V	All organs get same dose			
^{90}Sr	D	Bone surface	11.2	Bone surface	7.7
^{95}Zr	D	Bone surface	13.7	Bone surface	12.3
	W	Bone surface	4.9	Lungs	4.6
	Y	Lungs	6.1	Lungs	6.4
^{95}Nb	D	Bone surface	5.0	Bone surface	6.2
	W	Lungs	4.0	Lungs	4.4
	Y	Lungs	5.0	Lungs	5.4
^{103}Ru	D	LLI wall	2.1	LLI wall	2.2
	W	Lungs	5.5	Lungs	5.9
	Y	Lungs	6.4	Lungs	6.3

(*Contd...*)

Radio-nuclide	Class	Adult		3 months	
		Organ	*H/E*	Organ	*H/E*
^{106}Ru	D	LLI wall	1.6	LLI wall	1.6
	W	Lungs	6.6	LLI wall	1.3
	Y	Lungs	7.7	Lungs	8.0
^{129}I	D	Thyroid	32.0	Thyroid	32.9
^{131}I	D	Thyroid	32.9	Thyroid	33.3
^{132}I	D	Thyroid	16.2	Thyroid	18.0
^{134}Cs	D	Soft tissue	1.2	Lungs	1.2
^{137}Cs	D	Bone surface	1.1	Lungs	1.5
^{144}Ce	W	Liver	4.6	Lungs	4.3
	Y	Lungs	7.9	Lungs	7.8
^{237}Np	W	Bone surface	25.5	Bone surface	14.1
^{239}Np	W	LLI wall	4.7	LLI wall	4.9
^{238}Pu	W	Bone surface	18.2	Bone surface	11.1
	Y	Bone surface	9.6	Lungs	6.5
^{239}Pu	W	Bone surface	18.3	Bone surface	11.6
	Y	Bone surface	10.4	Lungs	5.8

(*Contd...*)

Radio-nuclide	Class	Adult		3 months	
		Organ	*H/E*	Organ	*H/E*
^{241}Pu	W	Bone surface	19.6	Bone surface	15.6
	Y	Bone surface	13.6	Bone surface	9.5
^{241}Am	W	Bone surface	22.7	Bone surface	14.1

H: committed equivalent dose to organ; *E*: committed effective dose

CHAPTER 27

RADIONUCLIDE RELEASES INTO THE ENVIRONMENT: ASSESSMENT OF DOSES TO MAN

27.1 INTRODUCTION

Radioactive materials are present in man's environment as the result both of natural processes and of technological developments. It is important to humanity's future that the added radioactive materials due to these developments be limited and controlled so as to protect populations and their environment.

The releases of man-made radioactive material to the environment from nuclear installations give rise to some radiation doses, which may range in some cases up to tens of millirems per year (hundreds of microsieverts per year). There will be a wide range of doses, and the number of people receiving smaller doses will be much greater than the number of people receiving the higher doses. Both the level of dose and the number of people will depend on the properties of the release and the environment to which it is made. The recommendations of the ICRP dose limits for individual members of the public (which apply no matter how many individuals are exposed), as well as the ALARA directive (which involves also consideration of the number of people exposed and the dose distribution among them) are applicable to such situations. National and regional authorities have the tasks of setting limits on deliberate releases of radioactive materials in the environment and of planning interventions after accidental releases.

ICRP publication 29, 'Radionuclide release into the environment: assessment of doses to man' (1979) is aimed at indicating how these tasks can be undertaken in a way to be consistent with ICRP recommendations. The report is also intended to help the operators of installations concerned with such releases by providing them with mathematical models which can be used to predict the doses or dose commitments that may result from their proposals.

The report describes two mathematical methods, viz. concentration factor (CF) and systems analysis (SA) methods, which are useful for this

purpose. This process usually takes the form of successive approximations, each improved by incorporation of more precise or more comprehensive kinetic data, and leading to a final appraisal in which the results of environmental monitoring programmes are used to correct the predictive models of earlier stages. This report and ICRP report 43, 'Principles of monitoring for radiation protection of the population' (1985) (which has superseded ICRP publication 7, 'Principles of environmental monitoring related to the handling of radioactive material' (1966)) are supplementary to each other.

ICRP 7 and ICRP 43 have introduced the concepts of critical nuclides, critical exposure pathways, and critical population groups, and explained how they could be applied in the design of environmental monitoring programmes and used in interpreting environmental monitoring data. The rationale and formalism recommended in ICRP 29 can be used to identify the critical nuclides, pathways and population groups. As operations begin and environmental monitoring results accumulate, inputs to the predictive models will improve, coming closer to reality, as will the focus and relevance of the monitoring programme.

27.1.1 Types of Release

Releases may be of the following four types:

27.1.1.1 Planned vs. Unplanned Releases

Planned releases are subject to limitation on doses or dose commitments. Unplanned releases, while they may be subject to engineered safeguards and emergency plans, will not always give rise to doses lower than the limits and will be subject to different types of decisions.

27.1.1.2 Chronic or Acute Releases

Chronic releases may be essentially continuous, albeit subject to day-to-day or year-to-year variations. Acute releases take the form of short-period releases associated either with one event or with a short series of events. The two categories mentioned in Sec. 27.1.1.1, viz. planned and unplanned releases, may each be of either of the latter two types, viz. chronic or acute.

For example, most discharges of liquid or gaseous effluents from nuclear plants are of the planned, chronic type, while those associated with peace-

ful uses of nuclear explosives would be planned and acute. Environmental releases from nuclear power plant accidents or from transport accidents would be unplanned and acute. Releases from land burial of wastes in humid environments or from waste ponds would be unplanned and chronic. The distinction of major practical importance is between continuous and short-term releases.

27.1.2 Exposure Pathways

Simplified representations of the pathways are shown in Figs. 27.1 and 27.2. Reference may also be made to Chapter 32 which summarizes the contents of ICRP 43, 'Principles of monitoring for the radiation protection of the population'.

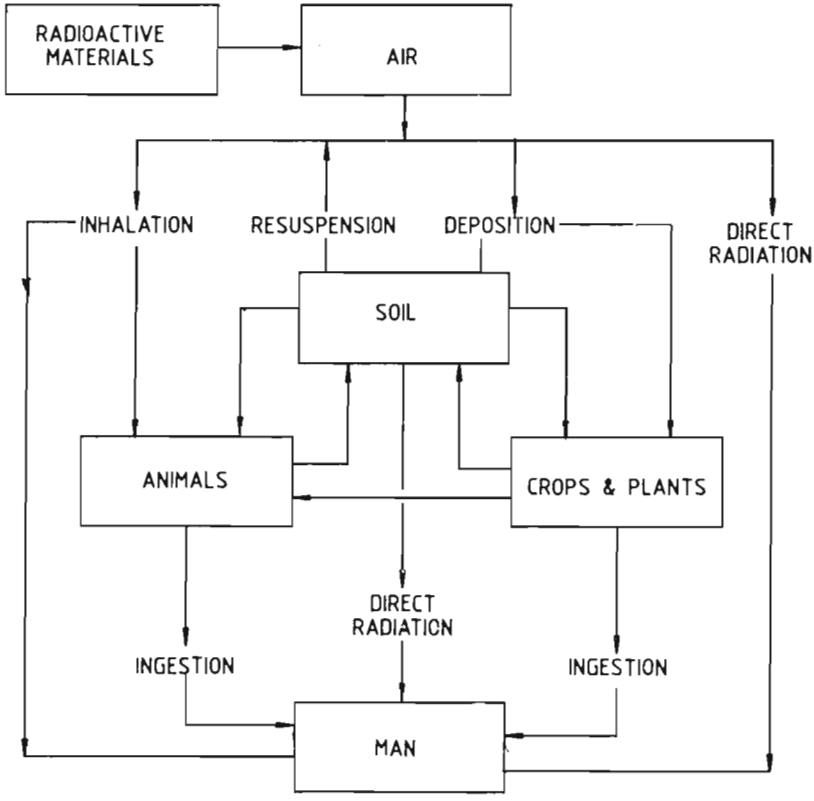

Figure 27.1. Simplified pathways to man from radioactive materials released to atmosphere (from ICRP 43).

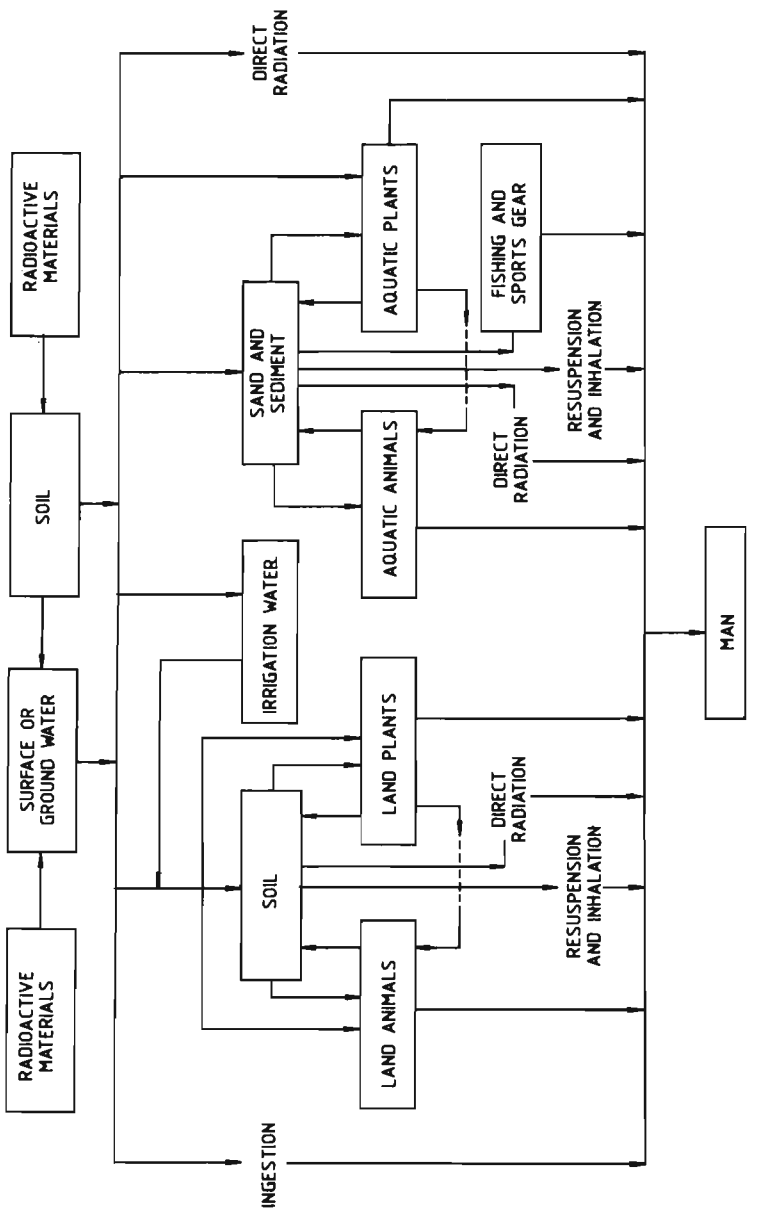

Figure 27.2. Simplified pathways to man from radioactive materials released to ground or surface waters (including oceans) (from ICRP 43).

27.1.3 Pre-operational Assessment of Releases

Pre-operational assessment of releases involves the following three considerations:

(a) Properties of postulated planned releases: types, quantities, physical and chemical form; location and medium for initial release (air or water); temperature, density, etc.; estimates of possibilities of various types of accidents and consequential unplanned releases (based on a detailed review of the installation);

(b) Natural and man-made features of the environment; its utilization for agriculture, fisheries, water and food supplies, industry and recreation; distribution and habits of the populations in the environs (age distribution, types of housing, habits);

This complex evaluation should be made in steps, the first being semiquantitative, and should identify the critical nuclides, pathways and groups, followed by an estimate of doses resulting from a postulated release; the next step is to check whether there are any large groups exposed, even at levels well below the exposure levels of the critical group, and to predict their collective dose;

(c) Combination of inputs from (a) and (b) with other relevant factors such as the ease with which releases can be reduced, future intentions which might be compromised by the presence of radioactivity, and the need for simple standards for convenient methods of inspection against these standards. This is essentially a matter for local or national decisions.

In the majority of situations the preliminary assessments will show that the predicted doses are extremely small compared with the dose limits. In this case little work is required to refine the preliminary assessments which would already have provided sufficient information for decision making. In the few cases where this is not so, further environmental studies may be necessary.

At the preliminary stage there will be some gaps and uncertainties. The gaps are normally filled by conservative assumptions. (For optimization the models should be realistic rather than conservative.) Sensitivity and robustness analysis should accompany these preliminary estimates.

In the equilibrium conditions associated with prolonged routine releases, annual individual and collective doses are convenient and satisfactory. For releases (deliberate or accidental; single or multiple), the life-

time doses to an individual and dose commitment to populations are more appropriate. The aim should be to limit the consequences of each year of practice as effectively as they would be limited if all the doses to individuals occurred within the year. Continuous routine releases, grossly nonuniform releases, and single releases can then be treated on an equal footing.

In some cases dose rate to individuals is only slightly dependent on the individual characteristics (e.g. ^{85}Kr, ^{14}C, tritiated water, and to a smaller extent, ^{90}Sr and ^{137}Cs). Substantial groups may live in environments where the annual dose is very small but where the collective dose may be substantial. Derived and authorized limits will have to be established to relate the monitored parameters to the primary doses.

27.2 METHODS FOR ASSESSING THE CONSEQUENCES OF PLANNED AND UNPLANNED RELEASES

A suitable model should be formulated, from which the doses should be predicted, and an estimate also made of the robustness of these predictions to parameter uncertainties. Starting with an initial simple model, further refinements should be made if the resulting predicted doses are sufficiently high or sufficiently uncertain. The model should take into account engineering evaluation of the facility; meteorological and hydrological modelling; ecological transfer mechanisms, including coupling among the various levels of the ecosystem of food chains; foodstuff distribution patterns; physiological models for ingestion and inhalation pathways for dosimetry.

Setting up a food chain or inhalation pathway consists of the following steps: (i) defining the objective for modelling; (ii) forming system block diagram; (iii) identifying the translocation parameters of the system; (iv) using either the CF or the SA method to predict system response; and (v) analyzing this response for the critical nuclides and pathways and the effect of parameter uncertainties.

The modelling objective should be identified. For example, the objective might be to predict doses from external radiation, and the ingestion and inhalation pathways.

27.2.1 Forming the Block Diagram

As illustrated in Fig.27.1, the block diagram is a pictorial representation of the kinetic system of the model. Conceptualized kinetic compart-

ments are defined by labelled boxes and translocation pathways by directed interconnecting arrows between boxes. The dependent variables in the model are the concentrations of activity in the labelled compartments.

Usually, there is a spatial dependence due, for example, to dispersions of the radionuclide since its release, topographical variations, or foodstuff distribution patterns. If such dependences can be ignored, a simplified block diagram such as that in Fig.27.1 will be adequate.

Some compartments may be excluded from a model because they are unimportant to the end result, or because transfer through these compartments is very rapid compared to the time scale of the model. For example, after a pulsed input of radionuclide to the grass, inclusion of the rapidly responding compartments of uptake into the gut, blood and milk of the animal pathway would be unimportant compared to the more sluggish meat pathway, if the time scale were in years.

In addition, the connecting arrows between compartments must represent realistically the transport of materials through the system, and be associated with physically possible, important, and well-defined transfer mechanisms. The model must also ensure that mass, energy, and activity (corrected for decay) are conserved; these conservation laws put constraints upon the numerical values of the parameters.

27.2.2 Identifying and Determining Translocation Parameters

Determination of transport and transfer parameters is a difficult task. A choice has to be made from the range of values for the same parameters which will be available from literature survey, and supplemented by *de novo* determinations if necessary. In the CF method, concentration factors, which are ratios of steady-state compartments, are to be determined. For the SA method, the transfer coefficients or functions, which are the loss rates for each compartment, and transfer rates between connected compartments, are to be determined. The SA parameters are the more general; concentration factors may be calculated from the transfer coefficients, but not *vice versa.*

While arriving at the first solution using the model, it is useful to note that all translocation parameters (concentration factors or transfer coefficients) must be non-negative, and that no solution to the system equations must yield a negative quantity or concentration in a compartment. The transfer of radionuclides is usually determined by biogeochemical cycling, which is sometimes the same for the stable and radioactive isotopes of the

same element, when they are in the same physical and chemical form. For elements with complex chemical or metabolic behaviour this may not be true.

Additionally, equilibrium concentrations in the compartments and equilibrium transfer rates between compartments are determined by nonlinear combinations of transfer coefficients and total loss rates. This means that more than one set of parameters may describe a system equally well. The model chosen should therefore have compartment interconnections and parameter values that make sense, and obey constraints based on knowledge of the system being modelled, so that the model can be used to extrapolate or predict the behaviour of the system.

Either static or dynamic techniques can be used. In static fitting the time derivatives of the quantities in the compartments are equated to zero, thus specifying an equilibrium condition for each compartment. In biological terms, equilibrium conditions imply constant uptake for some compartments and constant transfers to others. The solution is compared with measured equilibrium concentrations of the stable isotopes of each element in the compartments, thereby yielding nonlinear equations relating to the parameters.

Dynamic techniques can involve an experiment where an input is connected to a compartment until its concentration reaches a convenient level; when the input is then removed, the concentration decreases exponentially with a rate constant determined by the total loss rate from the compartment. Another technique is to compare the response of the system to an instantaneous impulse with that of the model equations using a computer.

27.2.3 Predicting the Response of the System

27.2.3.1 The CF Method

After the translocation parameters have been determined, an analytical technique is used to predict model response to various conditions. The CF method is adequate for many situations involving chronic releases, because these often involve only a few predominant nuclides reaching man *via* a few predominant exposure pathways. This should permit identification of critical nuclides and pathways. In the pre-operational assessment of chronic discharges, an equilibrium may be assumed between the rate of discharge and the steady state concentrations in the environment.

Conditions can be averaged over prolonged periods if the rate of discharge is more or less constant.

In this case the system response equations are simple. Each compartment is associated with a concentration and each arrow with a concentration factor. Each concentration may then be computed by multiplying each donor compartment concentration by the appropriate concentration factor and summing over all donor compartments contributing to the given donee compartment. First, the proposed rate of release is used to determine the resultant steady state concentrations in the principal medium of the environment (air or water). Processes involving dilution or removal of radionuclides from the medium by deposition or sedimentation are considered at this stage.

The next stage is the estimation of the CF from the medium to any air, water and/or food taken in or used by humans. If the medium is directly consumed by humans, or if he or she is directly exposed to the medium, then the CF becomes unity for this application. The resultant concentration can be combined with the rate of intake of the material by members of the public and the period of exposure of the public to the medium or material, and an estimate can be made of the annual intake of radioactive material and of the annual dose. Computations are made for possible pathways, and those unlikely to be critical are dismissed. This is a fairly simple step-by-step approach.

27.2.3.1.1 Non-uniform Release Rates

A suitable period may be specified over which the non-uniform discharge rate can be averaged without invalidating the calculation based on uniform discharge rates.

27.2.3.1.2 Dose Predictions for Acute Releases

If the equilibrium annual intake or annual dose D is known for an annual release of ϕ, then a single release of ϕ in average environmental conditions will result in a total intake or dose commitment equal to D.

It will be appropriate to modify the relationship adopted for average conditions for a range of values for the different dispersion and reconcentration factors that may exist at the actual time of the release. The resultant range of predictions may give guidance on the choice of timing of planned releases. For unplanned releases, it will give a measure of the range of possible consequences.

27.2.3.2 The SA Method

The dynamic behaviour is modelled with a set of coupled compartments through which the temporal passing of radionuclides may be described by differential equations. It provides values as a function of time in non-equilibrium situations for any type of release.

The techniques are mostly adopted from linear systems and control engineering mathematics. The most realistic model would be a system of partial differential equations with coefficients varying as joint functions of time and of location. In practice, linear approximations may be feasible in spatial and temporal discretization (spatially by dividing the total area into subareas, each with internally homogeneous transfer kinetics; temporally by choosing intervals like a week or a month during which time averaging will be a valid approximation). Segments may be cross-linked accordingly, as radioactivity is transferred from one pathway to another (e.g. by stream flow, or by shipments of milk, meat and food crops). Then for any one such time interval the kinetics of the whole model may be formalized in linked segmental systems of linear, first order, differential equations with constant coefficients. The formulation for a sequence of such kinetics-invariant time intervals may finally be linked to describe seasonal variations.

In the simplest representation, each input or loss term is expressed as a rate constant multiplied by the quantity or concentration of material in the donor compartment. Analytical solutions are available in the form of exponential series involving the eigenvalues and eigenvectors of the resultant rate constant matrix. More complex models could be suitable algorithms coded for a computer.

27.2.3.2.1 Writing the Systems Equations

The systems equations can be written easily once the block diagram is completed. The rate of change of radionuclide in each compartment is equated to the difference between instantaneous external input plus input transfers from other compartments, and losses due to both radioactive decay and to output transfers to other compartments and to sinks.

Most generally, a system with initial transfers of radionuclides to and from all of m compartments as well as losses by radioactive decay and to sinks would be specified by a system of m simultaneous differential equations, each comprising a driving term, and m transfer rate coefficients. The ensemble of these m^2 transfer coefficients may be arranged in the

form of an m x m matrix of which loss rates for the various compartments constitute the diagonal. Since transfer pathways may not physically exist between each compartment and all others, many of the terms in the matrix will be zero. (In the 12-compartment model discussed in the Appendix to ICRP 29, only 32 out of the 144 transfer coefficients are non-zero.)

27.2.4 Analyzing the Response of the Model

27.2.4.1 Parameter Sensitivity

At each step of refinement of the model (whether CF or SA), sensitivity and robustness analysis should be undertaken. Sensitivity can be defined as the percent change in the dose to an organ resulting from a one percent change in any transfer coefficient. The maximum possible value of the sensitivity is unity. First, the relative importance of the various pathways can be inferred (and the critical pathways identified) by applying parameter sensitivity analysis techniques to preliminary data on the initially selected pathways. Then priorities for experimental investigation of specific pathways in the real system may be established. Parameters which have a dramatic effect on the situation if they are varied even by a small amount will have to be measured with greater precision.

27.2.4.2 Dose-Prediction Robustness

Robustness analysis seeks to quantify the degree of vulnerability of dose predictions to the imprecision in all parameters jointly. A 'robustness index' can be worked out for any specific set k of joint parameter perturbation. This index can range from zero to one, increasing with increasing closeness of the predicted dose arrived at by taking into account the joint imprecision of the predicted dose from nominal, or average, parameter values. A conservative value for the mean index of robustness $<R>$ can be arrived at. The anticipated margin of inherent imprecision in dose prediction would be $1/<R>$. It then might be, even after making allowance for this degree of uncertainty, that the predicted dose could be judged to be decisively above or below a prescribed limit. These considerations provide a logical basis for the decision-making process on dose prediction described above. As parameters become better determined, their contemplated range of uncertainty will be progressively reduced and predicted dose robustness enhanced.

27.3 USE OF DOSE PREDICTIONS IN DECISION MAKING

27.3.1 Planned Releases

One of the aims of a pre-operational assessment is to help in compliance with dose limits at an early stage of planning when it is still possible to effect changes. The probable exposure of the critical group should be established, taking into account all sources. If the predicted annual doses are more than 10% of the dose limits, national or regional authorities will need to consider apportionment of the dose limits to various sources. If the predicted dose from the proposed release is only around 1% of the dose limit, then this contribution may be regarded as minor and the concept of the critical group will then be of reduced importance. Contributions from noncritical groups should also be kept in mind, since these groups may be numerically larger.

Quantities like rate of release into the environment, concentration in environmental media, resultant individual intakes and corresponding doses are important in decision making. Dose limits may be related to each of these quantities by the methods described in ICRP 29 so as to provide limiting values to all of them. More restrictive limits may perhaps be set by national authorities, either by stipulating an arbitrary reduction factor to the dose limit, or by optimization. Monitoring should be done after operations begin in order to confirm compliance.

Planned Acute Releases: In addition to justification and optimization, dose commitment to the critical group from one year of practice should not exceed the annual dose limit.

Unplanned Acute Releases: Major unplanned releases are rare. Minor ones are more frequent, and can be treated as part of the programme of planned releases. Preplanned emergency arrangements are needed if there is a possibility of large unplanned releases; intervention levels should be established in such cases.

27.3.2 Radiological Protection Aspects of Siting of Nuclear Installations

In the siting and early planning stages, information is gathered, environmental models postulated, and doses to members of the public predicted. Different potential sites can then be compared. Waste management technology today is now such that only rarely will planned releases

have a decisive factor in the choice of site. Potentiality for large accidental releases is still an important factor in site selection for power reactors, reprocessing plants and highly active waste storage facilities. Even in these cases, the level of environmental contamination and population distribution within some tens of kilometers are the factors that are of prime importance.

27.4 EXEMPLIFICATION OF DOSE PREDICTION MODELS

27.4.1 Introduction

To model the movement of radionuclides from the carrier medium (air or water) through an environmental system to man, the system must be conceptualized. Most generally, the system, taken to be contained within a region, is subdivided into subsystems, each occupying an ecologically distinct subarea. The food chain within any such subsystem is then compartmentalized. The population residing within each subsystem is categorized by compartmentalized reference-man types and reference dietary habits. It is assumed that each subsystem is homogeneous. The subsystem models may be combined to form a model for an entire environmental system.

The dose predictions made on the basis of such a model will be subject to both internal and external imprecision, the former generated by possible inaccuracies in the system, and the latter by simplistic shortcomings in the model formulation. Numerical investigation of the sensitivity and robustness of dose predictions to variations of the translocation parameters should be an integral part of both the CF and SA methods of analysis. Sensitivity analysis singles out the crucial parameters, thereby suggesting where the most careful scrutiny of uncertainties should be made. Robustness analysis provides a means of assessing the resultant uncertainties in the predicted doses to be apprehended from joint imprecisions in all parameters simultaneously.

An appendix to ICRP 29 considers 3 illustrative cases, viz. (i) and (ii), continuous and acute atmospheric releases of ^{131}I to a hypothetical terrestrial environment; and (iii) continuous release of ^{137}Cs to a hypothetical aquatic environment. Methods of tackling the problem by both the CF and SA techniques are described. Computations are made for both adult and 0-4 year old child. Appropriate notation has been developed for characterizing the compartments and the transfer parameters. As an illustrative

case, we shall consider here briefly only the CF technique of dealing with scenario (i), viz. continuous release of ^{131}I to a terrestrial environment.

27.4.2 Continuous Atmospheric Release of ^{131}I to a Terrestrial Environment: CF Method

A multipurpose subarea is conceptualized by the block diagram of Fig.27.3. (The notation is somewhat different from that used in ICRP 29.) Fruits, vegetables, and grain grown in this area are lumped into two compartments, depending upon whether the edible portion is above or beneath the soil surface. Foodstuffs grown for cattle consumption are lumped into the compartment labelled 'pasture grass'. The cattle are depicted by two compartments, depending upon whether they produce beef or milk. (Pork or poultry are subsumed by the beef compartment.) It is assumed that a contaminated atmosphere is the only source of radioactivity. Translocation patterns are specified both by arrows and by CF designations. Compartments are numbered and their contents denoted by the symbol X_i. Two digit subscripts indicate a transfer from right to left. For example, F_{63} denotes a transfer from pasture grass (3) to milk (6). [Fig.27.3].

27.4.3 Steps in the Computational Process

Step 1: Let ϕ (μCi/d) be the rate of continuous release of ^{131}I from a stack of height h metres. The air concentration X_0, expressed in μCi/m^3, at a distance x metres from the stack, can be approximated by the sector-averaged Gaussian plume formula (Slade, 1968):

$$\sum_{p=1}^{n_s}\sum_{r=1}^{n_w}\left(\frac{8\sqrt{2}}{\Pi^{3/2}}\right)\frac{\Omega_{pr}\varphi}{\sigma_{zp}U_r^x}\exp\left(\frac{-h^2}{2\sigma_{zp}^2}\right)$$

where σ_{zp} is the vertical atmospheric dispersion parameter (turbulence index p); u_r is the wind speed (m/s) (wind speed class index r); and Ω_{pr} is the frequency distribution used to characterize the average meteorological conditions.

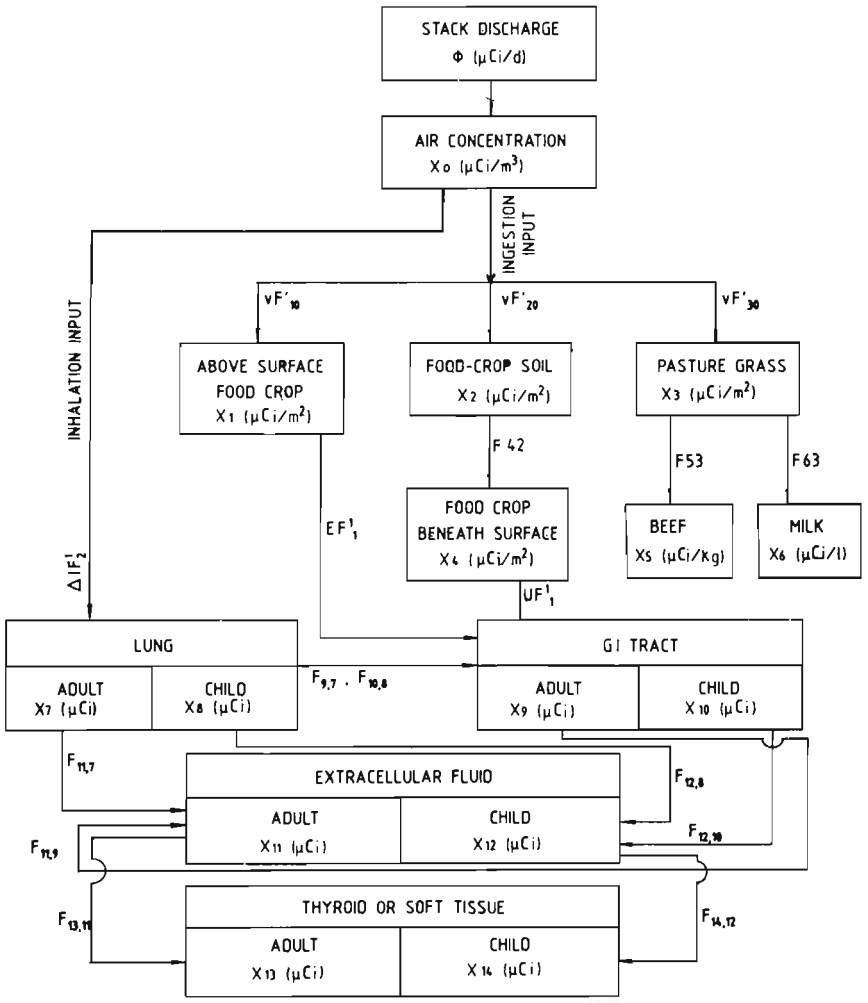

Figure 27.3. Conceptualized compartmentalization of terrestrial system for CF method (based on ICRP 29).

Step 2: The radioactivity concentrations (μCi/m^2) in compartments 1, 2 and 3 are given by vF'_{10}, vF'_{20} and vF'_{30} respectively, where v is the deposition velocity (m/s) and F'_{10}, F'_{20} and F'_{30} (in days) are the modified concentration factors for transfer from compartment 0 to compartments 1, 2 and 3 respectively.

Step 3: The concentration in compartment 4 (μCi/m^2) is governed by F_{42}, the CF for transfer from compartment 2 to 4.

Step 4: The concentrations (μCi/kg) in compartments 5 and 6 are governed respectively by F_{53} and F_{63}.

Step 5: The amount of deposition (μCi) in lung is given by $\Delta I F'_2$, where Δ is the lung retention factor, I the inhalation rate (m^3/d), and F'_2 (d) the modified concentration factor for transfer from compartment 1 to 7 or 8. While F'_2 is the same for both the adult and child, Δ and I values will be different.

Step 6: The average food-chain concentration can then by computed from the concentration parameters. The milk concentration (μCi/l), for example, is simply

$$X_6 = F_{63}(vF'_{30}).X_0.$$

Step 7: The amounts of deposition (μCi) in GI tract as a result of ingestion from compartments 1, 4, 5 and 6 are given by multiplying F'_1 with E, U, B and M respectively, where F'_1 is the modified concentration factor (d), and E, U, B and M are the age-dependent consumption of above surface food crops (m^2/d or kg/d), root crops (m^2/d or kg/d), beef (kg/d) and milk (l/d) respectively. The GI tract can also receive activity as a result of transfer from the lung compartment (governed by $F_{9,7}$, $F_{10,8}$).

Step 8: The absolute activities (μCi) rather than concentrations, and their annual integrals (μCi-d) are the quantities of primary interest as they are proportional to dose rates and doses delivered to the various tissues. They can be calculated in a step-by-step fashion by adding the contributions to any compartment from all other relevant compartments in the chain. For example, the absolute activity in the child's thyroid is given by $X_{12}F_{14,12}$.

Table 27.1A gives the specimen values of terrestrial concentration parameters for ^{131}I. Table 27.1B gives the ^{131}I concentration parameters (taken to be identical for the reference adult and reference child). Table 27.1C lists the adopted age-dependent consumption parameters.

27.4.4 Results

For an illustrative case of a ground level release (stack height h = 0) of ϕ = 1 Ci/year (37 GBq/year), computations have been made for a subarea located 1 km from the stack. Typical data have been used to characterize average meteorological conditions for a north-temperate 'prevailing westerlies' location.

Table 27.2A gives the predicted concentrations (specimen values) in compartments of the exposure pathway, while the predicted organ activities are given in Table 27.2B for adult and child.

27.4.5 Sensitivity and Robustness Analysis

A sensitivity analysis shows that the most sensitive parameter for thyroid dose is the deposition velocity v (sensitivity 0.95 to 0.99); next comes F_{30} (0.66 to 0.93), followed by F_{63} and M (0.55 to 0.90). The mean robustness index is around 0.3 for this scenario.

The conclusion is that even on a conservative appraisal, the predicted organ doses for both reference child and reference adult might be expected to be robust to terrestrial parameter inaccuracies internal to the model formulation used to within a factor of 3. Also, dose discrepancies by a factor of more than 10 can be apprehended in less than 25% of individual cases characterized by pronounced parameter misjudgements.

27.4.6 Release of ^{137}Cs to an Aquatic Environment: CF Method

Figure 27.4 gives the block diagram of the conceptualized compartments. For further details, reference may be made to ICRP 29.

27.5 COLLECTIVE DOSE PREDICTION MODELS

Appendix 2 of ICRP 29 discusses this question in detail. The problem is much more complex than for individual dose predictions. Variations in factors like radionuclide dissemination patterns, food production and processing, age-wise human population distributions over the subareas of interest, food consumption patterns, etc. have all to be taken into account.

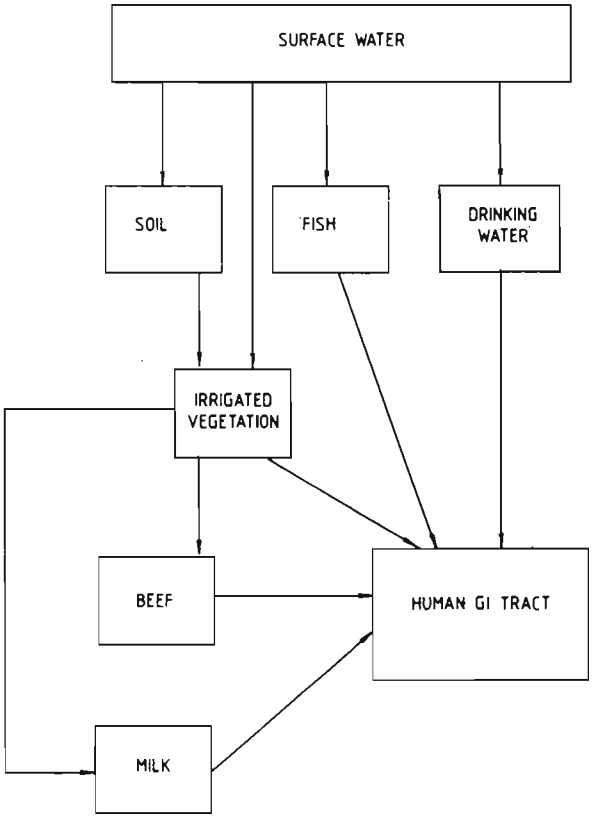

Figure 27.4. Conceptualized compartmentalization of aquatic system (based on ICRP 29).

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Table 27.1A: Nominal values of concentration parameters for ^{131}I: Terrestrial concentration parameters
(from ICRP 29)

Parameter	Nominal value
v	1.0 cm/s*
F'_{10}	1.4 d
F'_{20}	9.9 d
F_{42}	1.5×10^{-8}
F'_{30}	1.3 d
F_{53}	0.10 m^2/kg
F_{63}	0.38 m^2/l

* Specimen value only.

Table 27.1B: Nominal values of concentration parameters for ^{131}I for reference adult and reference child
(from ICRP 29)

Parameter	Nominal value
F'_1	0.058 d
F'_2	0.35 d
$F_{9,7}$, $F_{10,8}$	0.051
$F_{11,9}$, $F_{12,10}$	14
$F_{11,7}$, $F_{12,8}$	1.6
$F_{13,11}$, $F_{14,12}$	11

Table 27.1C: Specimen values of adopted age-dependent consumption parameters

(from ICRP 29)

Parameter	Unit	Nominal Value	
		Child	Adult
Lung retention factor, Δ	-	0.63	0.63
Inhalation rate, *I*	m^3/d	5.7	23
Above surface food crop consumption, *E*	kg^+/d^{++}	0.03	0.1
Root-crop consumption, *U*	kg^*/d^{**}	0.032	0.19
Beef consumption, *B*	kg^+/d	0.090	0.28
Milk consumption, *M*	l/d	0.82	0.36
Fish consumption, *P*	kg^+/d	0.0060	0.025
Water consumption, *L*	l/d	0.75	1.0

$^+$ wet mass; $^{++}$to convert to m^2/d, multiply by 0.7, and to convert to dry mass, multiply by 0.07; * dry mass; and ** to convert to m^2/d, multiply by 10.

Table 27.2A: Predicted concentrations in compartments of the exposure pathway: Specimen values [continuous ground level release of ^{131}I (1 Ci/y)]

(from ICRP 29)

Compartment	Predicted concentration, X_i
Air	0.27 pCi/m^3
Above-surface food crop	320 pCi/m^2
Soil surface below food crop	2300 pCi/m^2
Food crop beneath surface	$3.5x10^{-5}$ pCi/m^2
Pasture grass	310 pCi/m^2
Beef	31 pCi/kg
Milk	120 pCi/l

Table 27.2B: Predicted organ activity in man [continuous ground level release of ^{131}I (1 Ci/y)]

(from ICRP 29)

Compartment	Predicted activity, pCi	
	Adult	Child
GI tract	4.4	6.3
Lung	1.4	0.34
Extra-cellular fluids	63	88
Thyroid	700	970

CHAPTER 28

DATA FOR USE IN PROTECTION AGAINST EXTERNAL RADIATION

28.1 INTRODUCTION

ICRP publication 51, 'Data for use in protection against external radiation' (1987) is a report of a Task Group of ICRP Committee 3. ICRP 21 (1973) contained data on protection against ionizing radiation from external sources. The data were of two kinds, one on the relationships between various radiation quantities, the other on the shielding properties of various materials. ICRP 33 (1982) had some revised data on shielding from external sources used in medicine; these are not considered in ICRP 51.

The reasons for bringing out ICRP 51 were: (i) to adapt the data to ICRP 26 recommendations; (ii) to take account of ICRU Report 33 (1980) and ICRU Report 39 (1985) on radiation quantities and units; and (iii) to amend some data.

ICRP 51 is organized as follows: ICRP recommendations on control of external exposure are collected in Section 2 and definitions of relevant quantities are presented in Appendix A. Methods of determining dose distributions are discussed in Section 3, with special attention paid in Appendix B to transport calculations. Section 4 contains a compilation of data for interconverting radiation quantities in circumstances of idealized irradiation geometry, and there are detailed supporting data on organ doses in Appendix C. Section 5 gives information on the application of the conversion coefficients in practical circumstances. The use of the index, environmental, and individual quantities are discussed in Appendix D. Appendix E gives data (from ICRP 21) on the quality factors for charged particles. Thus the objectives of the report are to provide information on the relationships between radiometric, dosimetric and radiation protection quantities for external protection and their practical utilization. ICRP 51 is consistent with the ICRU approach but it is mainly about the protection of persons rather than matters of measurement.

28.1.1 Guidelines for Monitoring

Guidelines for monitoring of workers against external radiation have been given in ICRP 35 (1982). A similar approach is adopted here; gradually more complex or realistic procedures should be adopted as doses become higher. At low doses, dosimetric quantities may be used directly, as accuracy is not critical. Around the dose limits for workers, somewhat greater accuracy is warranted, and the conversion from dosimetric quantities to radiation protection quantities should be applied. For abnormal situations (as described in ICRP 28, 1978) actual absorbed doses in the body, from an assessment of the accident situation, should be used.

28.1.2 Limitations of Data

In general, there are adequate data for idealized irradiation geometries, mainly plane parallel beams. Extra data are needed for electrons, for non-idealized geometries, for radiations other than photons, etc. The data in ICRP 51 are applicable to workers and adult members of the public but not to children.

28.2 DETERMINATION OF DOSE DISTRIBUTIONS IN THE HUMAN BODY

28.2.1 Principal Factors

Factors affecting the dose distributions are: type of radiation; angular and spatial distribution of the incident radiation and its energy spectrum; orientation of the body in the radiation field; and configuration and composition of the body.

28.2.2 Quantities and Relationships

For expressing the basic limits, three *radiation protection quantities* (ICRU, 1980) are used, namely the mean equivalent dose in organs and tissues, the effective dose, and the dose-equivalent index (see Chapter 16 for definitions).

The derived limits are expressed in measurable quantities: (i) *radiometric quantities* related to the radiation itself (e.g. particle flux, particle fluence, energy fluence); (ii) *dosimetric quantities*, which are radiomet-

ric quantities combined with quantities associated with the interaction of radiation with matter (e.g. absorbed dose, kerma, ambient, directional and individual dose equivalent) (ICRU, 1985).

Many examples are given in ICRP 51 of the relationships between different quantities. The particle fluence is used often as a common denominator for many of the conversion coefficients. The relationships between effective dose and the deep equivalent dose index and ambient dose equivalent are of importance, since the latter two are substitutes for the first.

Dosimetric quantities, such as absorbed dose, kerma, exposure and ambient dose equivalent, can be measured and calculated. In many circumstances, the absorbed dose and dose equivalent distributions are identical when the radiation weighting factor is unity (as for photons and electrons). In other cases, the evaluation is complex.

Modelling has to be a compromise between realistic representation and simplicity for computations.

28.2.3 Phantoms of the Human Body

The ICRP 23 Reference Man (1975) is too complex for calculations. At the other extreme is the ICRU homogeneous sphere of tissue equivalent material, 30 cm in diameter (ICRU, 1980).

Homogeneous phantoms have long been used for photon dose measurements. Water phantoms, in the form of elliptic cylinders, have been widely used in radiotherapy. For neutron depth dose measurements, cubic phantoms, with a 30 cm edge, filled with tissue equivalent fluid, have sometimes been used (ICRU, 1977, 1978a, 1978b; Broerse *et al.*, 1981). Commercial phantoms (Alderson, Humanoid) are tissue equivalent for photon interactions, and a human skeleton and density-adjusted lungs are placed within a solid structure, which is sliced into transverse sections with a matrix of holes to receive dosimeters. Mr. Adam (Garry *et al.*, 1975) is the physical equivalent of the Fisher and Snyder (1967) heterogeneous phantom now in general use (Kramer, 1979). Radiation transport calculations have been made for nearly all types of phantoms.

The MIRD (Medical Internal Radiation Dosimetry Committee of the US Society of Nuclear Medicine) phantom (MIRD, 1969), originally developed for internal dosimetry, has been adopted for external dosimetry also. Elliptic cylinders represent the arms, torso and hips; a truncated

elliptic cone represents both the legs and feet; an elliptic cylinder represents the head and neck. Arms are not separated from the torso, legs are not separated, minor appendages (like fingers, feet, ears, chin and nose) are omitted, but gonads are specified. Its dimensions correspond to those of the ICRP Reference Man. Organs are represented by mathematical equations. There were 3 media in the original phantom: a skeletal region (including bone and bone marrow) of density 1.5 g/cm^2; a lung region of density 0.3 g/cm^2; and the rest of density 1.0 g/cm^2. Organs were composed primarily of H, C, N and O. In the skeleton, additional elements (mainly Ca, P) accounted for 18% of the mass. The lungs had a different tissue composition since they contain almost no fat and a larger fraction of blood than most other organs.

We now have a variety of phantoms: MIRD revised (1978); phantoms for various age groups (Cristy, 1980); sex-specific ADAM and EVA phantoms (Kramer *et al.*, 1982).

28.2.4 Transport Calculations

Two techniques are used: (i) numerical solution of the Boltzmann equation, and (ii) Monte Carlo simulation of particle interactions. The latter uses random sampling to describe the history of individual particles as they are transported through the phantom.

28.2.4.1 Boltzmann Equation

It is the general equation for transport of particles. Individual terms in the equation deal with uniform translation in space, collision of particles, continuous slowing down of charged particles, particle decay and sources (O'Brien, 1978). To get an analytical solution is very difficult. The equations are often solved numerically by replacing the continuous variables by discrete ones (Trubey and Maskewitz, 1968).

28.2.4.2 Monte Carlo Method

It is a simulation method, randomly sampling from cross-section and path length distributions to describe the history of individual particles as they are followed through the phantom. A particle history includes the initiation of a particle at its source, a random walk through the medium as it undergoes scattering interactions, and elimination (either through absorption or loss from region of interest) which terminates its history. In all Monte Carlo calculations there are statistical uncertainties associated

with an answer because an infinite number of histories cannot be followed. The relative statistical uncertainties are inversely proportional to the square root of the number of histories.

Some domains of the transport problem are more important than others in that they contribute more of the particle trajectories that make up the result. Importance sampling is a variance reduction technique which enhances the calculational effort in the areas of most interest and reduces the statistical uncertainties.

A variety of transport codes are now available. Reference may be made to ICRP 51 for details.

28.2.5 Irradiation Geometries

Figure 28.1 shows some irradiation geometries with an anthropomorphic phantom. They all relate to a plane parallel uni-directional beam of infinite extent. In the antero-posterior (AP) geometry irradiation is from the front to the back, with the beam at right angles to the long axis of the body. Conversely, in PA, the irradiation is from the back. Both these represent irradiation of a person whose orientation is fixed relative to the beam. The LAT geometry refers to lateral irradiation from the side. The ROT geometry is created by rotating the body along its axis at a uniform rate in a broad uni-directional beam at right angles to the axis of rotation. The same effect is created by rotating such a field around the long axis of the fixed body. These approximate to irradiation of a person moving randomly relative to the field orientation.

Isotropic irradiation occurs with a radiation field in which the flux of particles per unit solid angle is independent of direction. Such a geometry would be approximated by a body suspended in a semi-infinite cloud of radioactive gas.

For the ICRU sphere, four idealized geometries are sometimes discussed, three involving broad beams (single plane parallel beam, two opposed plane parallel beams, rotating plane parallel beam), with the fourth being an isotropic field. They are identified as PAR, OPP, ROT and ISO respectively.

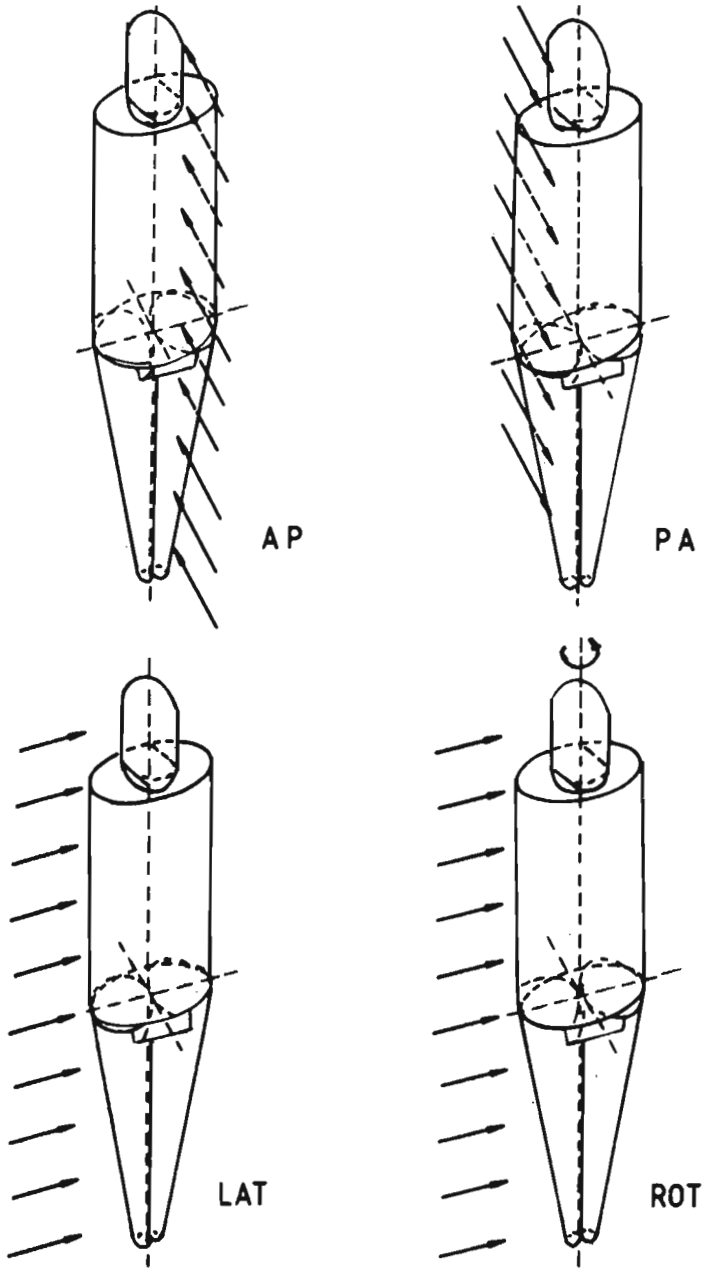

Figure 28.1. **Some irradiation geometries with an anthropomorphic phantom (from ICRP 51).**

28.3 CONVERSION COEFFICIENTS FOR IDEALIZED GEOMETRIES

28.3.1 Recommended Coefficients

Recommended conversion coefficients between the several quantities of interest in radiation protection are presented in ICRP 51. Attention is concentrated mainly on photons and neutrons. The data are presented in both tabular form and as graphs.

In the ICRP 26 system of weighting factors, a factor of $W_T = 0.06$ is applied to each of the 5 organs or tissues of the 'remainder' that receive the highest dose. The 5 organs may well be different for different energies and geometries. Special attention is to be given to the skin and eyes which are excluded from the determination of the equivalent dose.

Difficulties in the determination of conversion coefficients arise in the case of high LET radiations where the LET varies sharply with energy around the Bragg peak.

28.3.2 Data for Photons

28.3.2.1 Anthropomorphic Phantom

Conversion coefficients between effective dose and fluence are presented in Table 28.1 for photons with energies from 10 keV to 10 MeV. They are based on Monte Carlo calculations derived from the MIRD model (Kramer *et al.*, 1982; Williams *et al.*, 1985). The statistical uncertainties are less than 5%. Table 28.2 gives the relationship between effective dose and exposure for the same set of irradiation geometries and energies. Exposure is referred to a point in free air 1 m above the ground on the vertical axis of the phantom (Kramer and Drexler, 1982), but ground scatter is not taken into account. The conversion coefficients between effective dose and air kerma in free air can be approximated by dividing the numbers in the table by $0.873(\mu_{tr}/\mu_{en})$, where the expression in brackets is the ratio of the mass energy transfer and mass energy absorption coefficients for dry air; this ratio is nearly unity up to 1 MeV and differs from unity by only a few per cent at 10 MeV (Evans, 1968; Chilton *et al.*, 1984). The units are Sv/Gy.

28.3.2.2 ICRU Sphere

Tables 28.3 and 28.4 give the deep dose equivalent index $H_{i,d}$ and shallow dose equivalent index $H_{i,s}$ per unit fluence for photons incident in various geometries on the ICRU sphere (Williams *et al.*, 1983; Dimbylow and Francis, 1984). The statistical uncertainties are less than 1%. Table 28.5 gives the dose equivalent indices per unit absorbed dose to air in free air for photons incident in a plane parallel beam on the ICRU sphere.

The absorbed dose to air in free air per unit photon fluence is given in Table 28.6. ICRP 51 gives tabulations for doses to skin and lens of the eye for various irradiation conditions.

28.3.3 Data for Neutrons

After its Paris 1985 meeting, ICRP issued a statement increasing Q for neutrons from 10 to 20. The values quoted in ICRP 51 are based on calculations with the earlier Q value and have therefore to be multiplied by a factor of 2 to conform to the newer Q values.

28.3.3.1 Anthropomorphic Phantom

Table 28.7 gives the conversion coefficients for mono-energetic neutrons with energies from 2.5×10^{-8} MeV to 14 MeV, calculated by Monte Carlo methods on the modified MIRD phantom (Wittmann *et al.*, 1985; Burger *et al.*, 1981, 1985; Nagarajan *et al.*, 1981). The relative standard deviations are within 2%. Table 28.8 gives the relationship between effective dose and tissue kerma in free air.

28.3.3.2 ICRU Sphere

Tables 28.9 and 28.10 give the dose equivalent indices per unit fluence of neutrons incident on the ICRU sphere in a plane parallel beam and isotropically respectively. They were calculated by Monte Carlo method (Chen and Chilton, 1979a,b; Chilton, 1981; Chilton and Shiue, 1981; Shiue and Chilton, 1983; Lee, 1985; Lee *et al.*, 1986). Statistical uncertainties are of the order of 5% and the overall uncertainty is cautiously judged to be within 20%. In ICRP 51, values are also tabulated for a 30-cm thick semi-infinite slab phantom.

28.3.4 Data for Electrons

There appears to be relatively little information on conversion coefficients for electron irradiation of phantoms. Based on earlier work by several authors (for references, see ICRP 51), ICRP 51 developed a computer programme for Monte Carlo calculations for mono-energetic electrons with energies from 100 keV to 20 GeV for a plane parallel beam incident on a 30 cm thick infinite slab. The results are presented in Table 28.11.

28.3.5 Other Data

In ICRP 51, data are also presented for photons of energies greater than 10 MeV, as well as for muons, pions and protons. These data are not given in this Handbook.

28.4 APPLICATION OF THE CONVERSION QUANTITIES

Appendix D of ICRP 51 examines the hierarchical structure of the ICRP radiation protection quantities and links them to the practical equivalent dose quantities of ICRU Report 39 (1985).

It is shown that the index quantities are cautious substitutes for effective dose or skin dose equivalent. The combined application of the deep and shallow indices provides adequate protection for the lens of the eye. The ambient dose equivalent is a satisfactory surrogate for both the effective dose and deep dose equivalent. There is a close relationship betwee individual dose equivalent, penetrating and superficial, and effective dose and skin dose equivalent. With these data it is possible to relate the response of an idealized detector on the human body to the effective dose.

28.3.4.1 Interface Phenomena

There are two important phenomena in a radiation field at the interface between the air and the human body or phantom: the degree of charged particle equilibrium and the degree of back-scatter.

Charged particle equilibrium exists if the energies, number and direction of the charged particles are constant throughout the volume of interest at or near the interface. The back-scatter factor is the ratio of the values of a quantity at or near the surface of the body or phantom facing the source of radiation and at the same location in free air.

With the exception of the photon results for the ICRU sphere, the conversion cooefficients quoted do not take into account the degree of charged particle equilibrium. As a result, some of the conversion coefficients at shallow depths may be somewhat understimated, since some degree of equilibrium will have been established. The effect is likely to be of the second order. The magnitude of the back-scatter can be a first order effect in some cases.

28.3.4.2 Non-idealized Situations

ICRP 51 gives some suggestions on how to adapt the idealized situation data to practical cases such as divergent beams, slant incident radiation, distribution of energies of incident radiation (as compared to monoenergetic sources), partial exposure of the body, etc.

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Table 28.1: Effective dose per unit fluence for photons incident in various geometries on an anthropomorphic phantom
(from ICRP 51)

Photon energy (MeV)	Conversion coefficient, 10^{-12} Sv cm^2				
Geometry:	AP	PA	LAT	ROT	ISO
0.010	0.062	0.000	0.020	0.029	0.022
0.015	0.157	0.031	0.033	0.071	0.057
0.020	0.238	0.087	0.049	0.110	0.091
0.030	0.329	0.161	0.086	0.166	0.138
0.040	0.365	0.222	0.123	0.199	0.163
0.050	0.384	0.260	0.152	0.222	0.180
0.060	0.400	0.286	0.170	0.240	0.196
0.080	0.451	0.344	0.212	0.293	0.237
0.10	0.533	0.418	0.258	0.357	0.284
0.15	0.777	0.624	0.396	0.534	0.436
0.20	1.03	0.844	0.557	0.731	0.602
0.30	1.56	1.30	0.891	1.14	0.949
0.40	2.06	1.76	1.24	1.55	1.30
0.50	2.54	2.20	1.58	1.96	1.64
0.60	2.99	2.62	1.92	2.34	1.98
0.80	3.83	3.43	2.60	3.07	2.64
1.0	4.60	4.18	3.24	3.75	3.27
1.5	6.24	5.80	4.70	5.24	4.68
2.0	7.66	7.21	6.02	6.56	5.93
3.0	10.2	9.71	8.40	8.90	8.19
4.0	12.5	12.0	10.6	11.0	10.2
5.0	14.7	14.1	12.6	13.0	12.1
6.0	16.7	16.2	14.6	14.9	14.0
8.0	20.8	20.2	18.5	18.9	17.8
10.0	24.7	24.2	22.3	22.9	21.6

Table 28.2: Effective dose per unit exposure in free air for photons incident in various geometries on an anthropomorphic phantom
(from ICRP 51)

Photon energy (MeV)	Geometry:	Conversion coefficient, 10^{-12} Sv/R				
		AP	PA	LAT	ROT	ISO
0.010		0.007	0.000	0.002	0.003	0.003
0.015		0.044	0.090	0.090	0.020	0.016
0.020		0.131	0.039	0.023	0.059	0.050
0.030		0.440	0.232	0.131	0.228	0.187
0.040		0.784	0.490	0.281	0.434	0.352
0.050		1.04	0.703	0.41	0.601	0.488
0.060		1.21	0.865	0.513	0.726	0.592
0.080		1.28	0.980	0.602	0.833	0.675
0.10		1.25	0.982	0.608	0.840	0.668
0.15		1.13	0.909	0.577	0.778	0.635
0.20		1.06	0.861	0.569	0.746	0.614
0.30		0.984	0.823	0.563	0.720	0.600
0.40		0.951	0.811	0.570	0.715	0.599
0.50		0.932	0.808	0.581	0.717	0.602
0.60		0.919	0.807	0.592	0.719	0.609
0.80		0.904	0.810	0.613	0.725	0.623
1.0		0.896	0.815	0.632	0.732	0.638
1.5		0.888	0.827	0.670	0.748	0.666
2.0		0.888	0.837	0.698	0.762	0.688
3.0		0.895	0.855	0.739	0.783	0.720
4.0		0.905	0.868	0.765	0.797	0.739
5.0		0.914	0.879	0.785	0.810	0.755
6.0		0.921	0.889	0.802	0.822	0.770
8.0		0.932	0.905	0.828	0.846	0.797
10.0		0.939	0.918	0.849	0.869	0.822

Table 28.3: Deep dose-equivalent index, $H_{i,d}$, per unit fluence for photons incident in various geometries on the ICRU sphere
(from ICRP 51)

Photon energy (MeV)	Conversion coefficient, 10^{-12} Sv cm^2			
Geometry:	PAR	OPP	ROT	ISO
0.010	0.077	0.039	0.013	0.007
0.015	0.843	0.422	0.233	0.165
0.020	1.02	0.543	0.356	0.293
0.030	0.788	0.395	0.332	0.306
0.040	0.619	0.312	0.283	0.271
0.050	0.533	0.273	0.256	0.249
0.060	0.508	0.264	0.251	0.247
0.080	0.537	0.286	0.278	0.276
0.10	0.620	0.340	0.340	0.329
0.15	0.892	0.540	0.540	0.504
0.20	1.19	0.767	0.767	0.703
0.30	1.81	1.27	1.27	1.14
0.40	2.38	1.76	1.76	1.56
0.50	2.89	2.23	2.23	1.97
0.60	3.39	2.68	2.68	2.37
0.80	4.30	3.53	3.53	3.12
1.0	5.13	4.32	4.32	3.83
1.5	6.92	6.05	6.05	5.43
2.0	8.48	7.59	7.59	6.85
3.0	11.1	10.1	10.1	9.29
4.0	13.4	12.4	12.4	11.4
5.0	15.5	14.5	14.5	13.4
6.0	17.5	16.4	16.4	15.3
8.0	21.5	20.2	20.2	18.9
10.0	25.4	23.9	23.9	22.4

Table 28.4: Shallow dose-equivalent index, $H_{i,s}$, per unit fluence for photons incident in various geometries on the ICRU sphere
(from ICRP 51)

Photon energy (MeV)	Geometry:	Conversion coefficient, 10^{-12} Sv cm^2 PAR	OPP	ROT	ISO
0.010		6.91	3.46	3.07	2.97
0.015		3.04	1.58	1.46	1.44
0.020		1.72	1.05	1.02	0.856
0.030		0.875	0.601	0.601	0.445
0.040		0.622	0.429	0.429	0.315
0.050		0.533	0.361	0.361	0.269
0.060		0.505	0.349	0.349	0.258
0.080		0.532	0.391	0.391	0.287
0.10		0.616	0.478	0.478	0.348
0.15		0.891	0.761	0.761	0.542
0.20		1.19	1.07	1.07	0.757
0.30		1.85	1.72	1.72	1.22
0.40		2.46	2.32	2.32	1.67
0.50		3.04	2.87	2.87	2.10
0.60		3.59	3.40	3.40	2.53
0.80		4.60	4.39	4.39	3.34
1.0		5.49	5.25	5.25	4.09
1.5		7.32	7.09	7.09	5.71
2.0		8.85	8.62	8.62	7.11
3.0		11.5	11.2	11.2	9.57
4.0		13.8	13.5	13.5	11.7
5.0		15.9	15.6	15.6	13.7
6.0		17.9	17.6	17.6	15.6
8.0		22.0	21.4	21.4	19.3
10.0		26.1	25.1	25.1	22.9

Table 28.5: Dose equivalent indices per unit absorbed dose to air in free air for photons incident in a plane parallel beam on the ICRU sphere (from ICRP 51)

Photon energy	Conversion coefficient, Sv/Gy	
(MeV)	$H_{i,s}$	$H_{i,d}$
0.010	0.930	0.010
0.015	0.974	0.271
0.020	1.02	0.607
0.030	1.21	1.09
0.040	1.45	1.44
0.050	1.65	1.65
0.060	1.75	1.76
0.080	1.73	1.75
0.10	1.66	1.67
0.15	1.49	1.49
0.20	1.39	1.39
0.30	1.34	1.31
0.40	1.30	1.26
0.50	1.28	1.21
0.60	1.26	1.19
0.80	1.25	1.17
1.0	1.23	1.15
1.5	1.20	1.13
2.0	1.18	1.13
3.0	1.17	1.12
4.0	1.15	1.12
5.0	1.14	1.12
6.0	1.13	1.11
8.0	1.13	1.10
10.0	1.13	1.09

Table 28.6: Absorbed dose to air in free air per unit photon fluence
(from ICRP 51)

Photon energy (Mev)	Conversion coefficient, 10^{-12} Gy cm^2
0.010	7.43
0.015	3.12
0.020	1.68
0.030	0.721
0.040	0.429
0.050	0.323
0.060	0.289
0.080	0.307
0.10	0.371
0.15	0.599
0.20	0.856
0.30	1.38
0.40	1.89
0.50	2.38
0.60	2.84
0.80	3.69
1.0	4.47
1.5	6.12
2.0	7.50
3.0	9.87
4.0	12.0
5.0	13.9
6.0	15.8
8.0	19.5
10.0	23.1

Table 28.7: Effective dose per unit fluence for neutrons incident in various geometries on an anthropomorphic phantom
(from ICRP 51)

Photon energy (MeV)	Conversion coefficient, 10^{-12} Sv cm^2			
Geometry:	AP	PA	LAT	ROT
2.5×10^{-8}	(4.00)	(2.60)	(1.30)	(2.30)
1.0×10^{-7}	(4.40)	(2.70)	(1.40)	(2.40)
1.0×10^{-6}	4.82	2.81	1.43	2.63
1.0×10^{-5}	4.46	2.78	1.33	2.48
1.0×10^{-4}	4.14	2.63	1.27	2.33
1.0×10^{-3}	3.83	2.49	1.19	2.18
1.0×10^{-2}	4.53	2.58	1.27	2.41
2.0×10^{-2}	5.87	2.79	1.46	2.89
5.0×10^{-2}	10.9	3.64	2.14	4.70
1.0×10^{-1}	19.8	5.69	3.57	8.15
2.0×10^{-1}	38.6	8.60	6.94	15.3
5.0×10^{-1}	87.0	30.8	18.7	38.8
1.0×10^{0}	143	53.5	33.3	65.7
1.5×10^{0}	183	85.8	52.1	93.7
2.0×10^{0}	214	120	71.8	120
3.0×10^{0}	264	174	105	162
4.0×10^{0}	300	215	131	195
5.0×10^{0}	327	244	151	219
6.0×10^{0}	347	265	167	237
7.0×10^{0}	365	283	181	253
8.0×10^{0}	380	296	194	266
1.0×10^{1}	410	321	218	292
1.4×10^{1}	(480)	(415)	(280)	(365)

Note: These conversion coefficients are for neutron Q of 10 as per ICRP 26. They should be multiplied by a factor of 2 to obtain the revised equivalent dose with the revised 1985 value of $Q = 20$ for neutrons. Values in parentheses are extrapolated from original data.

Table 28.8: Effective dose per unit tissue kerma in free air for neutrons incident in various geometries on an anthropomorphic phantom
(from ICRP 51)

Photon energy (MeV)	Geometry:	Conversion coefficient, 10^{-12} Sv/Gy			
		AP	PA	LAT	ROT
2.5×10^{-8}		(19.1)	(12.6)	(1.30)	(2.30)
1.0×10^{-7}		(42.7)	(26.2)	(13.6)	(23.8)
1.0×10^{-6}		146	83.9	42.7	78.5
1.0×10^{-5}		384	240	115	214
1.0×10^{-4}		307	195	94.1	173
1.0×10^{-3}		37.9	24.7	11.8	21.6
1.0×10^{-2}		4.76	2.71	1.33	2.53
2.0×10^{-2}		3.24	1.54	0.80	1.60
5.0×10^{-2}		2.79	0.933	0.549	1.21
1.0×10^{-1}		3.07	0.884	0.554	1.27
2.0×10^{-1}		3.90	0.870	0.702	1.55
5.0×10^{-1}		5.51	1.95	1.18	2.46
1.0×10^{0}		5.77	2.16	1.34	2.65
1.5×10^{0}		6.78	3.18	1.93	3.47
2.0×10^{0}		7.04	3.95	2.36	3.95
3.0×10^{0}		7.31	4.82	2.91	4.49
4.0×10^{0}		7.21	5.17	3.15	4.69
5.0×10^{0}		7.25	5.41	3.35	4.86
6.0×10^{0}		7.45	5.69	3.58	5.09
7.0×10^{0}		7.21	5.59	3.58	4.64
8.0×10^{0}		7.31	5.69	3.73	5.12
1.0×10^{1}		7.22	5.65	3.84	5.14
1.4×10^{1}		(7.31)	(6.32)	(4.26)	(5.56)

Note: These conversion coefficients are for neutron Q of 10 as per ICRP 26. They should be multiplied by a factor of 2 to obtain the revised equivalent dose with the revised 1985 value of $Q = 20$ for neutrons. Values in parentheses are extrapolated from original data.

Table 28.9: Dose equivalent indices per unit fluence for neutrons incident in a plane parallel beam on the ICRU sphere
(from ICRP51)

Neutron energy (MeV)	Conversion coefficient, 10^{-12} Sv cm^2	
	$H_{i,d}$	$H_{i,s}$
2.5×10^{-8}	8.00	9.50
1.0×10^{-7}	10.1	11.1
1.0×10^{-6}	11.8	11.2
1.0×10^{-5}	11.6	9.2
1.0×10^{-4}	11.0	7.1
1.0×10^{-3}	9.50	6.2
1.0×10^{-2}	8.60	9.5
2.0×10^{-2}	14.5	19.9
5.0×10^{-2}	35.0	50.6
1.0×10^{-1}	69.0	96
2.0×10^{-1}	124	151
5.0×10^{-1}	258	279
1.0×10^{0}	339	364
1.5×10^{0}	363	375
2.0×10^{0}	358	369
3.0×10^{0}	388	418
4.0×10^{0}	418	439
5.0×10^{0}	396	402
6.0×10^{0}	402	415
7.0×10^{0}	416	444
8.0×10^{0}	435	464
1.0×10^{1}	464	481
1.4×10^{1}	520	520
1.7×10^{1}	610	640
2.0×10^{1}	650	660

Note: These conversion coefficients are for neutron Q of 10 as per ICRP 26. They should be multiplied by a factor of 2 to obtain the revised equivalent dose with the revised 1985 value of $Q = 20$ for neutrons.

Table 28.10: Dose-equivalent indices per unit fluence for neutrons incident isotropically on the ICRU sphere
(from ICRP 51)

Neutron energy (MeV)	Conversion coefficient, 10^{-12} Sv cm^2	
	$H_{i,d}$	$H_{i,s}$
2.5×10^{-8}	2.10	3.00
1.0×10^{-7}	2.80	3.40
1.0×10^{-6}	3.4	3.30
1.0×10^{-5}	3.3	2.80
1.0×10^{-4}	3.10	2.40
1.0×10^{-3}	3.00	2.00
1.0×10^{-2}	3.10	4.30
2.0×10^{-2}	4.30	9.27
5.0×10^{-2}	9.76	25.0
1.0×10^{-1}	22.0	49.0
2.0×10^{-1}	42.7	91.6
5.0×10^{-1}	84.0	136
1.0×10^{0}	135	184
1.5×10^{0}	159	195
2.0×10^{0}	175	211
3.0×10^{0}	205	224
4.0×10^{0}	236	252
5.0×10^{0}	239	247
6.0×10^{0}	250	258
7.0×10^{0}	266	277
8.0×10^{0}	277	300
1.0×10^{1}	306	340
1.4×10^{1}	380	400
1.7×10^{1}	420	440
2.0×10^{1}	490	520

Note: These conversion coefficients are for neutron Q of 10 as per ICRP 26. They should be multiplied by a factor of 2 to obtain the revised equivalent dose with the revised 1985 value of $Q = 20$ for neutrons.

Table 28.11: Maximum equivalent dose per unit fluence and equivalent dose per unit fluence at 10 mm depth for electrons incident in a plane parallel beam on a 30 cm thick semi-infinite slab phantom
(from ICRP 51)

Electron energy (MeV)	Conversion coefficient, 10^{-10} Sv cm^2	
	Max.	At 10 mm depth
1.0×10^{-1}	19.2	-
2.0×10^{-1}	12.3	-
5.0×10^{-1}	7.68	-
1.0×10^{0}	6.15	-
2.0×10^{0}	5.33	0.023
5.0×10^{0}	4.74	4.37
1.0×10^{1}	4.17	3.67
2.0×10^{1}	3.65	3.24
5.0×10^{1}	3.52	3.18
1.0×10^{2}	3.74	3.19
2.0×10^{2}	4.26	3.16
5.0×10^{2}	5.84	3.18
1.0×10^{3}	7.19	3.18
2.0×10^{3}	8.48	3.21
5.0×10^{3}	10.3	3.22
1.0×10^{4}	12.1	3.21
2.0×10^{4}	14.1	3.21

Note: Where there are no entries, the depth exceeds the range of the electrons.

Part IV

Operational Radiation Protection including Monitoring

CHAPTER 29

GENERAL PRINCIPLES OF MONITORING FOR RADIATION PROTECTION OF WORKERS

29.1 INTRODUCTION

ICRP publication 35, 'General principles of monitoring for radiation protection of workers', a report of ICRP Committee 4 (1982), supersedes ICRP publication 12 (1969) with the same title. A complement to ICRP 35 is ICRP 43, 'Principles of monitoring for the radiation protection of the population' (1985).

Monitoring is the measurement of radiation or activity for reasons related to the estimate or control of exposure to radiation or radioactive material. The term includes the interpretation of the measurements. ICRP 35 aims at establishing the broad principles on which monitoring programmes can be based in order to meet the objectives both effectively and economically. Some aspects of monitoring may be the subject of national or regional requirements.

The objectives of the monitoring programme, its design to meet these objectives, and the basis for the interpretation of the monitoring results, should all be recorded. The design should incorporate guidance on the necessary records to be maintained. All these aspects should be reviewed periodically. Monitoring is merely a technique of radiological protection; it is not an end in itself.

The aim of monitoring in ensuring the existence of satisfactory working conditions is to provide the information needed for estimating the exposure of workers in terms of their compliance with the primary or secondary dose limits. Some parameters like organ doses or intakes of radionuclides cannot, in practice, be measured directly, but have to be estimated on the basis of other, directly measurable, quantities. The interpretation of the results of measurements is based on a model which describes, in a quantitative way, the relationship between the measured and estimated quantities.

The primary dose limits relate to the sum of the effective dose (or equivalent dose to an organ) from external exposure during one year and the committed dose from the year's intake of radionuclides. In practice, use is made of the two secondary limits for external exposure, the annual limit for the deep and shallow equivalent dose indices, and the secondary limit for intake, the annual limit on intake (ALI). The use of monitoring methods capable of providing sufficiently accurate estimates of these quantities makes it possible to achieve the necessary level of protection by satisfying the following two conditions:

$$\frac{H_{I,d}}{H_{E,L}} + \sum_j \frac{I_j}{I_{j,L}} \leq 1 \quad \text{and} \quad \frac{H_{I,s}}{H_{sk,L}} \leq 1$$

where $H_{I,d}$ is the annual deep equivalent dose index, $H_{I,s}$ is the annual shallow equivalent dose index, $H_{E,L}$ is the annual limit of the effective dose, $H_{sk,L}$ is the annual limit of the equivalent dose to the skin, I_j is the annual intake of radionuclide j and $I_{j,L}$ is the ALI for radionuclide j. Conformity with both the limits for external exposure will, in most practical situations, afford compliance with the dose limits for the lens of the eye. A similar procedure may be followed to demonstrate compliance with authorized limits. Where the ALI is determined by deterministic effects, this procedure may be unnecessarily restrictive.

The decision whether to introduce individual monitoring may be based on the likelihood of external and internal exposure independently. In practice there will only be a few situations where both types of routine individual monitoring will be required. In most cases it will be sufficient to assess external and internal exposures separately. Moreover, reference levels for both types of exposure may, in most cases, be considered separately, making it unnecessary to adjust one with respect to the other. In the case of abnormal exposures, more detailed programmes of monitoring may be needed.

In some cases such as monitoring for external radiation, the relationship between a practical measurement and the dose is fairly straightforward. Even here the interpretation is based on a model of the relationship between the quantity measured (say, a dosimeter worn on a defined part of the body) and the dose. Relating a measurement of the surface contamination in a workplace to the individual dose is more complex.

A *derived limit* provides a quantitative link between the quantity being measured and the relevant dose. In specifying a derived limit the

intention should be to establish a figure such that adherence to it will provide virtual certainty of compliance with the primary limits or the operational ALARA limits. Operations should be conducted such that the derived limits are not exceeded. Changes in derived limits should be made neither lightly nor frequently.

Limits introduced by the competent authority or the management, generally on the basis of optimization, are *authorized limits*. These should be lower than the primary limits or appropriate derived limits.

A *reference level* is the predetermined value for any monitoring result which will require a certain course of action to be taken in the event that the value of the quantity exceeds (or is predicted to exceed) this level. The most common forms of reference levels are *recording*, *investigation*, and *intervention* levels.

The recording level is a formally defined value for the dose or intake above which a result from a monitoring programme is of sufficient interest to be worth keeping. ICRP has recommended that the recording level for individual monitoring should be based on one-tenth of the fraction of the annual limit corresponding to the period of time to which the measurement refers. The rationale for introducing a recording level arises from the fact that, in general, monitoring results are easy to obtain and difficult to interpret, and that a large amount of data collected in the programme is only of transitory value. The use of a recording level will mean that any result from a monitoring programme that is smaller than this value can be discarded and should be treated as zero for purposes of radiation protection. It can be entered in personal records simply by the statement that it was below the recording level. Recording levels are also useful in assessment of collective doses.

Experience has shown that much of the information obtained from a monitoring programme merely confirms that the situation is satisfactory and that no action is required. An investigation level is a value of the dose or intake (usually set in relation to a single measurement, rather than to the accumulated dose or intake in a year) above which the result is sufficiently important to justify further investigation. It is often appropriate to base the investigation level for individual monitoring on three-tenths of that fraction of the relevant annual dose limit corresponding to the period of time to which the individual measurement refers. An investigation level is not intended to be a limit and exceeding it is not to be regarded as a failure of protective measures.

An intervention level is a pre-established value for any monitoring quantity such that if this value is not exceeded or predicted to be exceeded, it is highly improbable that intervention will be required.

The purpose and nature of record keeping are influenced by national requirements (for example, compliance with local regulations, evaluation of collective doses, trends in exposure, medico-legal purposes, or for epidemiological studies). Records of doses and intakes, not merely the uninterpreted data, must be kept for at least 30 years.

29.2 THE FUNCTIONS OF MONITORING

Monitoring is of three types, *routine*, *operational*, and *special*. Routine monitoring is associated with continuing operations, operational monitoring is conducted to provide information about a particular operation, and special monitoring is applied to an actual or suspected abnormal situation. It is convenient to discuss separately monitoring of the workplace and individual monitoring of the worker.

29.2.1 Monitoring of the Workplace

Routine monitoring is largely of a confirmatory nature, intended to show that the working condition is satisfactory. It may also include fixed detectors to identify the onset of abnormal or emergency conditions (e.g. failure to return a radiographic source to its shielded container, or a criticality accident).

Operational monitoring is intended to provide a check on a particular operation and to give, if necessary, a basis for immediate decisions on the conduct of the operation (particularly in the control of short-term procedures).

Special monitoring may cover either a situation where insufficient information is available to achieve adequate control, or an operation under abnormal circumstances, including accidents or suspected accidents. It should have a limited duration and clear-cut objectives.

29.2.2 Individual Monitoring

Individual monitoring means the making of measurements by personal dosimeters or estimating body burdens by excreta measurements. Routine

individual monitoring constitutes regularly repeated or continuous measurements on an individual worker. If the estimated doses and intakes are well below the relevant limits, it will be sufficient to assess the upper limits of the estimate rather than the actual value and to assess their importance by the use of, for example, investigation levels. Where routine individual monitoring may not be adequate to estimate doses or intakes, monitoring of the workplace may have to be additionally instituted.

Operational individual monitoring can be employed, for example, by the provision of additional dosimeters, during specific operations. Special individual monitoring should be carried out in actual or suspected abnormal situations including accidents.

The results of individual monitoring can give information about working conditions. The techniques are generally simple, low in cost and easy to interpret.

Models chosen should be of general validity and ensure that the risk of underestimating the exposure is acceptably small. If the results based on the general model indicate that the protection standard is exceeded or is likely to be exceeded, a more realistic model should be evolved, to ensure that the risk of overestimation is also reduced.

A monitoring programme should be self-adjusting, and, as experience is gained, its design should be reviewed periodically. Monitoring provides one of the bases for the selection of controlled areas. It also provides information for deciding on the extent and form of medical supervision.

29.3 MONITORING OF THE WORKPLACE FOR EXTERNAL RADIATION

A comprehensive survey should be conducted when any new installation is put into service or when substantial changes have been made in an existing installation. If the radiation fields are not normally liable to change, routine monitoring is rarely needed for checking purposes. If the fields are liable to change slowly and mildly, periodic checks at preestablished points will give timely warning of deteriorating conditions. If the fields are liable to increase rapidly and unpredictably, a system of warning instruments will be required either at the workplace or on the worker.

Operational monitoring is required when the operation to be conducted is likely to influence the radiation field. Particularly in the case of beta radiations, minor manipulations may cause substantial changes in the beta component of the dose rate with only small changes in the gamma component.

Interpretation of area monitoring results is difficult. Radiation fluence rates and quality of the radiation will vary in space and time. The equivalent dose indices measured at the workplace correspond adequately to the highest doses to the worker. It may also be convenient to assume that a person will be present for the whole of the working time in areas where the equivalent dose index is highest. Similar assumptions can also be made concerning the location of the hands and of other locally exposed parts.

29.4 MONITORING OF THE WORKPLACE FOR SURFACE CONTAMINATION

Although there is not necessarily a correlation between surface contamination in the workplace and the exposure of workers, the absence of surface contamination above a defined level usually indicates a high standard of primary containment.

The objectives of a programme of monitoring for surface contamination are: to detect failures or departures from good operating procedures, to restrict surface contamination to levels at which the general standards of good housekeeping are adequate, and to provide information for the planning of optimized programmes of individual and air monitoring, and for defining operational procedures.

Conventionally, a representative fraction of the surfaces in an area is monitored. Checking of cleaning mops, surfaces at the exits from areas, and shoes or gloves worn in the area can detect slowly deteriorating conditions. Surface contamination monitors on the clean side of changerooms are intended to ensure that a worker's hands and shoes are clean before he leaves the place. Checking of items leaving a controlled or supervised area is often useful. Where only sealed sources are used, a periodic check for leakage (once in a year or so) is needed.

Routine monitoring is usually supplemented by some operational monitoring, particularly while working with partial containment, e.g. fume hoods. There is a poor correlation between surface contamination and worker

exposure. ICRP publication 25, 'Handling, use and disposal of unsealed radionuclides in hospitals and medical research establishments' (1977) gives a number of derived limits.

For many types of operation, there is no difficulty in maintaining contamination levels well below the derived limits. In many cases it is convenient to have investigation levels.

29.5 MONITORING FOR AIR CONTAMINATION

Monitoring for air contamination is likely to be needed only in installations handling large amounts, that is, in quantities thousands of times greater than the ALIs. Good housekeeping and surface contamination monitoring would be adequate in most cases. Regular air monitoring will almost always be needed in the following cases:

(a) When gaseous or volatile materials are handled in quantity (e.g. tritium, tritiated water in heavy water reactors);

(b) Handling of radioactive material (including fuel fabrication, fuel reprocessing, machining of uranium) in conditions of substantial contamination of workplaces;

(c) Processing of plutonium and other transuranics;

(d) Uranium mining, milling and refining; and

(e) Handling of unsealed radionuclides in hospitals in therapeutic quantities and the use of hot cells and critical facilities.

The programme, when necessary, should meet three objectives, viz.

(i) To assess the probable upper limit of inhalation by workers; (ii) To warn of unexpected airborne contamination; and (iii) To help in planning of programmes of individual monitoring for internal contamination.

The most common form of air monitoring is by using samplers at a number of selected locations intended to be representative of the breathing zone of the workers. Less commonly, it is necessary to make the samples more accurately representative of the breathing zone by the use of personal air samplers. To detect unexpected airborne contamination, especially in the working faces of high flux reactors and in areas where plutonium is handled in glove boxes, it may be necessary to use continuously operating samplers with a warning system.

Area samplers, even if located close to the breathing zone of workers, may not always provide data adequately representative of intakes, especially where the contamination may be localized and variable with time (e.g. leaks in glove boxes), or when the particle size is non-uniform.

Personal air samplers have a low sampling rate and single results from samplers run even for a week may be misleading as a result of bias resulting from the collection of a single non-representative particle. This effect is of much less importance in long-term average results, and it is appropriate to interpret the long-term results of personal air samplers in terms of the Derived Air Concentrations (DAC), which relate to the air actually inhaled. However, this provides only long-term control after the event, whereas the situation calling for the use of personal air samplers is usually one where alerting the need for short-term remedial action will be the priority.

Results of long-term personal air samplers and area monitoring should be compared. Where such cross-calibration is lacking, it is sometimes adequate to assume that the inhalation exposure of the worker is an order of magnitude greater than that indicated by the area sampler. For short-term sampling the results of a personal air sampler may differ from those of the area sampler by 2 or 3 orders of magnitude. In these circumstances it is inappropriate to establish a derived limit. An investigation level may, as an alternative, be set which is low enough to detect significant abnormalities but high enough for it not to be reached too often in normal conditions.

The ALI and DAC values are based on a standard aerosol with an activity median aerodynamic diameter (AMAD) of 1 μm and are expressed as the total activity over all particle sizes. Normal air samplers adequately reflect these total aerosol values. Results of 'respirable dust' samplers need appropriate correction.

29.6 INDIVIDUAL MONITORING FOR EXTERNAL RADIATION

The first need in designing a programme of individual monitoring is to identify the individuals for whom it must be provided. ICRP 26 has recommended that workers in Working Condition A should be subject to individual monitoring.

(Author's note; This categorization of working conditions has been abolished in ICRP 60.)

For other workers individual monitoring is not required, monitoring of the workplace being sufficient. However, external radiation monitoring is simple and easier than a comprehensive monitoring programme for the workplace.

Work with substantial sources not enclosed in fully interlocked shields will clearly require individual monitoring. Work with adequately shielded sources, work with small sources, and transport workers can be excluded from individual monitoring.

29.6.1 Situations Not Requiring Routine Individual Monitoring

ICRP 35 has given guidelines for situations which do not call for routine individual monitoring for external radiation:

29.6.1.1 Low Activity Sources

29.6.1.1.1 Gamma emitters (beta radiation fully shielded): 50 MBq.MeV (~1 mCi.MeV)

This will correspond to a dose rate at 1 m of 7.5 μSv/h (or 15 mSv/year, which is three-tenths of the ICRP 26 dose limit).

29.6.1.1.2 Beta emitters (with or without gamma radiation)

$E_{max} \geq$ 0.3 MeV 5 MBq (~100 μCi)

$E_{max} <$ 0.3 MeV 50 MBq (~1 mCi)

(It is presumed that suitable precautionary measures are followed, and that in no case will the source be held in the bare hand.)

29.6.1.2 Specified Work Areas

Routine individual monitoring is not required in the following work areas where limited individual monitoring programmes have established that operating procedures are of a high standard:

Non-radiological work in radiography departments (medical and industrial) and radiotherapy departments, teletherapy departments, dental radiography; installations using nucleonic gauges.

The dosimeter should permit estimation of the equivalent dose received over the whole range of radiations and energies with reasonable accuracy. If only one dosimeter is used, the objective is to place it in a position where it will be representative of the most highly exposed surface of the trunk. Doses to the extremities, especially to the hands, may be somewhat higher.

In special situations, where protective clothing such as lead aprons is used, as in diagnostic radiology, more than one dosimeter may be required. If a single dosimeter is used, it should be worn outside the apron, usually high on the trunk. It will provide information on doses to the skin, eye, and unshielded parts of the body, but will overestimate the effective dose. When the recorded values indicate doses approaching the dose limits, two dosimeters should be used, one over and one under the protective apron, and the combined results appropriately interpreted.

The basic choice in monitoring for beta, gamma and X-radiation is between (i) a dosimeter giving information on the doses both at the surface and at a fixed depth (usually 10 mm), and (ii) a discriminating device to give some indication of the types of radiation and their energies.

For a wide range of energies, a simple two-element thermoluminescent dosimeter which exhibits small energy dependence, e.g., LiF, with one element covered by a tissue-equivalent filter, is an example of the former type. Measurements at the surface and at a depth of 10 mm will be sufficient in almost all practical cases; they will somewhat overestimate the doses. With a suitable phantom they can be calibrated in terms of the deep and shallow dose indices; they will be accurate for a unidirectional field and adequate even for a multi-directional field.

A multi-element dosimeter using either a photographic film or thermoluminescent elements with filters of materials of different atomic numbers and thicknesses is an example of the second type.

Personal dosimeters are not yet satisfactory for neutrons in some energy regions. In most cases neutrons contribute only a fraction to the total equivalent dose. But with neutron generators, particle generators and ^{252}Cf sources, neutron doses are substantial.

Monitoring for thermal neutrons is easy, for example, by measurement of capture gamma rays in cadmium by a photographic film. But situations where the neutron spectrum is largely thermal are limited. Where the neutron spectrum does not vary greatly, albedo dosimeters (which measure low energy neutrons scattered back to the dosimeter from within the body) are simple and sensitive. But their response is highly energy dependent and they must therefore be calibrated appropriately.

It is only in exceptional accident exposure situations that attempts need to be made to estimate organ doses. The uncertainties acceptable in routine individual monitoring for external radiation should be somewhat less than the investigation level. The uncertainty in the measurements of the annual values of the shallow and deep equivalent dose indices should not exceed by a factor of 1.5 at the 95% confidence level if the quantities are of the order of the relevant annual limits, and by a factor of 2 when the amount is less than 10 mSv.

29.6.2 Special Monitoring for Accident Situations

Severe exposures are possible in the following cases:

(a) Operational error or equipment failures in reactors, fuel reprocessing plants, and during transfer of large amounts of activity between shields;

(b) Failure of interlocks in X-ray sets, accelerators and hot cells;

(c) Industrial radiography sources inadvertently left unshielded; and

(d) Criticality accidents in handling fissile materials.

Warning dosimeters are useful in many of these cases. They need not be very accurate, but should be reliable. For criticality accidents installed equipment provide a more reliable and forceful warning. For planning purposes 5×10^{18} fissions may be assumed in a criticality accident. Those working in areas where a criticality accident is possible should have special dosimeters capable of reading 10 Gy of gamma dose and providing information on the orientation of the individual in relation to the source. Further, use of special energy-dependent multicomponent dosimeters which give information on the neutron energy spectrum, either worn by the worker or installed at strategic locations in the workplace, is justifiable. Such dosimeters would be read only in the event of an accident. Supplementary measurements of sodium and chlorine activation in

the body, ^{32}P content of hair or of clothing containing wool, would provide useful information in accident situations. A simple indicator of neutron exposure such as an indium foil is useful not only for the group of workers mentioned above but also for those who work in nearby areas and who may receive some dose but well within the dose limits. Chromosome aberration analysis can be used for gamma doses above 0.1 Gy; it can unequivocally establish whether a substantial exposure has occurred.

In major accidents where the absorbed doses may be high enough to require medical attention, an early indication of these doses will be required. Personal dosimeters are not of much help in giving information about the magnitude and distribution of absorbed doses within the body, although they can provide points of reference. In extreme cases it may be necessary to reconstruct the radiation fields causing the exposure, in particular, by making measurements using a phantom.

29.7 MONITORING FOR SKIN CONTAMINATION

Skin contamination is never uniform and occurs preferentially on certain parts of the body, notably the hands. For routine purposes, monitoring results can be interpreted on the basis of the average dose over an area of 100 cm^2. In practice, the reading is compared with the derived or authorized limit. No attempt is routinely made to assess the dose if these limits are not exceeded. For persistent or high level of contamination some estimates (which are often very imprecise) may be necessary. Uncertainties of two orders of magnitude are not uncommon.

29.8 INDIVIDUAL MONITORING FOR INTERNAL CONTAMINATION

Individual internal contamination monitoring programmes are usually restricted by their cost in technical manpower and equipment, and, therefore, the selection of workers for such monitoring should be appropriate. Routine individual monitoring will almost always be needed in the following operations:

(a) Handling of large quantities of gaseous and volatile materials, e.g. tritium and its compounds in large-scale production processes, in heavy water reactors, and in luminizing;

(b) Natural and enriched uranium processing and reactor fuel fabrication in conditions of frequent contamination of workplaces;

(c) Processing of plutonium and other transuranics;

(d) Uranium milling and refining; and

(e) Production of large quantities of radionuclides.

The results of air monitoring and past experience of similar operations can also be used to assess the need for individual monitoring.

The interpretation of monitoring results in terms of the derived or authorized limits (which are normally expressed in ALIs) is difficult, depending particularly upon the pattern of intake into the body as a function of time. It is necessary to establish a metabolic model to correlate the two.

Since routine individual monitoring is conducted at selected times, not necessarily related to known intakes, it is necessary to assume a pattern of intake for the metabolic model. It can be assumed that the level of contamination of the workplace, and hence the intake, has been uniform. Alternatively, it can be assumed that the intake took place at the midpoint of the interval between monitoring times. The model should then provide information on radionuclide content of organs and rates of excretion from the body, against which the results of the monitoring can be compared.

In routine monitoring at regular intervals, the investigation level can be defined as an intake corresponding to $0.3/n$ of the ALI, where n is the number of sampling periods in an year. The recording levels are one-third of the investigation levels. (It is not always possible to apply the methods suggested above.)

In routine individual monitoring for internal contamination, unlike the case of external radiation monitoring, uncertainties of around 50% are rarely possible because of many inherent errors due to lack of knowledge of several parameters and errors in measurement.

For special monitoring, considerably more information will usually be available about the time and manner of intake. Where available, individual metabolic data may be used in place of the standard data to estimate committed organ and effective doses. Final estimates should take

account of all the available monitoring data, both individual and of the workplace.

29.9 QUALITY ASSURANCE

Any monitoring programme should include, as an integral part, a quality assurance programme which will ensure that equipments function accurately and correctly (by periodic checks and calibrations), that procedures are correctly established and implemented, that analyses are correctly performed, that systematic errors do not arise and that random errors are minimized, that records are correctly and promptly maintained, that personnel are suitably trained and experienced, and that results of the monitoring programmes are related to national standards where appropriate.

CHAPTER 30

INDIVIDUAL MONITORING FOR INTAKES OF RADIONUCLIDES BY WORKERS: DESIGN AND INTERPRETATION

30.1 INTRODUCTION

ICRP publication 54, 'Individual monitoring for intakes of radionuclides by workers: Design and interpretation' (1987) is a report of a Task Group of Committee 4 and supersedes ICRP 10, 'Evaluation of doses to body tissues from internal contamination due to occupational exposure' (1968) and ICRP 10A, 'The assessment of internal contamination resulting from recurrent or prolonged intakes' (1971). The report gives guidance on the design of monitoring programmes, the calculation of derived reference levels and the interpretation of monitoring results. General guidance is given on the models used to provide a link between the measured quantities and the appropriate limits or reference levels. Specific values of derived reference levels are given for radionuclides for their potential importance in occupational exposure. They have been calculated for intake by inhalation only since this is the most likely route of intake. This report should be read in conjunction with ICRP 35, 'General principles of monitoring for radiation protection of workers' (1982).

Individual monitoring for internal exposure is normally based on the direct measurement of radionuclides in the whole body or specific organs, on measurement of radionuclides in excreta, or an estimate of intake obtained by the use of a personal air sampler. From the monitoring results, primary quantities (such as committed effective dose or committed dose equivalent to an organ), or secondary quantities for intake (like the annual limit on intake, ALI) are assessed. In many circumstances, intakes of radionuclides are small compared with ALIs and a simple comparison of measurement results with appropriate derived reference levels (DRL) may be adequate for the control of occupational exposure. The ALIs are generally set on the basis of the limit on the effective dose, or in cases where it is more restrictive, on the non-stochastic (deterministic) limit in any organ.

30.2 DERIVED LEVELS

The concepts of *reference*, *investigation*, and *recording levels* have been described in Chapter 29.

For routine monitoring, the investigation level is set in relation to the monitoring interval to which the measurement result refers, and is taken as three-tenths of that fraction of the ALI corresponding to the monitoring interval. Thus, for N monitoring intervals per year, the investigation level for intake of any radionuclide is

$$IL_R = (3/10).(ALI/N).$$

For special (and operational) monitoring the IL is taken as one-tenth of the ALI since it is unlikely that more than a few unusual events involving the same individual will occur in a year.

$$IL_S = (1/10).(ALI)$$

The recording levels are taken as one-third the corresponding investigation levels.

The biokinetic models used are based on ICRP 30.

30.3 METHODS OF INDIVIDUAL MONITORING

Individual monitoring of intakes may be achieved by body activity measurements, excreta monitoring, air sampling with personal air samplers, or any combination of these techniques. Area monitoring results can supplement the above techniques in some cases. Routine monitoring programmes usually involve only one type of measurement if adequate sensitivity can be achieved. Special monitoring may often involve a combination of techniques.

30.3.1 Body Activity Measurements

Direct measurement of body or organ content is quick and convenient. It is feasible only for those radionuclides emitting radiations that can escape from the body (gamma ray emitters, bremsstrahlung from energetic beta emitters). Detection of the soft X-ray emissions from some alpha emitters requires special instrumentation. Whole body monitors have one or more high efficiency detectors housed in a shielded, low background

environment. Thallium-activated sodium iodide detectors are commonly used in whole body monitors. Their energy resolution (to distinguish between different gamma emitters) is generally adequate. Germanium detectors provide excellent energy resolution. Whole body monitors can be adapted for measurement of activity in a region of the body like the thorax or the thyroid.

Gamma emitters like ^{131}I, ^{137}Cs or ^{60}Co can be detected with simple equipment like a single detector with local shielding at levels corresponding to very small fractions of the derived reference levels. Alpha emitters with long half-lives such as ^{226}Ra, thorium, uranium isotopes and the transuranics present difficulties in measurement by body counting.

Care has to be taken to remove surface contamination before body activity measurements.

Wound monitoring is easy for beta-gamma contaminants but somewhat difficult for alpha contamination because of the absorption, by intervening body tissue, of the soft X-rays following alpha decay.

30.3.2 Excreta Monitoring

Excreta monitoring may be the only measurement technique in some cases. Urine analysis is the most frequently used method, although fecal analysis may be required in some circumstances, such as to assess the clearance of class Y materials from the lungs. Fecal excretion has two components: systemic fecal excretion which represents removal of systemic material via the GI tract; and direct fecal excretion of the material passing unabsorbed through the GI tract. For specific investigations, nose blows, nasal smears and exhausted breath analysis may provide some information.

Care has to be taken to avoid adventitious contamination of urine samples. Usually 24-hour urine collection is preferred. Fecal samples are best collected over a period of several days to even out daily fluctuations in fecal excretion. Gamma emitters can be directly measured with scintillation or semiconductor detectors. Analysis of alpha emitters requires chemical separation followed by appropriate counting techniques.

Interpretation of activity excreted per unit time involves considerable uncertainty, especially for radionuclides with long retention times in the body.

A few radionuclides present particular difficulties in measurement. These include thorium, uranium, plutonium and the other transuranics. The ALIs are small, the radionuclides cannot be measured in the body at levels corresponding to intake of one ALI, and excretion data are difficult to interpret. Most of them emit low energy photons following alpha decay which are highly attenuated in the body, thus posing problems of detection efficiency and calibration. Multiwire proportional counters, dual phosphor detectors and high resolution germanium detectors have been developed for these applications (lung and total body counting). In these cases, considerable attention is placed on excreta measurement for routine monitoring.

Personal air samplers are also used in routine monitoring programmes for such situations. The result is used to estimate intakes from a knowledge of the breathing and sampling rates. The activity collected on the filters at the end of the exposure period can give early warning of any unusual event.

30.4 MONITORING PROGRAMMES

Guidance on the subject is given in ICRP 35. Unlike external radiation monitoring, uncertainties as small as 50% are rarely possible in monitoring for intakes. The frequency of measurement in a routine monitoring programme will depend on the biological behaviour of the radionuclide, the sensitivity of the measurement and the acceptable uncertainty in the estimate of intake. The frequency of the routine monitoring should be chosen to reduce the uncertainty arising from the unknown time of intake, and in the variations of retention and excretion between individuals and that assumed in the standard models. In the interpretation of the monitoring result, it is assumed that the intake occurs at the midpoint of the monitoring interval. A factor of three uncertainty due to the unknown time of intake in each monitoring period is acceptable; if there are several monitoring periods each year, the overall uncertainty in the estimate of annual intake will be less than a factor of three. The recording level is taken to be one-third the investigation level. Thus even in the event of a maximum underestimation in one monitoring interval, information relating to an intake above the investigation level will not be lost; it will at least be recorded.

On this basis, appropriate monitoring intervals are proposed in the appendix to ICRP 54 and the corresponding derived reference levels given for radionuclides with potential importance in occupational exposure. If intake occurs towards the end of the monitoring interval, overestimation

of the intake may exceed a factor of three because of early excretion due to short-term components of retention.

30.4.1 Use of Derived Reference Levels in Routine and Special Monitoring

The Lung Model outlined in Chapter 21 can be used to compute values of body or organ content or excretion rate as a function of time following unit intake of any radionuclide. These functions give the predicted values per unit intake of the various quantities that can be measured, i.e. activity in the body or in an organ, or activity excreted per unit time, at time t, after a unit intake. They are denoted by the generic function $m(t)$. The corresponding measured values following an unknown intake are denoted by M. As mentioned earlier, the investigation level for routine monitoring is:

$$IL_R = (3/10).(ALI/N) = (3/10).ALI.(T/365)$$

for N monitoring periods each of T days.

Similarly, the recording level for routine monitoring is:

$$RL_R = (1/10).ALI.(T/365).$$

The derived investigation level for routine monitoring is then:

$$DIL_R = IL_R\, m(T/2)$$

since intake is assumed to occur at the midpoint of the monitoring interval. Similarly, the derived recording level is:

$$DRL_R = RL_R m(T/2).$$

As an additional long-term check it may be useful to compare the individual monitoring results with the values that would result from a continuing intake at the rate of ALI/365 per day; such values are given in the appendix to ICRP 54 for some radionuclides.

In the case of special monitoring, the time of intake is known and measurements are likely to be made in the few days following intake. The appendix to ICRP 54 gives values of $m(t)$ for 1 to 7 days after intake. As in the case of routine monitoring, we have the following expressions for special monitoring:

$IL_S = (1/10).ALI; \quad RL_S = (1/30).ALI;$

$DIL_S = IL_S m(t); \quad DRL_S = RL_S m(t).$

Values of DIL_S are given in the appendix to ICRP 54 for an aerosol AMAD of 1 μm. The graphs given there enable calculations to be made for aerosol diameters different from 1 μm AMAD using the standard respiratory tract model.

30.5 IMPLEMENTATION

30.5.1 Routine Monitoring

If the chemical form is unknown, the most restrictive values appropriate for the elements must be used.

If the measured value of M is less than DRL_R, there is no need to assess intake and committed dose equivalent. The result may be treated as zero for recording; the measured values should, however, be retained as part of the individual's record. If the measured value exceeds the DRL_R but is less than DIL_R, it should be interpreted on the basis of standard models and the intake or dose should be recorded in the formal dose record. If M exceeds DIL_R, further investigation is required (repetition of measurement, use of additional monitoring techniques, review of working conditions and circumstances of exposure, review of the frequency of routine measurements).

30.5.2 Special Monitoring

Procedures similar to those outlined in the previous paragraph for routine monitoring can be followed for special and operational monitoring.

30.5.3 Mixture of Radionuclides

In the case of a mixture of radionuclides, those radionuclides which would make a significant contribution to the committed dose equivalent should be identified and monitoring programmes designed for assessing their intake. Investigation and recording levels for each of the significant radionuclides can be calculated.

When the composition of the mixture is well known and constant, a single radionuclide which can easily be measured and which has a well known metabolism can be chosen for routine monitoring, and the intakes of other radionuclides inferred therefrom. DIL and RL for this radionuclide can be developed on the basis of a reduced limit of intake, i.e. a value less than its ALI, such that the committed effective dose for the mixture does not exceed the stochastic limits or that the committed dose equivalent to individual organs does not exceed the non-stochastic limits.

In general, measurement of total alpha or beta activity in a biological sample is not recommended for assessing intakes, especially when intakes are likely to exceed the recording levels.

30.5.4 Physical and Chemical Characteristics of the Contaminant

It has already been pointed out that appropriate corrections have to be made when the aerosol AMAD is different from 1 μm. If no information on the biological behaviour or chemical form is available, the most restrictive clearance class relevant for the particular element should be assumed (i.e. the class that gives the lowest values of DIL), or efforts made to determine the appropriate clearance class.

30.5.5. Deviations from Standard Models

Where the monitoring results indicate an intake in the current year in excess of (3/10).ALI, consideration should be given to the use of models or parameters that represent the individual and the exposure conditions more closely than the standard models.

30.5.6 Routes of Entry other than Inhalation

Only inhalation has been considered in ICRP 54, as this is the most likely route of intake for occupational exposure. In the event of an accidental intake by ingestion, DILs for special monitoring programmes can be calculated from the models and appropriate metabolic data.

Intact skin provides an effective barrier against entry of most radioactive materials into the body (except for tritiated water). When skin is punctured, any contaminated wound must be subject to medical investigation and a programme of special monitoring should be instituted. Measurement of radioactive material at the site of the wound and a subsequent

series of measurements (by body monitoring or excreta analysis) should be undertaken to determine uptake in body tissues.

30.5.7 Effect of Medical Intervention

In cases of intakes exceeding the ALI, medical intervention to reduce uptake or enhance excretion may be considered. A special monitoring programme should be undertaken to determine the retention and the data obtained therefrom used to estimate the committed dose equivalents.

30.6 MONITORING DATA FOR INDIVIDUAL RADIONUCLIDES

In the appendix to ICRP 54 report data are given for 37 radionuclides of 22 elements which have been selected for their potential importance in occupational exposure within the nuclear industry, in research and in medical procedures.

Metabolic data relevant to all isotopes are given for each element of interest. These include: f_1 values, inhalation class, distribution and retention in body tissues and organs (data taken from ICRP 30), and excretion functions.

The section on general metabolic data is followed by detailed calculations for each important radioisotope. The following information is given: Physical half-life; mode of decay, and radioactive emissions that are useful for individual monitoring; methods of individual monitoring with typical detection limits; dosimetric data for inhalation of a 1 μm AMAD aerosol [inhalation class, committed effective dose per unit intake in Sv/Bq, committed dose equivalent to the most exposed organ per unit intake in Sv/Bq; whether the ALI is determined by stochastic (S) or non-stochastic (NS) limits]; graphs of measured parameters (whole body/organ activity, or urinary/fecal sample activity) as a function of time after an acute intake for periods up to 10^4 days for aerosols of 0.2, 1 and 10 μm AMAD; numerical data for both routine and special monitoring programmes for each appropriate measurement technique.

For routine monitoring the data are in the following form: Monitoring interval, T, in days; predicted activity as a fraction of intake, $m_1(T/2)$; and DIL for each measurement technique.

If the predicted activity as a fraction of intake is less than one-third the DIL, (i.e. less than the recording level), the intake can be assumed to

be less than (1/10).ALI.*(T*/365*)*. If the result exceeds the derived recording level, it is necessary to estimate the intake in the monitoring interval, by dividing the measured result by the appropriate value of $m(T/2)$. The committed effective dose and the committed dose equivalent to the most exposed organ can then be calculated for this intake. If the measurement result exceeds the DIL, an investigation should be made into the circumstances of the exposure.

For special monitoring the data are given in the following form: Time after intake, t (days); predicted activity as a fraction of intake, $m_f(t)$, and the DIL for each measurement technique. Values are given for each day up to 7 days after intake.

A procedure similar to the one outlined for interpretation of the routine monitoring results can be adopted here also.

Information is also given on maximum achievable levels in the body, in organs, or in excreta, for the hypothetical case of chronic exposure at (1/365).ALI per day for 50 years.

For radionuclides with long biological half-lives and for which detection limits are high compared with the reference levels, graphs are given to show the increase with time of the activity in the body, in organs, or in excreta. These illustrate the difficulty of estimating small increments on existing levels due to previous intakes.

A result in excess of the DIL or any unusual result will often lead to the collection of another sample a few days after the first. If the activity of the second sample is very much less than that of the first, this suggests that either the first sample was contaminated with unmetabolized material or that it was collected shortly after intake.

Metabolic and dosimetric data, and ALI values of the radionuclides considered in ICRP 54 as well as several other important radionuclides have been given earlier in Tables 25.2, 25.3 and 25.5 of Chapter 25.

CHAPTER 31

PRINCIPLES AND GENERAL PROCEDURES FOR HANDLING EMERGENCY AND ACCIDENTAL EXPOSURES OF WORKERS

31.1 INTRODUCTION

When a source of exposure is or has been out of control, workers may receive doses beyond the dose limits. Such uncontrolled exposures are called abnormal exposures and they are either emergency (voluntary) or accidental (involuntary). ICRP 28, 'Principles and general procedures for handling emergency and accidental exposures of workers' (1978) is a general guide for the management of the worker under abnormal exposure situations, and is concerned with the administrative, physical and medical aspects of the planning of actions which should be followed in the above situations, and with the initial actions which should be taken following such exposures. It does not deal with the subsequent medical care of workers who incur abnormal exposures nor with the actions to be implemented to bring the source of exposure under control. (Planned special exposures are outside the scope of ICRP 28.)

31.2 RESPONSIBILITIES OF MANAGEMENT

(a) Assessing the likelihood of abnormal exposures and making plans for dealing with them, including provision of necessary services and allocation of responsibilities;

(b) Initial testing of effectiveness of emergency plans and their subsequent maintenance through appropriate exercises;

(c) Arranging for a radiation protection team which will review in advance all abnormal situations that may occur (The team will include the physician responsible for medical supervision of the workers).

31.3 INDICATIONS FOR ACTION

In the event of a suspected abnormal exposure (which may not always be recognized promptly), investigations should be undertaken as soon as possible to establish the sequence of events which gave rise to the abnormal exposure and the levels of the doses incurred by the exposed workers. Sources of relevant information are: clinical course of exposed workers, biological and biochemical studies, physical dosimetry, and statements made by persons actually or potentially exposed. Any unexpected readings in installed monitors or personal dosimeters should be immediately investigated. Action should be taken to prevent the recurrence of the same type of situation in future.

31.4 PHYSICAL, CLINICAL AND BIOLOGICAL DATA FOR ASSESSING SEVERITY OF EXPOSURE

In preliminary screening of workers who may have incurred an abnormal exposure, two broad categories should be considered, viz. external exposure and internal contamination; both of these may be associated with external contamination.

31.4.1 Information from Workers

Information from the workers must be collected carefully and quickly, supplemented by an assessment of the location of the workers in relation to the source, the train of events which led to the situation, and the duration of the exposure. It may be best to assume that all workers in the relevant area have incurred abnormal exposures and to rely on other methods to make positive identification at a later stage.

31.4.2 External Contamination

External contamination may modify the management procedures for external and internal exposures. Suspected persons should be subjected to external monitoring of body surfaces and of clothing. In the event of a suspected significant internal contamination, smear samples of the worker and his clothing should be monitored.

The control of external contamination of the worker should normally be secondary to the earlier phases of examination and investigation, unless (i) the contamination level is high enough to pose a hazard to the affected

worker, or (ii) there is a chance that he may spread contamination significantly.

31.4.3 External Exposure

Personal and area dosimeters should normally give an indication of the doses received (except for exposures from narrow beams or limited fields of irradiation). The data from personal dosimeters should be interpreted with care. Attempts should be made to get information on the radiation fields, the worker's orientation to the fields, and the mean quality factor of the radiation. If the preliminary assessment indicates a high level of exposure, more detailed investigations should be taken up urgently. Any indication of grossly non-uniform irradiation would be valuable.

Neutron exposures would produce detectable induced radioactivity in the worker, in objects worn or carried by him, and in materials in the area. Induced activities in hair, finger nails, woollen clothing, or in metallic objects would give some idea of the level of exposure. A more reliable estimate can be obtained from the 37 min ^{38}Cl and 15 h ^{24}Na induced activities in blood. In case the personal dosimeter includes an indium foil, the induced activity (half-life 54 min) can be roughly checked immediately by a portable monitor, followed quickly by a more sophisticated assay.

31.4.4 Internal Contamination

Positive monitoring results of air and nearby surfaces may, *prima facie*, warrant a preliminary assumption of internal contamination of all workers in the area. Measurements on the workers with wound probes, external probes, gamma cameras, scanners, or whole body monitors may give positive findings. However, results may be equivocal in the event of external contamination or neutron exposure.

Swabs of nasal passage and of the mouth may be immediately checked. In the event of a positive finding, this should be followed by simple assays of samples of urine, feces, and, in some cases, blood. If necessary, radiochemical assays of the urine or feces may be done subsequently; this procedure is time consuming. The frequency of subsequent sampling should be based on the results of the analysis obtained in the first few days. Early information following an abnormal internal exposure will be

limited, in practice, to a preliminary appreciation of the nature of the exposure.

31.4.5 Clinical Observations and Biological Investigations for External Exposure

Since reliable dosimetric data may not always be available, early assessments of the severity of the exposure must, in most cases, be largely based on clinical signs and symptoms, haematological data and chromosomal analysis. A rough appraisal of the prognosis should be given to the patient and his family as early as possible.

31.4.5.1 Clinical Observations

Initial symptoms may serve as a reasonable guide for the assessment of the severity of exposure. Around the LD_{50} range, the main symptoms are anorexia, nausea and vomiting. The shorter the interval between exposure and the onset of nausea and vomiting, the more severe these symptoms and the longer their continuance, the more serious the prognosis. (Psychosomatic symptoms may complicate the picture.) Early neurological symptoms such as apathy, ataxia or convulsions indicate a very high dose.

Early erythema and conjunctivitis are useful indicators of the spatial distribution of the dose and its magnitude. The higher the dose, the shorter the latent period for erythema; the skin should therefore be frequently examined. Colour photographs of the development of skin changes should be obtained over a period.

(Author's note: The discussion of effects on the skin given in ICRP 28 is not summarized here; reference may be made to Chapter 10 which deals in detail with deterministic effects in various organs including the skin, and Chapter 11, Section 4 which deals specially with effects on skin).

For very high doses, a neurological examination (and an EEG) should be arranged. For possible high doses to the thorax, a cardiovascular examination and an ECG may be indicated.

31.4.5.2 Haematological, Biological and Biochemical Investigations

Haematological data are of importance in assessing the severity of exposure; granulocyte and lymphocyte counts are the most useful for an early assessment.

The granulocyte count increases sharply in the first day, falling back to the previous value in 1-2 days, and thereafter drops progressively. To observe the initial peak, the amplitude of which is roughly related to the dose, blood counts should be made several times during the first 2 days. The lymphocyte count decreases rapidly after exposure and reaches a minimum in 2-3 days. The sharper the slope of the fall and the lower the value of the minimum, the more severe the exposure is.

Blood samples should be taken within a few hours of the accident for chromosomal analysis, as this investigation would give a quick assessment of the whole body dose. Blood and urine samples taken immediately after the event will often provide a useful baseline for subsequent comparisons. Blood examinations should include standard cell counts and may include biochemical assays for plasma electrolytes and blood sugar. Urine samples may be assessed for sugar, creatinine, and aminoacids. Radiochemical assays of urine for excreted radionuclides should be done if there is a suspicion of internal contamination.

Sperm counts showing a diminution of spermatozoa some days after exposure would indicate a gonadal dose of several Gy. Bone marrow examination (which must be carried out in a specialized hospital department) may also be useful, especially for evaluating partial irradiation at high doses.

The symptoms and clinical signs of radiation sickness are summarized below:

Prodromal Manifestations

Anorexia	Apathy	Fever
Nausea	Prostration	Respiratory distress
Vomiting	Perspiration	Hyperexcitability
Diarrhoea	Erythema	Ataxia
Fatigue	Conjunctivitis	

Latent Period

No symptoms

Main Illness

Fever	Infection	Shock
Anorexia	Haemorrhage	Ataxia
Lassitude	Erythema	Agitation
Fatigue	Tanning	Disorientation
Weakness	Epilation	Convulsions
Weight loss	Aspermia	Coma
Diarrhoea	Ileus	

The frequency of incidence of the prodromal reaction among the exposed persons increases with time and reaches a maximum around 6 h; it occurs in 25% of the exposed persons at 1.5 Gy, in 65% at 2 Gy, and in over 90% at 3 Gy (Upton, 1969).

The acute radiation syndrome can be classified into 3 forms, depending on the level of the dose: haematopoietic, gastrointestinal, and central nervous system. Table 31.1 summarizes the symptoms, therapy and prognosis of radiation injuries following acute whole body irradiation.

31.5 INDICATIONS FOR ACTION

31.5.1 External Exposure

(a) *Estimated Exposure < 0.1 Gy:* Action essentially administrative: Investigation into circumstances of abnormal exposure and confirmatory physical measurements;

(b) *Estimated Exposure 0.1 to 0.25 Gy:* More detailed administrative enquiry; Assessment of possible biological consequences; Physician to advise on nature of investigations and management; and

(c) *Estimated Exposure > 0.25 Gy:* Supplement actions in (b) with a medical examination of worker; Reconstruction of circumstances leading to the abnormal exposure, if necessary.

The higher the dose, the more important becomes the need for accurate dosimetry by a combination of clinical, biological, biochemical and physical assessments. If clinical signs and symptoms become apparent, these may be a more important guide to initial treatment than the early estimate of dose.

31.5.2 Internal Exposure

If the internal contamination is suspected to be greater than the ALI, the best possible evaluation of the intake should be done (by the techniques indicated earlier), and therapy should be considered. In most cases the initial indications for therapy are almost always qualitative. For therapy to enhance excretion of radioactive material taken in, efficacy is greatest if therapy is applied early. Early assessments of intakes may have to be followed up by subsequent investigations to provide further indications for therapy.

31.6 PRINCIPLES OF URGENT MEDICAL CARE

Initially, the management of any severe injuries (e.g., trauma and burns) is to be given priority over radiation injuries. The following subsections summarize the action plans.

31.6.1 Whole Body Exposures

(a) *0-6 hours*

Life-saving treatment of associated injury;
Mild sedation;
Check for external contamination; decontaminate if required;
Collect dosimetric information;
Blood, urine analysis;
Make provisional prognosis;
Start clinical observations if suspected dose exceeds 0.25 Gy.

(b) *6-71 hours*

Suspected dose < 1 Gy

Mild sedation;
Ambulatory medical surveillance; hospitalization required only after a few days, if necessary;

Suspected dose > 1 Gy

Give symptomatic treatment and sedation;
Transfer to a specialized hospital;
Make complete clinical examination with particular attention to erythema, neurological signs, nausea and vomiting;

Take ECG, EEG;
Carry out periodic blood analysis (thrice a day in first 24 hours and daily later) and daily urine analysis;
Reassess doses;
Reconstruct accident if warranted;
Reassess prognosis.

(c) *After 72 hours*

Ambulatory medical surveillance for a week or more;
Discharge admitted patient from hospital when appropriate;
Arrange follow-up medical surveillance;
Reassess dose, evaluating information from various inputs;
Continue blood/urine analysis as appropriate;
Reassess prognosis;
Decide on future employment of worker;
Consider medico-legal aspects.

31.6.2 Partial Body Exposures

No specific treatment for affected area;
Injury may be more serious than initial assessment may suggest;
Damage may progress for months;
Prognosis should be guarded;
Avoid infection of exposed areas;
If infection occurs, start local and systemic therapy.

31.6.3 External Contamination

(a) *General*

Immediate first aid;
Check for nature and degree of contamination;
Proceed without delay with external decontamination and emergency therapy for internal contamination;
Decontaminate as effectively as possible before removal to hospital.

(b) *Skin Contamination*

Wash with soap and water (worker himself can often do this);
Stop decontamination procedures before appearance of skin abrasion;
Decontaminate eyes, nose and mouth with water or isotonic irrigants;
Refer persistent contamination to physician.

(c) *Contaminated Wounds*

Wash with water;

To avoid spread of contamination from skin to wound, isolate wound with barriers or drapes;

Promote bleeding;

Repeatedly irrigate wound and monitor with special wound probe;

For actinide/lanthanide contamination, treat wound with DTPA; supplement with intravenous DTPA to minimize deposition in organs of material which enters circulation during surgery;

Assay tissue removed during debridement.

31.6.4 Internal Contamination

Attempt enhanced excretion of radionuclide;

For radioiodine contamination, administer stable iodine as iodide or iodate to block thyroid;

Use irritants and expectorants to minimize respiratory absorption; administer DTPA as aerosol mist for lanthanide/actinide contamination; (efficacy and safety of pulmonary lavage technique still under investigation);

To minimize GI absorption, accelerate excretion with a laxative like magnesium sulphate;

Administer Prussian Blue or sodium alginate for cesium and strontium elimination respectively;

Use chelating agents like DTPA to achieve blood clearance and reduce bone absorption, particularly for lanthanides/actinides;

For soluble uranium compounds, give alkaline diet, administer suitable diuretics, infuse potassium carbonate.

31.7 ADMINISTRATIVE ACTION FOLLOWING AN ABNORMAL EXPOSURE

The decision whether or not to remove a person from radiation work should take into account his social, occupational and economic responsibilities, and the physician should have considerable latitude in making his recommendation.

Abnormal exposures are rare and the possibility of further abnormal exposure is not usually a major factor in deciding the worker's future employment (unless by his own actions he contributed to his accidental exposure).

If the exposed worker shows clinically observable injuries his duties may have to be appropriately modified. (A local radiation injury to superficial tissue does not necessarily preclude future employment.) A critical review of the circumstances leading to the abnormal exposure should be made to reduce the likelihood of a similar situation in future.

The physician should explain to the worker any limitations which are advisable (particularly for abnormal exposure of male gonads or of pregnant women). All records of health physics measurements, medical histories and medical follow-up of workers should be kept. Periodic medical checkups are important even for those workers who showed no signs and symptoms immediately after an abnormal exposure.

31.8 ORGANIZATION OF MEDICAL SERVICES IN ANTICIPATION OF ABNORMAL EXPOSURES

Because of the variety of possible situations, it is not practicable to provide a detailed general plan. For each installation, planning should be based on an evaluation of the risks of different types of abnormal exposure which exist therein. As a general principle, planning should provide for three functional levels: immediate first aid to deal with all non-radiation injuries; treatment by 'local' medical services; and treatment at a specialized facility.

31.8.1 First Aid at Site

Specified workers should be trained in appropriate first aid, should be given detailed directives, and should be readily available. First aid equipment should be frequently inspected. Lines of communication should exist between the first aid services and the radiation protection services.

31.8.2 Treatment in 'Local' Medical Services

These services may be either within the installation or through advance liaison with a nearby hospital. Appropriate facilities and staff as well as advice of the radiation protection services should be readily available. A well-defined plan of action should be prepared by the physician responsible for the medical care of the workers for each potential abnormal exposure situation, including criteria for transfer of the worker to the 'local' medical service. Arrangements should be made for maintaining comprehensive medical histories and follow-up records.

31.8.3 Treatment at a Specialized Facility

A specialized medical facility to take care of serious abnormal exposures should have the resources of a major medical centre together with the specialized personnel and facilities. With the help of rapid transport, one such facility could serve a large area.

Arrangements should be made in advance to facilitate the transfer of an exposed worker to such a facility (including transport, criteria for transferring to the specialized facility, and liaison between the physician responsible for the medical care of the workers in the establishment and the specialized facility).

REFERENCES

Upton, A.C. (1969). *Radiation Injury: Effects, Principles and Perspectives*. Chicago.

UNSCEAR (1967). United Nations Scientific Committee on the Effects of Atomic Radiation, United Nations, New York.

Table - 31.1: Summary of symptoms, therapy and prognosis of radiation injuries in man following acute whole body irradiation

(from ICRP 28, based on UNSCEAR, 1967)

	1-10 Gy Therapeutic range (range where therapy may be effective)				> 10 Gy Lethal range	
Range	0-1 Gy	1-2 Gy	2-6 Gy	6-10 Gy	10-15 Gy	> 50 Gy
Therapeutic needs + possibilities	None needed	Clinical observation	Effective therapy	Possible therapy	Palliative therapy	
Appearance of vomiting	No	1 Gy: 5% 2 Gy: 50%	3 Gy:100%	100%	100%	100%
Time of delay of nausea + vomiting	-	3 h	2 h	1 h	30min	30 min
Main organ	No		Blood-forming tissue		Gastrointestinal tract	Central nervous system
Characteristic symptoms	-	Moderate leucopenia	Heavy leucopenia,purpura, haemorrhage,infection,epilation, > 3 Gy		Diarrhoea, fever, electrolytic imbalance	Cramps, tremor, ataxy, lethargy
Critical period after exposure	-	-	4-6 weeks		5-14 days	1-48 h

(contd...)

Range	1-10 Gy Therapeutic range (range where therapy may be effective) 0-1 Gy	1-2 Gy	2-6 Gy	6-10 Gy	> 10 Gy Lethal range 10-15 Gy	> 50 Gy
Therapy	Psycho-therapy	Psychotherapy, haematological observation	Transfusion of blood; antibiotics	Transplantation of bone marrow is possible and white cells or platelets	Support of electrolytic balance	Symptomatic
Prognosis	Excellent	Excellent	Guarded	Guarded	Poor	Hopeless
Time of recovery	-	Several weeks	6-8 weeks 1-12 months	Prolonged	-	-
Lethality	0	0	0-80%	80-100%	90-100%	100%
Time of death	-	-	2 months	2 months	2 weeks	2 days
Cause of death	-	-	Haemorrhage - infection		Enterocolitis	Irreversible circulatory collapse, cerebral oedema

CHAPTER 32

PRINCIPLES OF MONITORING FOR THE RADIATION PROTECTION OF THE POPULATION

32.1 INTRODUCTION

ICRP publication 43, 'Principles of monitoring for the radiation protection of the population' (1984) is a report of Committee 4 of ICRP. It is a revision of ICRP publication 7, 'Principles of environmental monitoring related to the handling of radioactive materials' (1966), intended to make the recommendations consistent with the current radiation protection philosophy and to extend the scope to all types of monitoring outside the workplace. The emphasis is on the normal monitoring programme. (ICRP publication 40, 'Protection of the public in the event of major accidents: Principles for planning' (1984) deals with emergency situations.) ICRP 43 supplements ICRP publication 35, 'General principles of monitoring for radiation protection of workers' (1982).

Some sources of exposure arise at clearly defined locations, in which case they can be monitored specifically; others are widely dispersed, and render direct monitoring more difficult. In either case, the need for environmental monitoring as well as source monitoring should be assessed, and the appropriate monitoring programmes developed. Doses may sometimes be assessed solely by measurement, but more usually a combination of monitoring and modelling is required.

Monitoring of the source is concerned with the measurement and assessment of dose rates in air and of quantities of radionuclides released into the environment from the source. The monitoring usually takes place close to the point beyond which there is no possibility of controlling releases of radioactive materials or preventing access to radiation fields. Environmental monitoring is concerned with measurement of dose rates in areas beyond this point and with measurement of radionuclide concentrations in selected environmental media. Environmental monitoring is not necessarily directed at a single source but can be used to assess the result of a number of sources on a local, regional or global scale.

The type and amount of both kinds of monitoring will vary with the condition of operation of the installation or practice: pre-operational, plant commissioning, operational, decommissioning, and post-operational, during normal operations and emergencies. At each phase the monitoring programmes should be designed to fulfill specific objectives (as discussed later), and should be reviewed from time to time. There will be many situations in which a pre-operational study will show that no monitoring is required or that a very simple programme will suffice.

32.2 EXPLANATION OF TERMS

Monitoring is the measurement of radiation or activity for reasons related to the estimation or control of exposure to radiation or radioactive material; it includes interpretation of the measurements. A *monitoring programme* must specify the type and frequency of measurements, and procedures for sampling, measurement, analysis, data handling, interpretation and recording. The network of sampling stations must be carefully chosen with regard to the source and pathways of exposure. The final part of the programme may involve computations of doses to individuals or populations, or it may merely require comparison of measurements with appropriate derived authorized limits.

Source monitoring will normally be the responsibility of the operator of the installation and measurements will usually be made at the point beyond which the operator can no longer affect the behaviour of radionuclides released or prevent access to the radiation fields, i.e., in a discharge stack or at the site boundary fence.

Environmental monitoring is conducted outside the site. It may be *source related*, in order to demonstrate compliance with authorized limits, to measure deviations from expected levels, and for public information, and may also be of use in optimization studies. It will often be the responsibility of the operator of the source, but may be supplemented by regulatory or other agencies. *Person-related* monitoring is environmental monitoring in circumstances where there may be several sources irradiating the same group of people. It will usually be carried out by the appropriate national authorities, its main objective being to assess doses from all sources. Individual monitoring of members of the population is rarely made under normal circumstances, but may be of use following an accident.

Monitoring programmes may be for normal and emergency situations. The normal programme will not only demonstrate compliance with appro-

priate dose limits but also provide baseline values for unusual occurrences or emergency situations. Monitoring will be done during various phases of operation: pre-operational (to gain experience and to determine baseline values), commissioning, operational, decommissioning, and post-operational. The frequency of the monitoring may be reduced as experience is gained in the early part of the operational phase. During the operational phase monitoring must be adequate to demonstrate compliance with authorized limits. The decommissioning phase may require a more intensive or different programme. The length for which the monitoring should be continued in the post-operational phase depends on individual circumstances. Emergency monitoring must be able to provide information quickly to aid decisions on emergency management.

32.3 MODELLING AND MONITORING

The assessment of individual or collective doses will involve the use of both models and measurements. At one extreme, no measurements may need to be made; for example, doses from external irradiation may be calculated from the knowledge of the source characteristics. At the other extreme, it may be necessary to measure individual doses and body contents directly, and no environmental transfer models are required; but, for practical reasons, such measurements are infrequently done. Doses from intakes of radionuclides can be assessed by measurement of the radionuclide content of air and foodstuffs, combined with a knowledge of inhalation and consumption. Furthest removed from man is the measurement of radionuclides or dose rates at the source, or at the point of release, or in the early stages of the environmental transfer chains; models are required here to predict the dilution and dispersion of the radionuclides, transfer through environmental pathways and the resultant dose to man. Especially for environmental releases, a combination of the two is required.

32.3.1 The Use of Models

A derived limit (e.g. dose rate, rate of discharge of a radionuclide, or concentration of a radionuclide in an environmental medium) can be used to relate the measurement to the relevant dose limit by suitable models. A measurement showing a value below the derived limit indicates virtual certainty of compliance with the primary dose limit. A higher reading will not necessarily indicate that the dose limit has been exceeded, but will indicate the need for a careful review.

32.3.2 External Exposure Models

Calculation of individual external doses from dose rate measurements made near the source will involve 2- or 3-dimensional models of varying complexity to relate the two, taking into account intervening shielding and other factors.

32.3.3 Environmental Transfer Models

Simplified pathways between release to air or water and man have been shown in Figs. 27.1 and 27.2. ICRP publication 29, 'Radionuclide releases into the environment: Assessment of doses to man' (1979) (summarized in Chapter 27) gives detailed descriptions of environmental models. Especially where the dose from a particular pathway is small and monitoring would not be economically justified, dose assessments based on models are useful. The simplest model that will give a result of the required accuracy should be used.

32.3.4 Metabolic and Dosimetric Models

ICRP publication 23, 'Reference Man: Anatomical, physiological and metabolic characteristics' (1976) and ICRP publication 30, 'Limits for intakes of radionuclides by workers, Part 1' (1979) give metabolic and dosimetric models for calculation of doses and ALIs. (These are summarized in Chapters 17 to 24). In calculating doses to members of the public it is necessary to take account of varying factors: difference between workers and members of the population which may comprise various age groups; variations in chemical/physical form of the radionuclide (since the population intake is from radionuclides incorporated in food where gastrointestinal absorption may be more than for the chemical compounds found in the workplace); variation between the individuals comprising the critical group and the Reference Man.

In the assessment of committed doses to critical groups, maximizing assumptions are appropriate to ensure compliance with dose limits, whereas for collective doses in optimization studies, realistic assumptions should be used.

32.3.5 Interaction between Monitoring and Modelling

Both the model and the monitoring programme must be subject to review in the light of experience. Techniques such as sensitivity analysis

should be used to identify those factors that account for the greatest uncertainties.

32.4 GENERAL OBJECTIVES OF MONITORING

The major objectives are: to assess actual or potential doses to critical groups; to demonstrate compliance with authorized limits and legal requirements; to check the condition of the source and to provide early warning of an unusual condition. The subsidiary objectives are: to inform the public; to identify changes in the relative importance of transfer pathways in order to refine the monitoring programme; to survey trends in exposure; to conduct scientific studies on transfer mechanisms.

32.5 THE REQUIREMENTS FOR MONITORING PROGRAMMES

Many sources may require no monitoring programmes, some may require routine source monitoring but only occasional checks on environmental levels, and some may require comprehensive source and environmental monitoring with emergency monitoring capability. The pre-operational phase review will indicate the frequency and type of monitoring required.

A source expected to give a dose to the most exposed individual of the order of 1 μSv could be regarded as minor, requiring no monitoring. Examples are sealed sources in many industrial applications, small amounts of unsealed sources used in research or teaching, and consumer products. For sources of an intermediate nature, it will often be sufficient to sample the discharges or make occasional measurements of dose rates.

Installations associated with the nuclear fuel cycle clearly require both source and environmental monitoring programmes. Routes of discharges will have to be sampled frequently to provide information on quantities of each important radionuclide discharged, chemical form, particle size, etc. Meteorological and hydrological data will have to be collected regularly. Periodic monitoring may suffice if the potential for rapid variations in external dose rate or discharge rate or radionuclide composition is small. Accident potential should always be kept in mind; the radionuclide composition and physico-chemical characteristics are likely to be different in normal and accident situations. Airborne effluents are normally discharged continuously, although the operation itself may be discontinuous, while liquid effluents may be discharged continuously or

stored and subsequently discharged on a batch basis. For each type of source and for each route of potential exposure, it is necessary to consider whether continuous monitoring is necessary, and to fix the monitoring locations.

Exposure of the public can result from the widespread distribution of radionuclides in various forms. Some of them can be monitored at source or before use, e.g. building materials, natural gas, mineral waters, consumer products.

32.6 MONITORING OF THE ENVIRONMENT

The requirement of periodic review is more important for environmental monitoring programmes than for source monitoring programmes. Source-related environmental monitoring programmes are oriented mainly towards assessment of average doses to critical groups.

For *minor sources* for which there is no requirement of source monitoring, environmental monitoring will also not be needed. A source such as a research reactor can be minor as regards normal operation but can have a significant potential to cause higher doses in accident situations. For these sources, it will be necessary to have an emergency plan incorporating provision for environmental monitoring. For transport of radioactive materials, the most appropriate would be a well practised emergency plan. For fixed sources, a continuous source monitoring programme, along with some preliminary environmental monitoring during the pre-operational phase to determine baseline values, would suffice.

Continuous monitoring of external dose rates and continuous sampling of airborne and water-borne radionuclides is recommended for major installations; somewhat infrequent sampling of other environmental materials has to be carried out periodically.

For all except minor sources, there will be need for a prepared emergency environmental programme which will be triggered in the event of an accident.

For *major sources*, the key factor in determining environmental monitoring programmes is identification of the potentially critical nuclides, pathways, and groups. A diagram like Fig. 32.1 may be useful. Usually the critical group would be a few tens of persons. The results of a habit survey at a particular point in time should be regarded as an indicator of

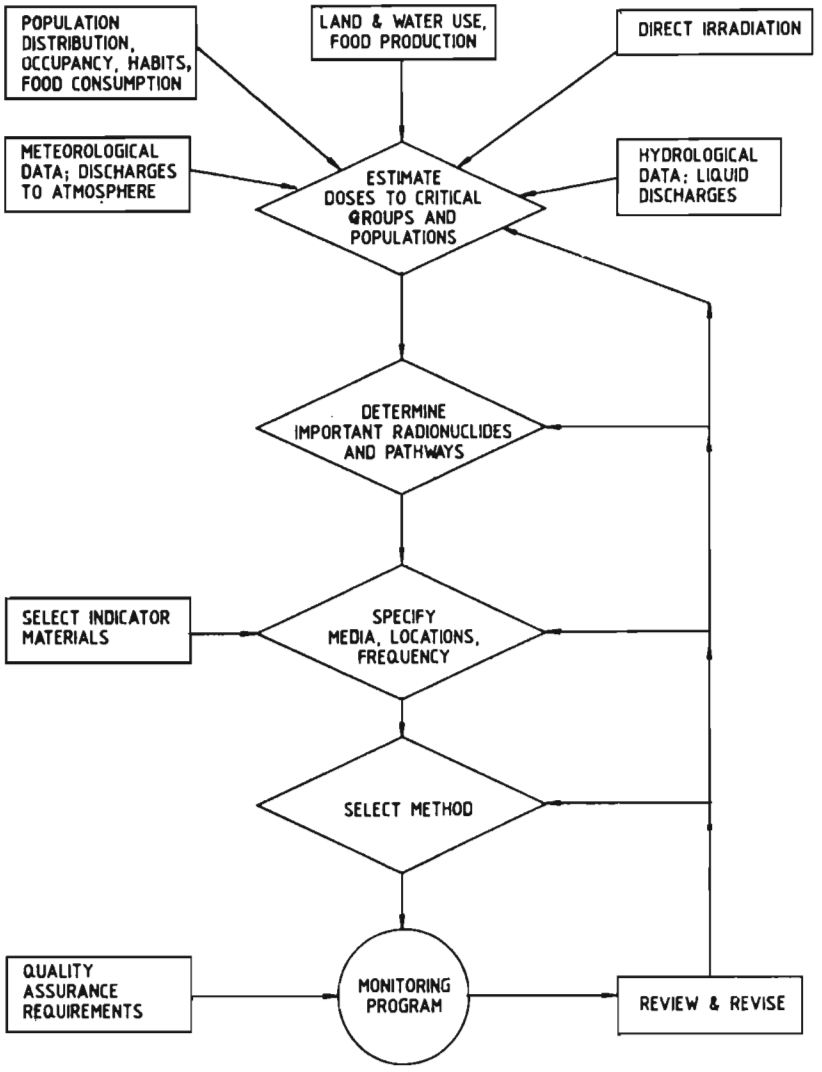

Figure 32.1. Typical design (flow chart) of an environmental monitoring programme for a major source (from ICRP 43).

an underlying distribution. Appropriate mean values should be chosen for factors such as food consumption or occupancy parameters. However, metabolic parameters should be chosen to be typical of the age group (e.g. fetus, infant, child, adult).

Some individuals may receive doses in excess of the calculated mean dose. If the mean dose to the group is less than about one-tenth the source upper bound, a critical group should be regarded as homogeneous if the distribution of individual doses lies within a factor of 3 on each side of the mean. At higher fractions, the total range of the distribution of doses should preferably be not more than 3 for a critical group to be considered homogeneous.

32.6.1 Person-related Environmental Monitoring Programmes

Although many sources of radionuclide release or external dose rates are localized and environmental monitoring programmes can be focused on them, there are also widespread or diffuse sources which cannot be treated in this way. Person-related environmental programmes in such cases are characterized by wide geographic coverage.

32.6.2 Design of Environmental Monitoring Programmes

The programmes should be directed to those nuclides and pathways indicated by the pre-operational studies to be significant. Each situation is unique as regards topography, meteorology, demography, etc. The location of the sampling points, frequency of sampling, whether to assess maximum or mean exposures to local communities, etc. should be decided by good professional judgement.

In addition to measurements on direct pathways to humans, consideration should be given to measurements on 'indicator' materials such as sea weeds that are not part of the food chain but which concentrate radionuclides and are therefore easier to monitor, and provide a measure of trends in activity levels.

32.7 MONITORING OF INDIVIDUALS IN THE POPULATION

Under normal conditions individual monitoring of members of the population will rarely be appropriate. It may be done in the rare situations when particular individuals are exposed at a level close to the limit. Following a severe accident, monitoring of selected individuals is likely to be the most reliable means of assessing internal doses. Even if the doses are expected to be small, some limited individual monitoring can be useful for public reassurance.

32.8 QUALITY ASSURANCE

Monitoring programmes should include, as an integral part, a quality assurance programme. The QA programme should include sampling procedures, calibration, maintenance and performance of instruments, verification of analytical procedures, training and experience of personnel, traceability of the results of the monitoring programme to a national standard, data interpretation, and type of documentation.

The objective of each part of the programme must be clearly defined. Records should demonstrate the adequacy of the programme, demonstrate that authorized limits have not been exceeded, and help to evaluate the impact of a source or practice. The records should contain the rationale for the choice of the programme, and details of calculations. The information must be periodically reviewed and updated. The records should be kept for a long period (some decades from the cessation of operation) and as specified by national regulations.

CHAPTER 33

PROTECTION OF THE PUBLIC IN THE EVENT OF MAJOR RADIATION ACCIDENTS: PRINCIPLES FOR PLANNING

33.1 INTRODUCTION

ICRP publication 40, 'Protection of the public in the event of major radiation accidents: Principles for planning' (1984) set out general principles for planning intervention after an accident, particularly from major installations (specifically nuclear power plants), where the potential exists for releases of such magnitude that off-site emergency plans are required.

ICRP 60 has given general recommendations on intervention following accidents and emergencies. Any protective action interferes to some extent with normal conditions and may itself involve some risk. The benefits include: averted individual/collective doses, reassurance to individuals, and recovery of land for economic use, while the disadvantages include: physical risk, dose to workers involved in the protection action, financial cost, individual inconvenience, social disruption, and anxiety. The countermeasures forming a programme of intervention should be justified in the sense that they should do more good than harm. Their form, scale and duration should then be optimized so as to maximize the net benefit. ICRP recommends against the application of dose limits for deciding on the need for, or scope of, intervention. Nevertheless, at some level of dose, approaching that which would cause serious deterministic effects, some kind of intervention will become mandatory.

ICRP publication 63, 'Principles for intervention for protection of the public in a radiological emergency' (1993) updates and extends ICRP 40, and includes quantitative guidance on intervention levels. These general guidelines should be translated into appropriate emergency response plans by competent national authorities. It will be appropriate to institute countermeasures only when their social cost and risk will be less than those resulting from further exposure. Emergency plans should contain guidance on action levels (the setting of which is the responsibility of the

national authorities) at which various countermeasures have to be considered. To avoid confusion and to enhance the effectiveness of control measures, emergency plans should contain guidance, predetermined to the extent possible, on the levels of dose at which particular countermeasures should be introduced. Protection comes primarily from competent and trained staff, engineered safeguards and good design, quality in construction, knowledge of the location of radiation sources, and effective quality assurance in operation and maintenance. These measures reduce both the probability and the possible magnitude of the consequences of any accident.

33.2 BASIC PRINCIPLES FOR INTERVENTION

In the event of an accident, doses to the population at risk (projected doses, PD) should first be estimated for each exposure pathway without taking into account possible protective actions. The key concept for an intervention is the averted dose (AD) for each pathway, which is the dose saved by implementing a protective action. The averted dose should be sufficient to justify the intervention, irrespective of the implementation strategy.

The duration of the exposure is an important consideration since protraction of the dose influences the threshold at which deterministic effects appear. Intervention may not be fully effective, either because dose has already been received, or because the intervention may only partly reduce the projected dose. The remaining dose from each pathway (PD - AD) is the residual dose (RD). Each intervention is judged on its merits. However, the sum of residual doses from all pathways after implementation of protective actions should be kept under review because of the possibility of deterministic effects.

For each major installation, the range of consequences resulting from different accidents, corresponding protective actions and emergency response plans should have been worked out beforehand. Some protective actions must be implemented quickly. Operational intervention levels (based on optimization) help in taking quick decisions. These levels need not be on averted dose, but rather in terms of quantities which can be easily measured or estimated (e.g., time-integrated air concentration, ground deposit density or dose rate from it, activity concentration in food); such derived levels should be based on realistic rather than on conservative parameters.

ICRP 63 is mainly concerned with introduction, rather than the duration and discontinuance of intervention actions. Decisions about the continuation are less urgent and can be made as the situation develops. For some protective actions the duration is easily defined. For sheltering and evacuation, the duration is related to the course of the accident. For protective actions such as relocation and the control of foodstuffs, the decision not to continue should be made when the dose averted by continuation is insufficient to justify the continuation. Although costs of continuing a protective action may not be the same as those of initiating it, the averted dose used to justify continuation can be taken to be the same as that used to initiate the action.

In general, values can be set for intervention levels of averted dose above which it is likely that the intervention is almost always justified (generically justified intervention levels), or in the form of generically optimized intervention levels in any relevant quantities which correspond to the averted dose.

33.3 APPLICATION OF THE PRINCIPLES FOR INTERVENTION

The first concern is to keep the exposure to individuals below the deterministic thresholds. The protective action should first be justified from the viewpoint of those individuals at risk. After that, consideration should be given to justification of the action from the viewpoint of society. The societal considerations may extend the protective action to cover a larger group or they may set limits to the practical or financial feasibility of the action (e.g. evacuation of a city). Even if the proposed action is not justified from the viewpoint of the individual, decision makers may still seek to reduce the collective dose. For individual doses, both the average dose and the distribution of doses within the population should be considered. Preliminary work on economic and environmental models and on accident forecasting is needed for these assessments.

Small scale, short duration, intervention is costly without being effective. As the scale and duration are increased, the effectiveness initially increases without a marked increase in costs. Eventually, the net benefit will begin to fall. The initial planning for emergencies should include the choice of intervention levels which are justified and optimized.

Each protective action should be judged independently on the basis of the dose averted by it. The doses that would be incurred via all the pathways, some subject to protective action and some not, should be assessed.

Justification should begin by considering the average avertable dose for the whole of the exposed population. (There might be subgroups whose characteristics differ significantly from the average and for whom intervention might be justified.)

In the decision-making process, some inputs may be more easily quantified than others (e.g., averted individual or collective doses, physical risks, monetary cost of protective action), while the anxiety caused by implementation or non-implementation of the action and societal disruption are difficult to quantify. The final decision on intervention may depend on factors (e.g. social or political) other than radiological protection. But the competent authorities should be prepared to provide the radiation protection inputs in a systematic manner.

There are difficulties in expressing the various parameters on a common scale, but several decision-aiding techniques (e.g. placing a monetary value on averted dose, the "willingness to pay" approach based on public opinion poll, extended cost-benefit analysis) have been developed (ICRP 37, 55). The public should be informed of the logical basis behind the decision-making.

33.4 CATEGORIZATION OF EMERGENCIES

There is a variety of conceivable accidents to be considered in the preparation of emergency plans (e.g., those occurring in nuclear facilities, in medical or industrial uses, or in transport; loss of or damage to sources). Of the serious radiation accidents (including deaths) reported over the past 45 years, two-thirds of the events involved non-nuclear facilities (industry, research, medicine) (IAEA,1988). However, major accidents at nuclear facilities would have the most widespread and long-lasting effects.

33.4.1 Exposure Pathways

Exposures may be either external or internal, and may be incurred through various pathways (Table 33.1). Possible exposure pathways must be identified in each case. Releases to the hydrosphere will generally entail smaller risks than those to the atmosphere.

External doses may vary considerably between urban and rural environments. Seasonal variations (e.g. crop growth stage, availability of pasture) can influence contamination levels of agricultural produce.

33.4.2 Temporal and Spatial Aspects

Categorization of radiological emergencies in terms of their temporal and spatial aspects is useful. For example, external exposure to, and inhalation from, a plume may be of more immediate significance than exposure from ground contamination or ingestion of contaminated food. There would be a variability in the spatial extent of contamination due to variability of meteorological parameters; these must be considered in the emergency response planning.

Three time stages can be considered: Pre-release, release, and post-release.

The pre-release stage is that period from the time when potential or actual accidental exposure is recognized, to the time when significant amounts of radionuclides are released or the source is brought under control. During this stage urgent decision measures may be necessary. Environmental monitoring results are unlikely to be available (except in the case of extended releases). Scenarios should have been identified in advance and emergency response plans should include procedures for implementation which are based on limited inputs. The actual duration of release may vary from half an hour to a few days. For short releases, only limited off-site actions may be possible before the commencement of the release. In the case of extended releases, there may be unpredictable spikes in the release, and meteorological conditions may change in the course of the release, which would modify the dispersal patterns.

In the post-release stage there will be both decisions made concerning the implementation of further action and about the return of normal living conditions. The major routes of exposure will need to be periodically reassessed. In the post-release stage social, economic and technical factors are more important in the decision-making than in the earlier stages (e.g. nature of land use, living habits, size of population evacuated or relocated, ease of decontamination, perceptions of the population in returning to their homes). Periodic reviews of the protective actions should be carried out.

33.5 DERIVATION OF INTERVENTION LEVELS

The protective actions and routes of exposure to which they apply are summarized in Table 33.2. The most effective strategy would generally involve a combination of protective measures. Some of these actions are also applicable to workers and workplaces.

Where practicable, an Intervention Level (IL) (chosen on the basis of justification and optimization) should be established for each protective action, in terms of an appropriate parameter, such that the implementation of the protective action is triggered when the expected value of the parameter exceeds the IL.

33.5.1 Intervention at the Source

Action: Local shielding with high density materials (such as lead, steel, concrete); Applicable where source is limited in extent.

Advantages: Reduces doses to workers engaged in emergency operations; Limits spread of contamination.

Disadvantages: Not likely to avert doses to public in emergencies involving nuclear facilities; Workers involved in placing shielding in position may receive doses.

33.5.2 Control of Access

Action: Control of access should automatically follow immediately on recognition of emergency.

Advantages: Avoids exposure of people who would otherwise enter the area; Limits spread of contamination; Minimizes interference with emergency operations.

Disadvantages: Difficult to enforce for prolonged periods.

33.5.3 Sheltering

Action: Advice to people to stay indoors, close windows and doors, turn off ventilation for appropriate time; Effective means of communication should have been worked out in advance.

Advantages: Low risk countermeasure; Reduction in individual and collective doses by a factor of up to 10 depending on nature of building; Reduction of inhalation and ingestion hazards; Simple measures like

breathing through a wet handkerchief would reduce inhalation dose by a factor of up to 10; Very effective in early stages; Little financial cost for short-term and localized sheltering; Dose reduction by a factor of up to 10 can be effected from external radiation from ground deposit if sheltering is sufficiently prolonged; For prolonged sheltering, persons may be permitted to go outdoors for short periods.

Disadvantages: Societal disruption if sheltering is required for prolonged periods.

Factors Involved in Justification and Optimization: Monetary losses to individuals, society, industry, trade, agriculture; cost of enforcement (e.g. police).

Conclusion : On a generic basis sheltering will almost always be justified provided that an effective dose of 50 mSv can be averted. Optimized levels would be lower (but not by more than a factor of 10).

33.5.4 Evacuation

(*a*) *Preventive Evacuation Before Environmental Release*

Advantages: Most effective in averting dose.

Disadvantages: Decisions will have to be based on incomplete information; Most disruptive of early protective actions (anxiety, economic costs).

(*b*) *Evacuation Following Release*

Action: Dispersion in environment to be predicted quickly; People to be notified and transported as quickly as possible (A choice may have to be made between sheltering and evacuation); Negative and positive aspects of voluntary evacuation using private transport may have to be evaluated.

Advantages: Physical risks generally low.

Disadvantages: Financial and social costs high; Effectiveness will depend on meteorological conditions; Ill and elderly persons may be at higher risk.

Factors Involved in Justification and Optimization: Rural vs. urban populations, size of population, duration of evacuation, cost of transportation, additional cost of living in provisional location, compensation for loss of income and inconvenience, cost of surveillance of property left behind; special groups like pregnant women, children, hospitalized individuals, farmers.

Conclusion: Evacuation is a short-term measure and its continuation must be justified by a continuing hazard. Beyond one week, this should be considered as relocation and justified and optimized accordingly. Evacuation is almost always justified if the projected dose to whole body is likely to exceed 0.5 Sv in a day, or the averted individual effective dose for the duration of the evacuation is 0.5 Sv or 5 Sv skin dose. An optimized level of averted effective dose would be lower, but not by a factor of more than 10.

33.5.5 Administration of Stable Iodine

Action : In the event inhalation of radioiodine is likely to be a major pathway, administer 100 mg stable iodine, as potassium iodide or iodate, before or as soon as possible after exposure (50 mg to pregnant women and children above 3; 25 mg to children below 3). Administration at a period up to 6 h after exposure may reduce thyroid activity by a factor of up to 2. National authorities should work out ways of assuring quick availability to the affected population. If exposure continues beyond 2 days, a second dose of stable iodine would be useful.

Advantages: Side effects of stable iodine administration are extremely low.

Conclusion: On a generic basis iodine prophylaxis will almost always be justified provided that an average individual thyroid dose of 0. 5 Sv can be averted. Generically optimized level may be lower, but not by more than a factor of 10.

33.5.6 Personal Decontamination

Reference may be made to chapter 41, section 41.4, for decontamination procedures. Evacuation of a group of people should not be delayed by action to decontaminate individuals.

33.5.7 Injury Management

Action: Quickly assess likely doses from external radiation and internal contamination; Immediately decontaminate skin; Transfer quickly to a specialist burns unit in case of skin damage; Refer individuals with symptoms of bone marrow depression to specialist units; If required, carry out internal decontamination by suitable techniques under expert supervision.

33.5.8 Intervention in the Food Chain and Drinking Water

General: Action can be: (a) to directly restrict consumption of contaminated food and water; (b) to limit the transfer of radionuclides into the food chain from contaminated air, soil and water.

Intervention levels for these actions can be set independently for each of the food categories. Derived intervention levels can be set in terms of activity levels in the contaminated food as consumed (and not in a concentrated form such as in milk powder).

Actions complementary to the banning of food include different methods to process food (e.g. cheese production from milk), reducing incorpo-

ration of radionuclides into the food chain by changing the source of animal fodder.

Restriction on Food and Water:

The transfer of radionuclides through drinking water systems is generally unlikely to be of immediate importance.

Within the first day of release, priority must be given to controlling those foodstuffs which may be contaminated directly by deposition from the atmosphere and to controlling milk. By 1-2 days, measurements of activity in foodstuffs should be available.

Banning the consumption of contaminated foods imposes minimal risks, as long as alternate food supplies are available, but it may be costly.

For removing direct deposition on foodstuffs, outer layers should be removed from leafy vegetables, and items like fruit should be washed or peeled.

For any single foodstuff, an almost always justifiable intervention level is an averted effective dose of 10 mSv in a year. If alternate food sources are not available, higher IL would be justified.

Since the ultimate consumer is not always known, the collective dose can be optimized. (An example of generic optimization is given in an Annex to ICRP 63.)

For radionuclides with low values of dose per unit intake (most beta, gamma emitters), the optimized intervention level would be in the range of 1-10 kBq/kg, while for alpha emitters it may come down to 10-100 Bq/kg. The Joint World Health Organization/Food and Agriculture Organization Codex Alimentarius Commission (CAC) has given guidelines applicable for international trade (WHO/FAO, 1989).

Limitation of the Transfer of Radionuclides in the Food Chain : Stabling of grazing animals, moving them to uncontaminated pastures, or supplying them with alternate feeds (at least for a limited initial period) is less costly and less disruptive than the control of contaminated foodstuffs. (In the long-term, treatment of agricultural land to reduce transfer of radionuclides from soil to plants - such as by removing the upper layer of soil, ploughing, reclamation of arable land - may be considered.)

33.5.9 Relocation

Relocation refers to long-term removal of people from an affected area (in contrast to evacuation which involves a smaller time scale). It may be an extension of evacuation or may be introduced in the post-release (weeks or months later). It can be planned more carefully in advance and controlled better; but financial and social costs may be high. The efficiency of relocation in averting doses should be based on environmental measurements and modelling.

Relocation costs include those of transport, providing new social amenities, workplaces, compensation, as well as health effects associated with relocation itself. From generic considerations, an average averted effective dose of 1 Sv may serve as an almost always justified level of relocation.

(An example of generic optimization for relocation is given in an Appendix to ICRP 63. In optimization, monetary values will have to be allotted for the cost of relocation per person (which would include some initial cost plus some ongoing cost per unit time), as well as the cost for a man-Sv averted. Both these costs are likely to be higher for a developed country. There is an agreed international minimum of US $ 3000 per man-Sv (which would be applicable to developing countries); for USA, a figure as high as US $ 100,000 has been used. ICRP has assumed the recurring cost of relocation as varying between US $ 40 and $ 500 per man-month. The conclusion from a detailed calculation is that the optimized value for dose reduction in the case of relocation comes out to 10 mSv per month, with a variation of a factor of not more than 2 between developed and developing countries.)

33.5.10 Decontamination of Structures and Land Surfaces

Trained staff can carry out these tasks with little risk to the public. The larger the area involved, the harder will be the task and the less cost-effective will be the action. Means for storage and disposal of removed contaminated material will be required. Justification and optimization can be based on the collective dose averted.

33.6 PROTECTION OF INDIVIDUALS INVOLVED IN INTERVENTION

Three categories of working conditions can be defined: Urgent action at the site of accident, Implementing early protective actions and taking actions to protect the public, and Recovery operations.

Category 1: This category will comprise plant personnel and emergency service workers such as firemen. Emergency intervention will usually have a high degree of justification. Every effort should be made to keep doses below 1 Sv effective dose or 5 Sv equivalent dose to skin for all actions except life saving, for which higher doses may be justified.

Workers should be volunteers, trained in the actions required of them, and should be informed of the risks. They should be given protective devices (respiratory protection, protective clothing, iodine tablets). Their doses should be measured and recorded, and medical advice taken when needed.

Category 2: This category will comprise police, medical personnel and drivers. Since their exposures can be controlled, their work should be justified, and some crude optimization carried out. The aim should be to keep the doses within those permitted under normal conditions. (Some would be radiation workers and others not.) All the workers should be trained, informed of the risks, and given protective devices. Their doses should be assessed and recorded.

Category 3: The actions required may include repairs to plants and buildings, waste disposal and decontamination of areas. These actions can be planned and exposures can be controlled. Such workers should be subject to the normal system of protection for occupational exposure. Their doses should be assessed and recorded.

33.7 EMERGENCY RESPONSE PLANNING

33.7.1 General

The general considerations for emergency response planning have been given in ICRP 60 (see chapter, 7, section 8).

33.7.2 The Role of Intervention in Emergency Planning

The effective implementation of protective action will depend upon the adequacy of advance planning covering a wide variety of conceivable scenarios. It is easier to plan the response to emergencies in a large, centrally controlled facility (e.g. nuclear power plant) than in others (e.g. industrial radiography, medical installations, transport accidents). There will be need for a wider expertise than solely that of conventional radiation protection to develop generic and specific operational intervention levels at the planning stage for the emergency response as well as during the management of an actual emergency. The detail with which such planning is carried out should be related to the probability of occurrence.

Different source terms should be considered for each type of accident (including the nature, quantity and physico-chemical form of the radionuclide, time available for the release, and expected duration of release).

Where there is possibility of a transcountry impact of releases, national authorities should have appropriate emergency planning.

In view of the wide variety of scenarios, flexibility should be maintained in the scheme of intervention. However, a spectrum of intervention levels should be established beforehand for a number of possible accident scenarios. These analyses indicate the sensitivity of the intervention level to the significant variables and enable a spectrum of levels and relevant protective measures to be established appropriate to different circumstances, thereby providing flexibility whilst retaining sensible constraints.

33.7.3 Lost Sources

It is not easy to foresee the accident scenarios. The loss and damage of a high activity source may lead to wide spread of the contamination and exposure of many persons, before the accident is detected, and recov-

ery operations may require medical care and considerable resources for monitoring and cleaning up.

Records should be kept of the location and movement of sources that could be lost. When it is recognized that a source is lost, a detailed description of it should be communicated to those most in danger. Improving public recognition of the system of markings for radiation hazards would be a useful measure. Efforts should be made for rapid search for and recovery of lost sources. Emergency preparedness plans should cater for an infrastructure which, in addition to the management in charge of the concerned installation, may include local bodies, fire brigade, police, medical physicists, and medical specialists knowledgeable in the management of radiation injuries. Recognition of the nature of radiation injury depends on the education of non-nuclear workers also.

33.8 SUMMARY OF RECOMMENDED INTERVENTION LEVELS

Table 33.3 summarizes the almost always justified intervention levels, as well as the range within which the optimized intervention levels are expected to be found.

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Table 33.1: Principal exposure pathways of relevance to radiological emergencies

(from ICRP 63)

External exposure from :	Source or facility Plume Radionuclide contamination on surfaces Radionuclide contamination of skin and clothing
Internal exposure from :	Inhalation of radionuclides in plume Inhalation of resuspended radionuclides Ingestion of contaminated food and water Ingestion of radionuclides from contaminated materials Absorption through skin and wounds

Table 33.2: Protective actions for averting exposures via various pathways
(from ICRP 63)

Route of exposure	Protective action
External irraditaion from the source, plume and/or radionuclides deposited on the ground	Control of access
Inhalation or ingestion of radionuclides in the plume and/or resuspended radionuclides	
External contamination of individuals	
External irradiation from the source, plume and/or radionuclides deposited on the ground	Shielding
External contamination	Protective clothing
External irradiation from the source, plume and/or radionuclides deposited on the ground	Sheltering
Inhalation of radionuclides in the plume External contamination of individuals	
External irradiation from the source, plume and/or radionuclides deposited on the ground	Evacuation
Inhalation or ingestion of radionuclides in the plume and/or resuspended radionuclides	
External contamination of individuals	
Inhalation of radioiodine in the plume	Stable iodine administration
External contamination	Decontamination of individuals and clothing
Ingestion of radionuclides	Intervention in food chain and water supply (a) Restrictions on food and water - banning consumption - processing of food/water

(*contd ...*)

Route of exposure	Protective action
Ingestion of radionuclides	(b) Decreasing radionuclide incorporation into the food chain - alternative fodder - binders - deep ploughing and other changes in agricultural practice
Inhalation of resuspended radionuclides	- temporary fixation of contamination in ground (e.g.spraying)
External irradiation from radionuclides deposited on the ground	Relocation
Inhalation and ingestion of resuspended radionuclides	
Inadvertent ingestion	Docontamination of materials and controls on use of contaminated materials
External irradiation Inhalation of resuspended radionuclides	Decontamination of ground and other surfaces

Table 33.3: Summary of recommended intervention levels
(from ICRP 63)

Type of intervention	Intervention level of averted dose (mSv)	
	Almost always justified	Range of optimised values
Sheltering	50	
Administration of stable iodine: equivalent dose to thyroid	500	Not more than a factor of 10 lower than the justified value
Evacuation (<1 week):		
whole body dose	500	
equivalent dose to skin	5000	
Relocation	1000	5-15mSv per month for prolonged exposure
Restriction to a single foodstuff	10 (in 1 year)	1000-10,000 Bq kg^{-1} (beta/gamma emitters) 10-100 Bq kg^{-1} (alpha emitters)

CHAPTER 34

PRINCIPLES FOR LIMITING EXPOSURE OF THE PUBLIC TO NATURAL SOURCES OF RADIATION

34.1 INTRODUCTION

ICRP 26 explicitly stated that the recommended dose limits shall not apply to contributions from 'normal' natural radiation. However, ICRP 26 recognized that there may be levels of natural radiation which might have to be controlled, to the extent practicable, in much the same way as for artificial sources, but did not give any practical guidance on the principles for such control. ICRP publication 39, 'Principles for Limiting Exposure of the Public to Natural Sources of Radiation' (1983) gives broad guidelines on the subject.

Almost all exposures to natural sources of radiation are controllable to some extent but the degree of controllability varies very widely as does the complexity, cost and inconvenience of the possible control measures. Controllability is therefore a major factor in any system of dose limitation. From this point of view, there is a clear difference between *existing* exposure situations where any action would have to be remedial, and *future* situations, which can be subject to limitation and control at the stage of decision and planning. Remedial measures are more likely to be objectionable than are future controls because they withdraw existing facilities or freedoms, or require changes in life-style, or cause increased expenditure. Future control may do all these things but only by comparison with a situation which has not been allowed to develop. There is no appreciable sense of loss.

Examples of existing situations which can be influenced only by remedial action are: living in existing houses, maintaining present production of building materials, supplying water from existing facilities, flying in the present manner, burning natural gas from old wells, using fertilizers from operating mills and factories, and food produced in the present manner. A future counterpart to each of the existing exposure situations can be visualized where the exposure situation can be subject to control. By far the most important case is concerned with the presence of radon in houses.

There are some ill-defined borderline cases also. For example, an existing production of highly radioactive building material may be discontinued, if such *remedial* action is necessary; but it may, alternatively, be possible to treat it as an operation and to require it to conform with conditions intended for new production and *future* situations. If such changes are applied to existing practices, they are retrospective in nature and at variance with earlier judgement of the acceptability of the situation.

34.2 EXISTING EXPOSURE SITUATIONS

In existing situations, the exposures can be altered only by taking remedial action. The hazard or social costs involved in the remedial measure must be justified by the reduction of risk that would result. It may sometimes be possible to gauge, by an analysis of the effectiveness and costs of the remedial action, levels below which it would not be appropriate to take remedial action.

Action levels should be formulated specific to the initiation of the remedial action being considered. Since circumstances would vary from situation to situation, ICRP does not recommend action levels that would be appropriate for all occasions. The process of deciding how far to go with remedial actions, either singly or in combinations, should involve a process similar to that of optimization, which minimizes the radiation detriment to the exposed individuals plus the detrimental costs, both financial and social, of the remedial actions. The optimization process should be constrained by the requirement that the total cost of all the detriment to the individual should not exceed that corresponding to his exposure in the absence of remedial measures. (If, however, the level of individual risk in the absence of remedial measures is so high that society is not prepared to allow it, even if it were acceptable to the individual, a constraint at a lower level of detriment would have to be applied.)

When action levels are used, it is implicit that there is knowledge of doses and corresponding risks. For practical reasons, competent authorities should establish *investigation levels* to separate exposures that require investigation from those that do not.

Limited studies may indicate which sources (e.g. building materials) might need monitoring, and these sources may often be identified by other means than radiation measurements. For example, the type of building material, ground, ventilation, construction, and so on, may suffice for a

crude first classification. Water in deep wells is usually more active than surface waters. The concentration of ^{40}K in food will not influence the radiation exposure significantly because the potassium concentration in tissues is under homeostatic control; since this exposure situation cannot be administratively controlled, neither an investigation level nor an action level is needed.

Thus investigation levels will be applied to sample measurements and will be related to the likely action levels. In the case of radon in buildings, the day-to-day variation in levels will necessitate more extensive measurements since the action has to be decided on the long-term average exposure, not on the basis of a single measurement.

34.3 FUTURE EXPOSURE SITUATIONS

In this case the position is closer to that recommended by ICRP for artificial sources of exposure. Future exposures should be justified, and optimization should ensure that the detriment is minimized, i.e. the doses should be kept as low as reasonably achievable. However, ICRP's recommended dose limits should *not* be applied to these situations, since the limits were set with full recognition of doses from natural sources which were not to be included in the limits. Instead, the exposure of the most highly exposed individuals should be limited by the application of an *upper bound* of individual dose (set by the competent authority) in the optimization assessment. For natural sources, ICRP does not recommend a dose limit applying to the total exposure from all such sources, and hence the upper bound for each class of source of exposure must stand on its own. As in existing situations, there will often be merit in using investigation levels applied to simple monitoring techniques in order to select those situations which most need detailed attention. ICRP's advice on the application of cost-benefit analysis in optimization (e.g. ICRP 37 (1983) and ICRP 55 (1989)) modified in relation to the use of action levels and upper bounds, rather than dose limits as constraints in the optimization process, is also applicable against exposure from natural sources.

Administrative intervention in order to limit doses to the public from natural sources of radiation has already occurred in a number of countries. However, intervention has often occurred in cases where the problem has been local and where some organization has been identified as responsible for the enhanced radiation levels; such intervention is not necessarily consistent with the present (ICRP 39, 1983) recommendations of ICRP.

ICRP 39 discusses in some detail the problem of exposure to naturally occurring radon daughters in dwellings. This is dealt with in Chapter 42, Sections 4 and 5.

CHAPTER 35

PROTECTION AGAINST IONIZING RADIATION IN THE TEACHING OF SCIENCE

35.1 INTRODUCTION

The guidelines given in ICRP publication 36, 'Protection against ionizing radiation in the teaching of science' (1983) are based on the recommendations of ICRP 26 (1977). ICRP 36 supersedes ICRP publication 13, 'Radiation protection in schools for pupils up to the age of 18 years' (1976), where the recommendations were based on ICRP 6 (1966).

The present recommendations apply mainly to the teaching of science to pupils under 18 at secondary level. Large numbers of students are learning natural sciences and may be exposed during demonstrations and experiments in biology, physics, chemistry and engineering involving radiation sources. These recommendations do not apply to the staff.

It is obvious that all the protection recommendations which are dealt with in detail in various ICRP publications are also applicable, in a general way, in the present case, although the levels handled are very small. Therefore this chapter draws attention only to certain aspects specific to protection of pupils. In this report ICRP 36 has deliberately introduced didactic elements that are not found in other ICRP publications in order to foster a careful, rather than fearful, approach to radiation among pupils.

35.2 GUIDELINES

Pupils should not be exposed to radiation without valid reason; experiments should be relevant to the course of instruction and properly planned with protection in mind. Source strengths should be the minimum necessary. (Deliberate irradiation, such as the radiographing of hands, is unacceptable.)

These recommendations are intended to ensure that requirements for radiological surveillance are minimal. Pupils will not require personal monitoring, and members of the staff are unlikely to require it, but it may

sometimes be used for instruction. Monitoring of sources and experimental facilities will, however, be necessary to determine the dose equivalents from exercises, to check periodically the adequacy of shielding, and to detect contamination.

The responsibility for radiation safety falls to the governing body acting through the principal. A teacher should be appointed to supervise radiation protection and be suitably trained. If necessary, an advisory committee may be formed. The medical officer for the establishment should participate in the arrangements. The fire brigade should be informed of the sources held. Adequate facilities and financial support should be provided for the protection programme. All exercises should be planned and rehearsed. Where potential exposures may be very low, an exemption scheme might be devised, and, if necessary, got approved by the competent authority. It is important that only sources that have been approved by the competent authority are used. Close supervision of the pupils and availability of written instructions for the pupils are essential.

In cases of suspected significant exposure of any pupil, health physics and medical advice should be taken. Records of such incidents should be kept.

35.3 DOSE LIMITATION FOR PUPILS

The authorised dose limits for individual pupils have been set at the values given in Table 35.1. These values are below the dose limits for members of the public because of the low ages of the pupils and the large numbers learning natural sciences, and because no course of instruction need involve higher doses.

A sure way of keeping doses within the limits is to restrict the value of the dose from each teaching exercise to one-tenth of the above limits. Dose equivalent rates from sealed sources should similarly be restricted, and the amount of unsealed material used in each exercise should be restricted to one-tenth of the ALI for workers.

35.4 PROGRAMME OF PROTECTION

The following sections discuss the types of radiation sources likely to be used in teaching, indications of working procedures, and recommended restrictions on radiation levels and quantities of radionuclides.

35.4.1 X-ray Apparatus and Sources

It is convenient to distinguish apparatus that produces X-rays by design from electrical devices that are sources of unwanted X-rays. The former include X-ray sets for analysis, radiography and irradiation. The latter include a number of low pressure high voltage devices in which accelerated electrons impinge on matter, examples being discharge tubes, cathode ray tubes, microwave oscillators and amplifiers, and electron microscopes. Any device in which electrons are accelerated to more than 5 kV should be regarded as a potential source of unwanted X-rays, and carefully examined.

Each X-ray apparatus and the X-ray beam should be fully enclosed. The enclosure may be a cabinet or room. The equivalent dose rate at 5 cm from the surface of the enclosure should not exceed 5 $\mu Sv\ h^{-1}$. The enclosure should be electrically interlocked to the X-ray controls so that access cannot be gained without shutting off the set. The apparatus should have the radiation warning sign, a timer and an indicator lamp. The appointed teacher should make routine checks on the apparatus and enclosure.

35.4.2 Sealed Radioactive Sources

Examples of alpha, beta, gamma and neutron sources used in teaching are: ^{241}Am, ^{90}Sr, ^{60}Co and ^{210}Po-Be respectively. Sources should be held in a separate shielded ventilated lockable room with a radiation sign and appropriate legend on the outside. An inventory of the sources should be maintained by the authorized teacher. The sources should be inspected and tested routinely.

The equivalent dose rate outside the store should not exceed 5 $\mu Sv\ h^{-1}$ at 5 cm from the surface. The recommended maximum dose rates at 10 cm fom the sealed sources are given in Table 35.2.

The sealed sources should satisfy the following broad requirements. The radioactive material should be embedded or enveloped in a metal or other durable matrix. The encased material should be placed in a strong capsule or holder of appropriate size and shape to facilitate the use of handling tools. The emitting surfaces of alpha and beta sources, which are particularly vulnerable to damage, should be recessed and protected by screens. Details of the radionuclide and its activity should be marked on the holder. The whole should be stowed in a shielded container which

should carry the radiation sign. Leakage tests on the sources should be made by the appointed teacher every year, and when there is reason to doubt the integrity of the source.

Solid specimens of uranium metal and hard minerals of uranium and thorium should be regarded as sealed sources. The fact that they may emanate radon should be recognized.

35.4.3 Unsealed Radioactive Sources

These may be in the form of liquid, powder, or soft solid. The main hazard is by inhalation or ingestion. Examples of sources used in teaching are given in Tables 35.3 and 35.4 with the recommended restrictions on activities used for each separate teaching exercise. Normal recommendations on design of the radioactive laboratory and procedures for working with unsealed sources apply. In particular, the diluting, dispensing, and disposing of unsealed sources should be done by the designated trained teacher, and records should be kept. Facilities for management of spills, monitoring and decontamination of pupils should be readily available.

Table 35.1: Authorized equivalent dose limits for pupils
(from ICRP 36)

Effective dose	0.5 mSv in a year
Equivalent dose to any single organ or tissue	5 mSv in a year

Table 35.2: Recommended restriction on equivalent dose rates at a distance of 10 cm from sealed sources used in teaching exersises
(from ICRP 36)

Source	Dose rate ($\mu Sv\ h^{-1}$)
Beta	50
Gamma	10
Neutron	10

Table 35.3: Recommended restriction on activities of some unsealed radionuclide sources used for separate teaching exersises
(from ICRP 36)

Radionuclide	Activity (MBq)
^{14}C	10
^{32}P	1
^{35}S	10
^{45}Ca	2
^{59}Fe	1
^{110m}Ag	0.2
^{131}I	0.1

Table 35.4: Recommended restriction on unsealed quantities of thorium and uranium with natural compositions used for separate teaching exercises

(from ICRP 36)

Element	Activity (MBq)	Mass (mg)
Thorium	5	1
Uranium	50	5

The restriction on uranium is deemed to satisfy chemical toxicity and radiation protection requirements.

CHAPTER 36

RADIATION PROTECTION PRINCIPLES FOR THE DISPOSAL OF SOLID RADIOACTIVE WASTE

36.1 INTRODUCTION

This chapter is a summary of ICRP publication 46, 'Radiation protection principles for the disposal of solid radioactive waste' (1985).

The disposal of radioactive wastes is within the scope of ICRP's system of dose limitation. Previous recommendations have focussed on the discharges of radioactive wastes in the form of effluents and the protection of the public in the context of the resultant doses, together with the protection of the workers during the handling of radioactive wastes prior to discharge or in storage.

Disposed solid wastes are intended to remain isolated from people for a considerable period of time. The associated risks depend on the events and processes that could cause a release of radionuclides into the biosphere, or influence the rate of release or of transport of radionuclides through the environment. Some of these events are certain to occur, while others have constant or time-varying probabilities of occurrence. In this situation, it is difficult to apply any standards which consist solely of dose limitation because some circumstances - even if they have a low probability of occurrence - can be envisaged which, if they occur, would lead to doses above any selected limit. In the past, ICRP has recommended that the dose limits do not apply to situations in which the doses are not controlled, and can be limited only by intervention. This recommendation does not contribute to decisions concerning the objective of limiting the probability of occurrence of such uncontrolled situations. The Commission now wishes to extend its recommendations so as to deal with probabilistic situations in waste management. The requirement that all doses should be taken into account in the optimization of protection cannot always be applied without some consideration of the probabilities that the doses will be received. This situation does not arise in the case of routine discharges of gaseous and liquid effluents, where it is certain that radionuclides will enter the biosphere at a known rate, and hence the

system of dose limitation can be applied. Even in these cases, there is an implicit probability distribution of the magnitude of the individual doses and the expectation value is one of the parameters used to represent the distribution.

Another difficulty regarding solid waste disposal is the length of time periods of concern. Many wastes contain some long-lived radionuclides, and the radiation impact from their disposal will extend far into the future. There are difficulties in predicting future conditions, whether demographic, biological, physical or other aspects, with sufficient confidence. What weight is to be assigned to risks to future populations when disposal decisions are taken?

It is necessary to build on the present system of dose limitation to provide a more explicit method for judging whether any particular waste disposal option is radiologically acceptable. The system of dose limitation (particularly optimization) should be applied to the entire waste management system and could, for example, entail comparisons between discharges as effluents or disposals as solidified wastes.

The development of criteria for reuse of materials is not covered in ICRP 46 because they are not wastes.

36.2 RADIOACTIVE WASTES AND MANAGEMENT OPTIONS

Radioactive wastes arise from a wide range of activities: use of radioactive materials in hospitals, research laboratories and industrial processes, and nuclear power plants. It is electricity production and the associated fuel cycle of uranium mining, fuel fabrication and fuel reprocessing, that generate the largest quantities of wastes that require long-term disposal arrangements.

36.2.1 Waste Characteristics

The two principal characteristics of wastes that influence their management are their volume and radionuclide content. Wastes from medical procedures and from industrial processes generally contain short-lived radionuclides, but their volume can be large. (An example of an exception where long-term considerations arise is the disposal of radium sources, but the volumes are small.)

The nuclear fuel cycle generates wastes at every step. Mining of uranium results in accumulation of very large volumes of mill tailings containing radium, thorium and unextracted uranium. These radionuclides and their daughter products are long-lived. Uranium enrichment and nuclear fuel fabrication produce significant volumes of wastes, most of which is of low activity concentration; natural uranium is the only significant contaminant. The operation of nuclear power plants gives rise to a variety of wastes resulting from neutron activation of structural materials, and leakages of small amounts of fission products from the fuel. These wastes consist mainly of resins and filters used to trap radionuclides in cooling circuits, storage ponds and ventilation systems. Low levels of contamination with longer-lived actinides may be associated with failures of fuel cladding. The volumes of wastes are substantial but predictable. Reprocessing of spent fuel to recover uranium and plutonium allows over 99% of the radionuclides to be concentrated into a small volume of high-level waste, but also gives rise to waste streams containing lower levels of fission products and actinides. If spent fuel is not reprocessed, it must be eventually considered itself as a waste and will contain plutonium generated in the reactor, as well as other radionuclides that would have been in the high-level wastes and other streams from reprocessing. Decommissioning of nuclear installations will give rise to very large volumes of material with a wide range of radionuclide contents, varying from barely detectable contamination on dismantled structures to high levels of contamination and activation of some metal components in the reactor structure and containment.

36.2.2 Waste Management Options

Some fraction of the radionuclides may be removed from liquid and gaseous effluent streams before their release; this gives rise to a waste in a solid or slurry form. In some cases containment of this waste will allow a natural decrease through radioactive decay. But for long-lived radionuclides, containment in storage facilities will only provide a period of delay before dispersion takes place; even disposal in regions of high natural isolation will not provide a guarantee of indefinite isolation.

For liquid wastes, some treatment is generally required to convert them to a stable and solid form for storage and disposal.

Some wastes containing very small quantities of radionuclides and arising in large volumes may be disposed of as they arise, but interim storage (especially for short-lived nuclides with half-lives from a few days

to a few years) is often useful before eventual disposal. Interim storage implies surveillance and maintenance of the facility; this involves operational exposures to concerned personnel and risk of accidental releases. Subsequent disposal options will depend on the waste volumes, the degree and likely duration of containment. The current range of potential options is detailed in the following:

36.2.2.1 Ordinary Refuse Disposal

This is suitable for small quantities of short-lived wastes. Very large volumes of conventional wastes are disposed of in land-fill facilities, but the duration of control is generally short, and isolation cannot be guaranteed for more than a few years.

36.2.2.2 Trench Disposal

This gives more control than ordinary refuse disposal, but a physical barrier to water has to be provided to prevent access to leaching and dispersion.

36.2.2.3 Burial in Shallow Engineered Facilities

This is suitable for large volumes of solid wastes. The conditioning of wastes, the integrity of the engineered structures and the geochemical characteristics of the burial sites can immobilize and isolate the radionuclides for a considerable time (a few hundred years). The proximity of the wastes to the surface may require institutional surveillance of the site for considerable periods as a safeguard against the consequences of intrusion and dispersion.

36.2.2.4 Disposal on the Ocean Floor (Sea Dumping)

This option could also accommodate large volumes of wastes. The activity released into the deep ocean will be diluted and dispersed in a large volume of water and some may be adsorbed on to sediments. The inaccessibility of the ocean floor provides some isolation but there may be some eventual return of long-lived nuclides to man's immediate environment due to dispersal and environmental transport mechanisms.

36.2.2.5 Disposal in Continental Geological Formations

This can provide a very long period of isolation of high levels of long-lived wastes if ground water circulation is slow. Salt formations, granite, clay, basalt and volcanic tuff are potential geological formations of relevance.

36.2.2.6 Disposal into Geological Formations under the Deep Ocean Floor (Sub-sea Bed Disposal)

This is similar to disposal in geological formations but with the additional benefit of sea disposal.

36.2.2.7 Disposal into Space or Solar Orbit

Its technical and economic feasibility has yet to be determined.

36.2.2.8 Transmutation

Transmutation of long-lived nuclides into shorter-lived ones is theoretically feasible. An example is reuse of separated plutonium as fuel in a reactor. Its extension to other heavier elements, although technically feasible, would involve huge new facilities which would themselves generate secondary wastes involving additional occupational exposure.

36.3 CHARACTERISTICS OF RELEASE SCENARIOS

36.3.1 Mechanism of Radionuclide Release and Dispersion

Generally, the primary cause of release of contained waste into the environment is degradation of conditioned waste and its container by water. For shallow land burial, radionuclides may move with ground water or because of accidental intrusion by man. For deeper land burial and sea disposal, there can be transfer and dispersal by water movement, modified by reconcentration processes. Some of these will lead to a gradual release of radionuclides into the environment. Models drawing on *in situ* experiments (taking into account sorption, diffusion and other phenomena) often allow 'normal' release processes to be identified, leading to a reasonably predictable exposure pattern in space and time.

Other processes are not gradual but probabilistic (e.g. seismic and tectonic phenomena which modify water flows; future human activities like milling, mineral exploration, agriculture and building; changes in water tables; flooding; geomorphological changes; and meteorological effects). They may modify existing pathways or introduce new pathways, and give rise to immediate major impacts as well as initiate a sequence of processes leading to subsequent impacts.

ICRP's recommendations on environmental monitoring deal adequately with normal events. The results can be used for optimization and setting of upper source bounds.

36.3.2 Probabilistic Events

For probabilistic events, due to lack of information, low probability events have to be assigned a 'subjective probability' through 'best estimates' or 'engineering judgements'. A number is assigned to the likelihood of an event occurring in a definite period of time. The validity of this approach is dependent on maintaining coherence when assigning probability values to events; this means that all of the probabilities are assigned in compliance with the rules of the calculus of probabilities. (Thus the complement of an event with probability p is assigned a probability $(1-p)$; events which occur with a greater frequency are given larger probabilities; if event A is more probable than B, and B is more probable than C, then A is more probable than C.) In this system, probabilities assigned for various events will be consistent and continuous, and low probability events can be integrated with higher probability events for a complete analysis of options.

A conceptual distinction is to be made between the three probabilities, viz. probability of an unforeseen occurrence in a waste repository, the probability that the event will have a consequence for the integrity of the repository, and the probability than an individual will receive an exposure as a result of the event.

There are several uncertainties in the assessment of the radiation impact from waste disposal:

(a) Conventional uncertainties due to imperfect knowledge and inadequate modelling (e.g. present knowledge of future conditions and events affecting disposal site integrity and pathways; lack of precision in technical parameters involved in modelling radionuclide transport and transfer through food chains, air or water pathways to humans).

(b) Intrinsic uncertainties resulting from the expected outcome of low probability events, or from statistical treatment of the variables, however certain they may be.

Uncertainties increase as the period of prediction increases (e.g. demographic patterns, medical and other technologies that may reduce dose consequences). Assumptions on uncertainties should be cautious and reasonable, and presented explicitly. To a certain extent some of the uncertainties can be quantified within certain boundaries. Some events will have a large uncertainty associated with estimates of their probability. Low probability events will have an intrinsic uncertainty associated with the magnitude of their outcome. As a result, there will, in both cases, be a large uncertainty in the radiation impact.

36.4 INDIVIDUAL LIMITS

36.4.1 Individual Dose Limits

These apply to 'normal' scenarios and represent the lower boundary of a range of risks that are considered unacceptable, and apply to the estimation of the total exposures received from all sources and practices (excluding medical and natural sources). They are usually applied to limit doses to the critical group. Exposure at the dose limit from a single source should not be permitted for normal operations or over extended periods of time, since they would leave no margin for other exposures of the critical group. The critical group, i.e. those who are expected to receive the greatest exposure, must be identified. Allowance for other sources can be formalized by using a Source Upper Bound (applied to the maximum value of the average dose in the critical group, whether this occurs now or in the future), rather than the dose limit. The critical group should be representative of those individuals in the population expected to receive the highest dose and should be relatively homogeneous with respect to the location, habits and metabolic characteristics that affect the doses. For example, it may comprise a group of people living in an area near the repository and whose water would be obtained from a nearby ground water aquifer. It may comprise existing persons or a hypothetical group, in the present or the future. Because the actual doses in the entire population will constitute a distribution for which the critical group represents the extreme, this procedure is intended to ensure that no individual doses are unacceptably high.

36.4.2 Individual Risk Limits and Risk Upper Bounds

These apply to scenarios involving probabilistic events. Account is taken of both the probability of an exposure and its magnitude. A risk limit and risk upper bound can be established in direct analogy with the dose limit and upper bound for normal releases. We can either add the risk from routine and probabilistic situations and apply a limit to the sum (which is simpler in concept), or the two risks could be treated and limited separately. The design and operational features intended to limit the two kinds of risk may be different. ICRP recommends that its current dose limits should continue to apply to routine situations (including normal scenarios for repositories) and that risks from probabilistic events should be limited on a similar basis. ICRP also recommends that risks to future individuals should be limited on the same basis as are those to individuals living now.

ICRP was aware of the existence of probabilistic events when it set the numerical value for the dose limit for routine situations, so that specification of a separate risk limit does not imply a need for a corresponding reduction in the dose limits. Further, the exposure of members of the public rarely approaches the dose limit and the lack of precision in predicting future situations does not warrant modifying dose limits to accommodate the risk limit.

Just as an annual dose limit of 1 mSv over a lifetime implies a constraint on the average of the annual risk to a level less than about 10^{-5}, similarly, it seems reasonable to restrict the risk in a year to the critical group from probabilistic events so that it is also less than 10^{-5}.

36.4.2.1 Definition of and Expressions for 'Risk'

The term 'risk' is used with different connotations in various disciplines. It is preferable that risk should be defined in a simple, objective and quantitative manner. This does not preclude subjective consideration of the separate components making up a risk, but such factors would only enter into the decision through future consideration of the overall acceptability of disposal options or the optimization of protection, rather than through limiting individual risks.

Previously ICRP defined 'risk' to denote the probability of a serious detrimental health effect from a dose. To include probabilistic events, the Commission now recommends that risk be defined as the probability that a serious detrimental health effect will occur in a potentially exposed

individual or his descendants. The risk, R, to an individual or critical group from an event giving rise to a dose in the range D to $D + dD$ is given by

$$R = P(D).\ p(eff/D),$$

where $P(D)$ is the probability of an event giving rise to a dose between D and $D + dD$; and $p(eff/D)$ is the probability of a serious detrimental health effect in the exposed individual or his descendants from the dose D.

For doses in the stochastic region in which the effective dose E, can be used,

$$R = P(E).\ rE$$

where r is the probability of a serious detrimental health effect per unit effective dose. This is valid for small values of rE. For higher values, we have

$$R = P(E).\ (1 - e^{-rE})$$

At higher doses, the values of R will be larger than those calculated from the above expression, since non-stochastic (deterministic) effects will also have to be taken into account. To find the total risk, the values of R must be summed over all events, as explained later.

36.5 APPLICATION OF INDIVIDUAL REQUIREMENTS TO A SOURCE

36.5.1 Source and Risk Upper Bounds

The total dose to the critical group will be composed of doses from the source being assessed, and doses from other (local, regional or global) sources. Also, overlapping doses from different sources to the same critical group are not restricted to any given instant in time. The dose rate resulting from the combined effect of several annual releases may increase to some steady state, or, if the releases stop at some time in future, a maximum far in the future.

To allow for doses from present practices and to provide a margin for unforeseen future activities, ICRP recommends that national authorities select a fraction of the dose limit as source upper bound for each source of exposure, to ensure that the individual exposures will remain below the

dose limit. Similarly, a fraction of the risk limit may be selected by the national authority as a risk upper bound for each source.

For waste disposal, where normal release mechanisms are assumed to occur, the source upper bound for design constraint for the repository would be the difference between the dose limits and the summation of other exposures, and the allowance for future activities. The global contribution to the critical group may be assessed from forecasts by relevant international bodies, particularly UNSCEAR. A prudent fraction of the dose limits should be reserved for potential future sources. For the risk upper bound, a requirement would be that the sum of all risks from probabilistic events associated with a single source, which could expose the same critical group at any time, should be be less than the risk upper bound selected.

36.5.2 Assessment of Individual Risks

We require to know the probability of the exposure at various levels of annual dose. A distinction is to be made between the probability of an exposure and the probability of disruptive events at the waste disposal facility which may give rise to the exposure; the link between the two parameters involves the distribution over time of the radiation impact associated with the disruptive event.

The basic information to characterize the likelihood of a disruptive event consists of the probability of occurrence in a defined interval (usually a year) as a function of time. For some events, the probability of occurrence in a given time interval will be constant or slowly changing with time; the probability of there being an event in a given time interval can be assessed from Poisson statistics. For other types of events, both natural and man-induced, the probability of occurrence in a given time interval, $f(t)$, varies with time after closure of the repository, and may also depend on the occurrence of other events, so that more complex modelling becomes necessary. Also needed is an estimate of the *conditional radiation impact*, $g(t, T\text{-}t)$, (assuming the event has occurred), in terms of the individual dose rate as a function of time of occurrence of the initial event at time t, and the time after the event $(T\text{-}t)$. If the doses are in the stochastic region and assuming that the dose rate changes slowly over an individual's lifetime, L, the *conditional probability of death*, given the exposure, is $rLg(t, T\text{-}t)$. Combining the probability of the primary event with the conditional probability of death (given the exposure), we get for the unconditional probability of death

$$P(T) = rL \int_0^T f(t)\, g\,(t, T-t)\, dt$$

The annual probability incurred for this class of events $P^I(T)$ will be $P(T)/L$. The assessment of compliance with the limit will, therefore, be that $\Sigma\, P^I(T)$ never exceeds the annual risk limit of 10^{-5}, or the risk upper bound established below that risk limit, for any value of T, as illustrated in Fig. 36.1.

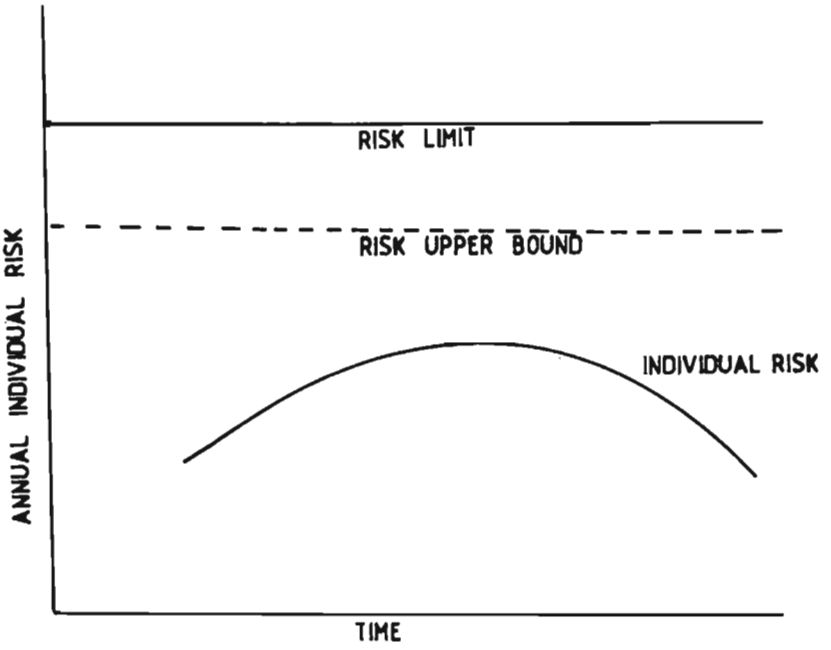

Figure 36.1. Illustration of comparison with the risk limit, or risk upper bound, of predicted individual risks at various times, derived from integration over probability distributions of exposures and conditional consequences, summed over all classes of events (from ICRP 46).

We can also derive the probability of a particular level of annual dose at any time T, and therefrom a curve can be generated illustrating the overall probability of exceeding a given level of annual dose, as illustrated by Fig. 36.2. This shows that, for a particular event, the probability of exceeding very low annual doses is reasonably constant and related to the probability that the event has occurred prior to time T. The shape of the curve will depend on the event or scenario under consideration.

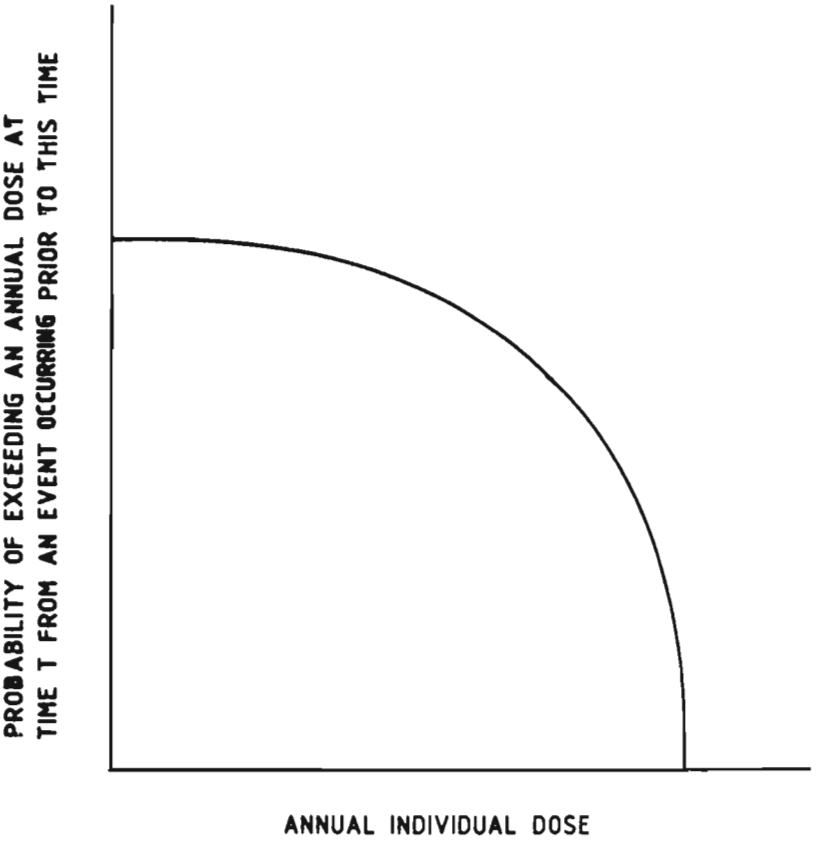

Figure 36.2. Illustration of the probability of exceeding various levels of annual dose for an individual living at time *T* in the future, as a result of one type of event (from ICRP 46).

For events that initiate a prolonged contamination of the environment (hundreds to thousands of years) the results expressed in Fig. 36.2 will primarily reflect the probability that the initiating event has occurred not only prior to the time when the individual lives, but also sufficiently long before so that the radionuclides have migrated through the geosphere into the biosphere. The probabilities of exposure are, therefore, related to the cumulative probability over the time-span of the disruptive event happening before the time *T*. At the other extreme, for events that give rise to exposures of short duration (such as during human intrusion) the results are related to the probability of the event occurring in the year being considered.

36.5.2.1 Criterion Curve

The basic requirement for compliance with the risk limit or risk upper bound is that $\Sigma\ P^{I}_{event}(T)$ is less than the appropriate limit. This overall condition is illustrated in Fig. 36.1. One procedure for applying individual-related requirement to probabilistic events is to express these limits in a criterion curve (Nuclear Energy Agency, 1984; Beninson and Lindell, 1984), as illustrated in Fig. 36.3. The curve has the following features:

(a) A probability limit of one for annual doses up to 1 mSv;

(b) An inverse proportionality region where only stochastic effects occur (values representing the product of the probability of a dose, the annual dose and the probability of a health effect per unit dose);

(c) A non-proportional region where deterministic effects can occur (for doses exceeding a few Sv), to take into account the increasing probability of death;

(d) A constant probability of death for lethal doses.

The risk upper bound can be incorporated directly into the criterion curve, as illustrated in Fig. 36.3 (some fraction of the limit being selected for the source under consideration). The criterion curve can indicate whether a given waste disposal option complies with the risk-related requirement, using the following procedure:

(a) The sequence of events with the potential to disrupt a waste repository is identified;

(b) The probability of occurrence of each event, the corresponding activity release and consequent exposure to the critical group are assessed;

(c) The point representing the probability of occurrence of the initial event and all other environmental factors and the corresponding maximum dose is plotted.

If the point is in the unacceptable region, the proposed option is rejected. If it is in the acceptable region, the proposed option is further assessed regarding its ability to meet the requirement that the total risk from all scenarios, resulting from the combination from different classes of events, complies with the risk upper bound condition.

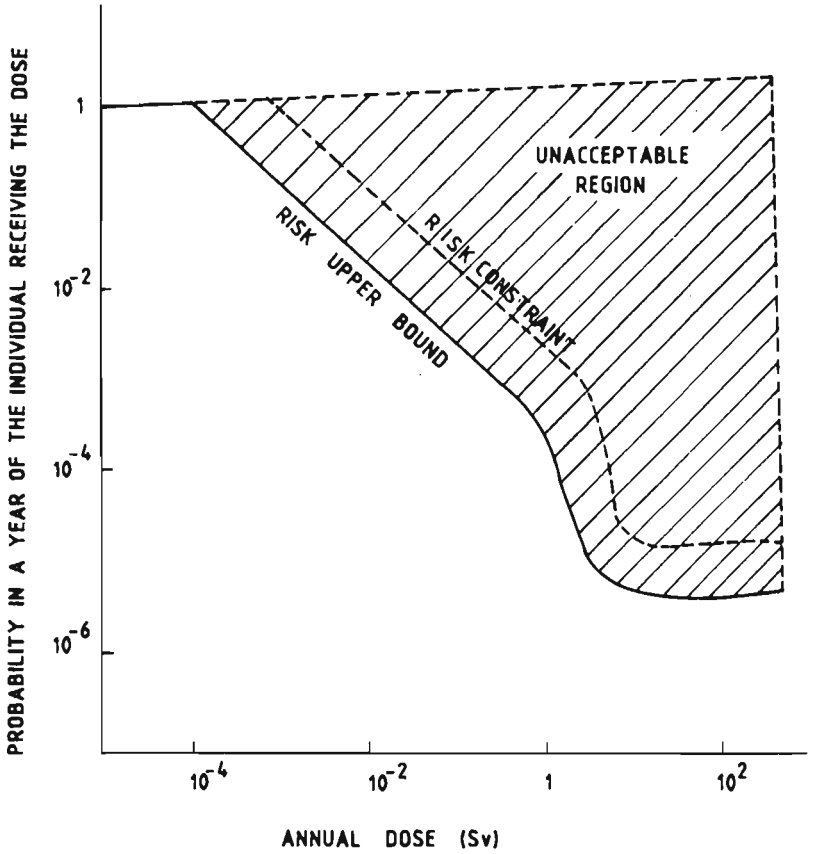

Figure 36.3. Criteria curve corresponding to an annual risk upper bound of 10^{-5} from all events and a corresponding risk constraint (based on ICRP 46).

36.6 OPTIMIZATION OF PROTECTION

Techniques of optimization discussed in ICRP 37 and ICRP 55 would be of use in optimization of waste disposal practices. Optimization of protection can apply at the following levels: Comparison of design alternatives for a waste repository; comparison of different disposal options and different overall management systems for particular waste streams; comparison of complete waste management systems, including conditioning, storage, transport, and disposal alternatives for a given source or practice.

A cost-benefit analysis would, in some cases, be part of the inputs into subsequent wider-ranging alternative waste management systems. Attention needs to be given to the inter-relationship between conditioning, storage, transport, and disposal. Optimization of protection is only one input to the total decision-making process, as discussed in ICRP 37.

36.6.1 Realistic Assessments

Best estimates, rather than conservative assumptions, should be applied for assessment of individual dose distributions, collective dose rates and the probabilities of various radiation impacts. Optimization studies will help to initiate actions that will affect the probabilities as well as to reduce the consequences.

36.6.2 International Aspects

Where there is an international component of the radiation detriment, criteria for protection of populations outside the national borders should be at least as stringent as those applied to the populations of the releasing country. The IAEA (1985) has issued guidance on costing of trans-boundary protection.

36.6.3 Time Scales

In the context of decision-making in choosing of alternatives, only those aspects of the radiation impact that clearly differ are relevant. For many disposal options for the same wastes, the long-term tails of likely collective dose rates beyond a certain time are indistinguishable, and the time integration of collective dose commitments should be truncated at this point to distinguish between options. In some cases truncation may have to be adopted due to large uncertainties associated with the long-term component of the collective dose rates.

On the ethical plane, the question is what weights are to be assigned for future detriment. ICRP has suggested that this is a matter for judgement by national authorities.

36.6.4 Application to Probabilistic Events

Low probability events have a large intrinsic uncertainty around the expectation value of the radiation impact (Beninson and Lindell, 1984).

In such cases the variation coefficient will be inversely proportional to the square root of the probability value. For example, if the probability of the event is taken as 10^{-4}, the variation coefficient of collective dose will be 100 times the expectation value. Therefore, expectation values are not a useful basis for decisions on optimization of protection involving low probability events. Techniques for decision-making under uncertainty (like the use of utility values) are available.

36.7 EXEMPTION RULES

In order that limited resources are not wasted on trivial problems at the cost of neglecting major problems, exemption rules should be established at levels of individual dose or risk, below which there would be no further need for radiation protection concern. As discussed elsewhere, an annual probability of death of the order of 10^{-6} is considered as an acceptable level of risk by the public; this would correspond to an annual dose of the order of 0.1 mSv.

Where an individual could be exposed to different sources, for the source-related assessment, an annual dose exemption level of 0.01 mSv for a particular source would almost certainly ensure that the total dose to the critical group from all sources would be within the value of 0.1 mSv.

Another criterion for exemption of a source concerns the relationship between the efforts and costs to provide data and evaluate the collective dose for optimization purposes and the magnitude of the detriment from the source; this dose is considered by ICRP to be of the order of 1 man-Sv. (The probability of there being even a single resultant serious health effect from a dose of 1 man-Sv is only a few per cent.) Thus a collective dose commitment lower than of the order of 1 man-Sv, made up entirely of annual individual doses less than 0.01 mSv, would be one basis on which a source could be exempted.

On the above basis, wastes below certain levels of total activity and activity concentrations can be exempted from some, or all, of licensing, registration, or notification. The selection of exemption levels and of individual and collective dose criteria are the responsibility of the competent national authority (and, where relevant, on the basis of international agreements).

Exemption from regulation should not be used to make it possible to dispose of large quantities of wastes in diluted form or in divided portions

that may result in violation of protection criteria by the addition of small doses.

36.8 OPERATIONAL ASPECTS

Standard regulations concerning the protection of workers and environmental monitoring would apply during the active phase of the disposal facility. The need for further institutional controls after the closure of the facility should be considered.

36.8.1 Protection of Workers

In the active phase of the disposal facility the situation is similar to that occurring in other parts of the fuel cycle and the system of dose limitation applies. Optimization procedures should take into consideration both occupational and public exposures.

In some circumstances the efforts to reduce the future detriment to the public may increase the detriment to the workers involved. For example, conditioning of waste to reduce leaching rate could involve significant occupational exposures and accident potential.

36.8.2 Monitoring for Protection of the Public during the Pre-closure Period

ICRP publication 43 deals with the principles of monitoring for radiation protection of the population. The objectives in the case of waste disposal should be: (i) to assess potential doses to critical groups and populations; and (ii) to demonstrate compliance with authorized limits and legal requirements. Where normal releases are not likely to be substantial, source monitoring would be minimal.

There is a need for an assessment of the base-line in the case of a repository, as for any other type of nuclear facility.

36.8.3 Institutional and Technical Control during the Post-closure Period

The design of the repository will have to be such that its safety will not depend on institutional control for very long periods after closure. But controls may have to be maintained for a period of time after closure.

Steps like maintenance of registers of land-use data and use of markers and signs round the site are passive means to reduce the probability of human intrusion.

Mill tailings from uranium ore constitute a special type of waste. Some mill tailings left even in shallow land burial are unlikely to remain safe for long periods in the absence of institutional control.

36.9 GENERAL CONCLUSIONS

In the evaluation of alternative waste management systems, interrelationships exist between waste treatment and conditioning processes, storage and transport options, and disposal alternatives. The practical alternatives must be first identified using engineering judgement, common sense, and possibly some rough cost-effectiveness analysis. This will provide an initial screening of options. Radiation protection constraints will then act as one input to the total decision-making process.

ICRP 46 has dealt with the extension of dose limitation to include both normal sequence of events and those that might happen as a result of natural processes or human actions. For the latter case, a risk-based criterion has been established taking into account the probability of the dose being received as well as the probability of serious health effects from the dose. In a comparison of waste management systems, the results of optimization of protection and the estimates of radiation impacts are one input to the total decision-making process which might involve non-radiation protection factors also.

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Part V

Radiation Protection in Medical Exposures

CHAPTER 37

BASIC APPROACH TO RADIATION PROTECTION IN MEDICAL APPLICATIONS

37.1 INTRODUCTION

Several ICRP publications deal directly with protection in the medical applications of radiation. They include: ICRP publication 33, 'Protection against ionizing radiation from external sources used in medicine' (1982) (which replaces ICRP publications 15 and 21, 'Protection against ionizing radiation from external sources' (1976)), ICRP publication 34, 'Protection of the patient in diagnostic radiology' (1982) (which supersedes ICRP publication 16, 'Protection of the patient in X-ray diagnosis' (1970)), ICRP publication 44, 'Protection of the patient in radiation therapy' (1985), ICRP publication 52, 'Protection of the patient in nuclear medicine' (1987) (which supersedes ICRP publication 17, 'Protection of the patient in radionuclide investigations' (1971)), ICRP publication 53, 'Radiation dose to patients from radiopharmaceuticals' (1987), and ICRP publication 57, 'Radiological protection of the worker in medicine and dentistry' (1989) [which includes updating of ICRP publication 25, 'Handling, use and disposal of unsealed radionuclides in hospitals and medical research establishments' (1977)]. ICRP publication 60 briefly summarizes the basic philosophy and approach towards protection in medical applications.

ICRP publication 62, 'Radiological protection in biomedical research' (1992) deals in detail with the ethical considerations related to biomedical research involving radiation exposure of humans (patients or volunteers) and gives guidelines governing the planning of such projects; the contents of this report are summarized in Appendix 37.1. ICRP 62 also contains an addendum to ICRP 53, where effective doses to normal adults from the radiopharmaceuticals listed in ICRP 53 have been recalculated using the revised tissue weighting factors recommended by ICRP 60, as well as detailed biokinetic and dosimetric data for some new radiopharmaceuticals; in addition it has an updated summary of current ICRP principles for protection of the patient in diagnostic radiology, taking into account the ICRP 60 recommendations.

In the context of medical applications, protection involves three aspects, viz. the occupational workers, the patients, and members of the public (including relatives of the patients). The major medical applications of radiation are: diagnostic radiology, radiotherapy, and nuclear medicine (both diagnostic and therapeutic). Medical research involving irradiation of volunteers also comes under this category. 'Balneotherapy' is a related subject of limited relevance in the general context; here, natural spas containing high quantities of radon and daughter products in water are used in the treatment of non-malignant diseases.

We shall first summarize the basic approach enunciated in ICRP 60 and subsequently deal with the detailed recommendations for various applications. It is worth emphasizing that, by and large, the principles and techniques of protection are very similar, whether it is for the worker, patient or a member of the public. It may be pointed out that almost all the specific recommendations on the subject have been published earlier to ICRP 60, and may therefore need some modifications. But the basic approach has remained the same; only in matters of detail changes will have to be made.

In these reports, the word 'shall' indicates that which is necessary and essential for protection, and the word 'should' indicates that which is considered to be desirable and intended to apply, whenever reasonable, in the interests of improving protection.

37.2 BASIC APPROACH OF ICRP 60

37.2.1 Medical Exposure

'Medical exposure' is confined to exposures incurred by individuals as part of their own medical diagnosis or treatment and to exposures (other than occupational) incurred knowingly and willingly by individuals helping in the support and comfort of patients undergoing diagnosis or treatment. Exposure of an individual to other sources, such as stray radiation from the diagnosis or treatment of other persons, is not included in medical exposure, nor is any occupational exposure of staff. Exposures incurred by volunteers as part of a programme of biomedical research are also dealt with on the same basis as medical exposure.

37.2.2 The System of Protection in Medical Exposure

37.2.2.1 The Justification of a Practice in Medical Exposure

The justification of a practice leading to medical exposures should be dealt with in the same way as the justification of any other practice. Most of the benefits and detriment accrue to the individuals undergoing diagnosis or treatment, but account should be taken of all the resulting exposures, including the occupational and public exposures, and of any potential exposures. In the first instance, the practice should be defined in broad terms. However, each procedure, either diagnostic or therapeutic, is subject to a separate decision, so that there is an opportunity to apply a further, case-by-case, justification for each procedure. This will not be necessary for simple diagnostic procedures based on common indications, but may be important for complex investigations and for therapy.

37.2.2.2 The Optimization of Protection in Medical Exposure

Because most procedures causing medical exposures are clearly justified and since the procedures are usually for the direct benefit of the exposed individual, less attention has been given to optimization in this area compared to other applications of radiation. As a result, there is considerable scope for dose reduction in diagnostic radiology. Optimization should be exercised in planning new installations. Simple, low cost, measures are available for reducing doses without loss of diagnostic information, but the extent to which these measures are used varies widely. Doses from similar investigations cover ranges of as much as two orders of magnitude. Consideration should be given to the use of dose constraints, or investigation levels, selected by the appropriate professional or regulatory agency, for application in some common diagnostic procedures. They should be applied with flexibility to allow higher doses where indicated by sound clinical judgement.

37.2.2.3 Dose Limits in Medical Exposure

Medical procedures are usually intended to provide a direct benefit to the exposed individual. If the practice is justified and the protection optimized, the dose to the patient will be as low as compatible with the medical purposes. Any further application of limits will be to the patient's detriment. ICRP therefore recommends that dose limits should not be applied to medical exposures.

It is also not appropriate to include the doses incurred by patients in the course of diagnostic examinations or therapy when considering compliance with dose limits applied to occupational or public exposure.

37.2.2.4 Medical Exposure of Pregnant Women

As discussed elsewhere, exposure of the embryo in the first 3 weeks following conception is not likely to result in deterministic or stochastic effects in the liveborn child. The necessary information on possible pregnancy should be obtained from the patient herself. If the most recent expected menstruation has been missed, and there is no other relevant information, the woman should be assumed to be pregnant. Diagnostic or therapeutic procedures causing exposures of women likely to be pregnant should be avoided unless there are strong clinical indications.

37.2.2.5 Dosimetry in Medical Exposure

The assessment of patient doses in medical exposure is of critical importance in radiotherapy and is dealt with by the ICRU (International Commission on Radiation Units and Measurements). Frequent measurements on equipment should form an important part of the quality control programme. In diagnostic radiology, there is rarely a need for routine assessment of doses, but periodic measurements should be made to check the performance of equipment and to encourage the optimization of protection. In nuclear medicine, the administered activity should always be recorded and the doses, based on standard models, will then be readily available.

APPENDIX 37.1

RADIOLOGICAL PROTECTION IN BIOMEDICAL RESEARCH

INTRODUCTION

Guidance regarding biomedical research involving human beings has been provided by the Declaration of Helsinki (adopted by the World Medical Assembly in 1964 at Helsinki and augmented by the Assembly in Tokyo in 1975) (WMA, 1975). (The Declaration only gives guidance and doctors are not relieved from medico-legal or ethical responsibilities under the laws of their own countries.)

The World Health Organization published a Technical Report in 1977 (WHO,1977) giving specific recommendations on the use of ionizing radiations on human beings for biomedical research, based on the general philosophy of the Declaration of Helsinki. ICRP 60 also touched on the topic. ICRP publication 62, 'Radiological protection in biomedical research' (1991) has elaborated on the subject.

BASIC FEATURES OF THE HELSINKI DECLARATION

The purpose of biomedical research involving human subjects must be to improve diagnostic, therapeutic and prophylactic procedures and the understanding of the etiology and pathogenesis of diseases. Such research procedures may involve hazards.

A distinction must be recognized between medical research in which the aim is essentially diagnostic or therapeutic for a patient, and medical research, the object of which is purely scientific and without direct diagnostic or therapeutic value to the person subjected to the research. Such research should also ensure that the environment is not affected.

Basic Principles

Biomedical research involving human subjects must conform to generally accepted scientific principles and should be based on adequately

performed laboratory and animal experimentation and on a thorough knowledge of the literature. The design of each procedure should be clearly formulated in an experimental protocol which should be submitted to a specifically appointed independent committee (the Ethics Committee) for evaluation. Studies should be conducted by qualified personnel and under the influence of a medical person on whom the responsibility of the subject must always rest. Confidentiality of information must be observed. The research protocol should contain a statement of the ethical considerations involved and should indicate that the principles of the Helsinki Declaration are complied with.

The importance of the objective should be in proportion to the risk to the subject, both of which should be carefully assessed beforehand. The hazards involved should be predictable. Investigations should cease if the hazards are found to outweigh the potential benefits. Concern for the interest of the subject must prevail over the interest of science and society.

Each potential subject should be adequately informed of the aims, methods, anticipated benefits and potential hazards of the study. He or she should be informed that he/she is at liberty to abstain from the study, or withdraw from it at any time. The doctor should then obtain the subject's freely-given, informed consent, preferably in writing.

The doctor should be particularly cautious if the subject is in a dependent relation to him or may consent under duress (e.g., employee, student, or even a patient). In such a case, the informed consent should be obtained by a doctor who is not engaged in the investigation. In the case of a legal incompetence (e.g., physical or mental incapacity, minors) informed consent should be obtained from the legal guardian in accordance with national legislation.

[Author's note: Monetary payments for out-of-pocket expenses are not to be regarded as an inducement; WHO (1977).]

PROTECTION IN BIOMEDICAL RESEARCH INVOLVING RADIATION EXPOSURE OF HUMAN BEINGS

Biomedical research involving radiation exposure of human beings, as in other aspects of biomedical research, is a necessary step in the introduction of new diagnostic and therapeutic procedures. The application of basic and clinical knowledge to patients requires knowledge of similar data in healthy individuals. In the case of exposure for research purposes with volunteers, the potential benefit to society, by increase of knowledge, must be weighed against the potential harm to the exposed individual.

ICRP 62 aims to provide advice to individuals planning such research, regulatory bodies, those engaged in evaluation of research projects, and the subjects themselves (patients, volunteers). The Helsinki Declaration provides the firm ethical basis for decisions.

Pregnant women should not be asked to take part in research projects involving irradiation of the fetus unless the pregnancy itself is central to the research, and then only if other techniques involving less risk cannot be used. In the case of children, the benefits of the proposed study to society should be obviously advantageous and substantially exceed the risk to the subject.

Risk Assessment

We have now adequate knowledge of the biological effects of radiation to make reasonable estimates of the risk. (ICRP 62 has a section summarizing the nature, types and magnitude of radiation risks.) To use this information a reliable assessment of the dose is necessary (average absorbed dose to the organ, equivalent dose and effective dose). The effective dose can serve as an approximate indicator of radiation detriment to the individual.

For new diagnostic radiopharmaceuticals, detailed studies of biokinetic behaviour in animals should be done, supplemented by investigations on a small number of volunteers, for reliable dosimetry. In the case of new radiopharmaceuticals for therapy, results of animal experiments must be checked against human data from volunteers, collected at low administered activity (in the diagnostic range); only then can the dosimetry be reliable and also give an indication of the possibility of deterministic effects. In cases of uncertainty, dosimetry should be conservative (leading to overestimate of the dose).

Principles of Research Design

Consideration should first be given as to whether it would be possible to obtain equivalent information by other methods not involving radiation (e.g., ultrasound, MRI). From among alternative methods using radiation the procedure delivering the lowest dose should be selected.

Procedures involving radiation exposure should conform to the general and specific recommendations provided by ICRP in its various publications connected with protection in the medical uses of radiation in the

fields of diagnostic radiology, radiotherapy and nuclear medicine. The number of individuals studied should be the lowest compatible with obtaining an unequivocal answer to the tested hypothesis.

Project Evaluation

To assist the Ethics Committees in their evaluation, projects can be divided into categories depending on the radiation dose. The basic criterion is the level of risk, which can be expressed as the total detriment from the exposure (probability of fatal cancers, weighted probability of non-fatal cancers, and probability of serious hereditary effects in succeeding generations). The risk can be transferred for normal average adults to a level of dose. For children the detriment per unit dose is 2-3 times larger than for adults, while for people above 50 the risk is one-fifth to one-tenth of that for younger generations; for those suffering from serious (possible terminal) disease the likely expression of radiation-induced risk is even lower.

The risk categories and associated information are given in Table APP 37.1.1. The lowest risk category is of the order of 1 in a million (generally considered trivial); the corresponding dose is less than 100 μSv (equivalent to the dose from natural background radiation in a few weeks, and considerably less than the variation in annual natural background dose to persons living in different locations). In this case, if the investigation is justified, the level of benefit needed as the basis for approval would be minor and would include those investigations expected only to increase knowledge.

At the other extreme, the highest risk category includes risks of the order of 1 in 1000, corresponding to doses of tens of mSv, which is greater than the current annual dose limit for occupational exposure. To justify investigations in this category the benefits would have to be substantial, and usually directly related to the saving of life or the prevention or mitigation of disease; even here the doses should be kept below those for deterministic effects (except for therapy).

Between these two there is a category in which the risk is neither trivial nor completely unacceptable (1 in 10,000 to 1 in 100,000). The balance between benefit and risk is difficult in this category. It may be helpful to divide this category into two sub-categories. Category IIa would comprise a comparatively minor level of risk (corresponding to dose limits to the public from controlled sources), and would correspond to a level of intermediate benefit, including increase in knowledge leading to health

benefit. For Category IIb (corresponding to annual doses typically received by occupational workers) the benefit will be more directly aimed at the cure or prevention of disease.

The research protocol should specify and the Ethics Committee should ensure that the same individual does not participate repeatedly in such investigations.

Procedures for Project Evaluation and Responsibilties

The responsibilities for those proposing the project and those approving it should be separated.

The research team proposing the project should include persons with appropriate qualifications and experience. Medical supervision of health of subjects should be ensured. In addition to the medical investigator/s, there should be a medical physicist who performs dosimetry, and, where required, a radiopharmacist. Competence for statistical evaluation of data should be available.

The competent authority of a country should create a framework for the formation of and providing guidelines to Ethics Committees. The Ethics Committee should be independent of the project investigators and comprise experts (mostly outside the institution proposing the project) in the concerned fields including radiological protection. Legal advice should be available. A negative opinion of the Committee should normally be binding unless there is a procedure for appeal to a higher body. The Committee should keep records of its activities and should evaluate periodically the progress reports on the project. Confidentiality of medical information must be observed.

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WMA (1975). World Medical Assembly: Declaration of Helsinki. Recommendations guiding medical doctors in biomedical research involving human subjects. Adopted by the 18th World Medical Assembly, Helsinki, Finland (1964); and revised by the 25th World Medical Assembly, Tokyo, Japan.

Table APP 37.1.1: Categories of risk and corresponding benefit
(from ICRP 62)

Level of risk	Risk category	Total risk	Corresponding effective dose range (adults) (mSv)	Level of societal benefit
Trivial	I	$< 10^{-6}$	< 0.1	Minor
Minor to intermediate	II			Intermediate to moderate
	IIa	~ 10^{-5}	0.1-1	
	IIb	~ 10^{-4}	1-10	
Moderate	III	$> 10^{-3}$	> 10*	Substantial

* To be kept below determinsitic thresholds except for therapeutic experiments

CHAPTER 38

GENERAL ASPECTS OF THE RADIATION PROTECTION PROGRAMME IN MEDICAL APPLICATIONS

38.1 THE CONTROL OF RADIATION HAZARDS

38.1.1 External Radiation

In diagnostic radiology and teletherapy the only hazard is, under normal working conditions, from sources outside the body with no possibility of internal contamination. The control of external exposure requires the use of suitable equipment and techniques. The most effective protection measures are those applied at or near the source (X-ray machine or teletherapy source), and the shielding of the primary source is of major importance. When the source is in use, there will be secondary sources of exposure due to scattering of the primary radiation by the patient and by other materials in the vicinity. Local shielding should be provided between the sources, both primary and secondary, and the workers (provided that this is not detrimental to the patient). The use of an interlocking system also provides extra protection against inadvertent exposure. Further measures include limiting the time spent by workers in the vicinity of primary and secondary sources, and by wearing protective clothing. Technical data pertaining to these problems are given in ICRP 33.

38.1.2 Control of Contamination

When dealing with unsealed radionuclides, the problems of external radiation are at least as important as when dealing with sealed sources, but they have to be supplemented by the control of contamination. The principal aims are to contain the radioactive materials at or near the point of use and to achieve a high standard of cleanliness so that contamination of the workplaces or the worker does not build up.

38.2 OVERALL ORGANIZATION

The management in each establishment has an overall responsibility for radiation safety. Within the management structure, there should be a clear chain of responsibility down to the worker. Protection depends upon the individual worker observing safety procedures. These should be normally in the form of written instructions.

In each facility a qualified/trained person should be appointed or designated to act as Radiation Safety Officer (RSO). His responsibilities include: preparation of written safety instructions, day-today attention to protection, coordination with the management authorities, emergency preparedness and management, training of workers in radiation safety, consultancy, and maintenance of appropriate records.

The management should have access to expert advice in radiation protection, particularly in the areas like: planning and design; performance specifications for equipment; organization of a Quality Assurance Programme; identification of 'controlled' and 'supervised' areas; drawing of safe working procedures and monitoring of their effectiveness; potential emergencies and their management; and management of actual/suspected high exposures of workers. This advice may be available from the Radiation Safety Officer or a Radiation Protection Adviser who may be a member of the staff or an external consultant. In large institutions a Radiation Protection Committee (in which the RSO will be a member) for policy decisions may be useful.

38.3 EDUCATION AND TRAINING

Any person involved in the administration of radiation to patients must have a level of specialized training appropriate to the task performed. Regulatory authorities may wish to define the appropriate level of training required. Initial training and continued education are necessary for all radiation workers. Concerned physicians should have a thorough basic education in radiation protection (including radiation biology, dosimetry and radiation physics) together with a detailed knowledge within their own professional spheres. Other practioners involved in radiological procedures, particularly cardiologists and orthopedic surgeons, should receive special training in radiation protection. Medical physicists should receive comprehensive training in theoretical aspects and practical training in radiation protection; a knowledge of basic human anatomy, physiology and radiation biology is required. Technicians should undergo a

course of study which includes radiation physics, radiation biology and radiation protection. Training of nurses who are to work in the department or attached to wards should include relevant information concerning the principles of radiation protection and practical aspects involved in nursing therapy patients. The patients should be given a balanced view to put radiation risks in perspective so that they accept without undue concern any diagnostic examination that is clearly indicated clinically.

38.4 PLANNING AND DESIGN

Before construction of the facility, the final plans should be reviewed by the Radiation Protection Adviser. After construction, it should be ensured that the design specifications have been incorporated. Copies of the plans should be readily available at site. Attention has to be given to design of primary shields, as well as secondary shielding against primary beams, leakage and scattered radiation. Planning must aim at the ALARA principle and optimization.

Adequate protection may be achieved by controlling the distance of workers from radiation sources, shielding, and duration of exposure. In the design, account should be taken of the maximum workload as well as occupancy of surrounding areas by workers, patients, and members of the public. 'Occupancy factors' and 'use factors' should be chosen judiciously. As a rule, subsequent alterations to shielding requirements are more difficult and expensive to incorporate.

Windows and doors in walls should also provide as much shielding as the walls themselves. Concrete must be homogeneous and of specified composition and density. Shielding should not be impaired at joints. Rooms where radiation work is undertaken should not be used as thoroughfares to gain access to other areas.

38.5 CLASSIFICATION OF AREAS

ICRP 57, in conformity with earlier convention, makes a distinction between Working Condition A (where the annual exposure might exceed three-tenths of the dose limit and where individual monitoring is essential), and Working Condition B (where it is most unlikely that the exposures would exceed three-tenths of the dose limit and where individual monitoring, although not necessary, may be - and often is - carried out in order to confirm that the working environment is satisfactory). A 'Controlled Area' is one where Working Condition A prevails, and a 'Supervised Area'

is one where Working Condition B prevails. It has been pointed out earlier that ICRP 60, while retaining the classification into controlled and supervised areas, has done away with the distinction between working conditions A and B. ICRP 57 points out that in medical applications workers rarely exceed one-tenth of the dose limit and there may be no practical advantage in introducing a Supervised Area in addition to a Controlled Area. The boundary of the Controlled Area shall be indicated by a warning sign and access to it limited. To avoid uncertainties, the boundaries should, when possible, be walls, doors, etc.

The revised (ICRP 60) recommendations for the occupational exposure of women have been discussed earlier in Chapters 5 and 8.

38.6 RADIATION MONITORING PROGRAMME

There should be a pre-operational survey of the facility to ensure that the planned safety requirements have been met. Subsequent surveys should be performed after any major modification. In addition, a programme of monitoring should be introduced to check that conditions continue to remain satisfactory. All radiation measuring devices should be regularly checked for consistency of performance. Records of the results of environmental and individual monitoring should be retained. Whenever monitoring indicates an increase in level of exposure above normal, the circumstances should be investigated.

38.6.1 Individual Monitoring

ICRP 57 points out that the majority of medical radiation workers fall under Working Condition B, where individual monitoring, while not necessary, is usually carried out. A minority of workers may require to be categorized as working in Condition A; this includes interventional radiologists and cardiologists, workers dispensing and administering unsealed radionuclides, and workers associated with brachytherapy or nuclear medicine therapy patients. As stated earlier, ICRP 60 has recommended that the decision to provide individual monitoring to a group of workers should be taken by the operating management, but should be subject to review by the regulatory agency. However, the criteria stipulated in ICRP 57 still hold good.

The personal dosimeter should be worn in a position representing the most highly exposed part of the surface of the trunk. In special situations, where protective clothing provides a significant attenuation of the incident radiation, the doses to unprotected parts of the body may make a

considerable contribution to the effective dose. In these circumstances, if a single dosimeter is used it should be outside the protective clothing, usually high on the trunk; this will normally overestimate the effective dose. Where appropriate, two dosimeters, one beneath and one outside the protective clothing, may be worn. In special cases finger dosimeters or forehead dosimeters (for assessment of eye doses) may be desirable.

38.6.2 Environmental Monitoring of the Workplace

Monitoring may be of routine, operational or special type. Routine monitoring is associated with continuous operations. Operational monitoring is performed to get information about a particular procedure. Special monitoring is applied to an actual or potential abnormal situation. Routine monitoring of the workplace should be carried out to confirm that the working environment is satisfactory for continued operations.

38.7 MEDICAL SURVEILLANCE

The occupational physician supervising the health of a group of occupational workers needs to be familiar with the tasks and working conditions of the worker. A routine medical examination of a radiation worker is of no value other than to establish the general level of health of the worker. With the present system of dose limitation no special arrangement (particularly with respect to working hours and length of vacation) is appropriate for workers as far as radiation work is concerned.

When it is suspected that a worker may have received an abnormally high exposure, a detailed assessment (including biological dosimetry by chromosomal aberration studies) may be necessary. If the worker has any clinical sign of radiation damage, prompt referral to a specialized centre becomes essential.

CHAPTER 39

RADIATION PROTECTION IN DIAGNOSTIC RADIOLOGY

39.1 INTRODUCTION

The present chapter is based on ICRP publications 33, 34 and 57 (the last, in addition to dealing with protection of the worker, includes a section on 'Summary of the current principles for protection of the patient in diagnostic radiology'), and ICRP 62 which contains an updated version of the summary given in ICRP 57 taking into account ICRP 60 recommendations.

The degree of safety in diagnostic radiology is now high and an X-ray examination, recommended on the basis of clinical judgement of a qualified physician, generally brings to the patient a benefit that outweighs the unavoidable radiation risk.

The largest part of human exposure to man-made radiation comes from diagnostic radiology (a few per cent of the dose from natural background radiation in developing countries to substantially higher percentages, around 20-50%, in developed countries) (UNSCEAR, 1977). Consequently, it is highly desirable to discontinue those X-ray examinations that are not expected to contribute materially to establishing a proper diagnosis and to minimize doses in the course of beneficial examinations without loss of diagnostic information.

Some diagnostic X-ray equipment, particularly fluoroscopic equipment, when obsolete or improperly operated, is capable of delivering doses that may be high enough to produce cellular reaction seen as acute tissue damage. However, in properly conducted diagnostic examinations, these acute effects do not occur because the doses are well below the threshold for such effects.

39.2 CLINICAL JUDGEMENT AND ADMINISTRATIVE PRACTICES

The purpose of an X-ray examination is (a) confirmation or elimination of a suspected condition, and/or (b) discovery of an unexpected condition.

39.2.1 Responsibilities of the Referring Physician, Radiologist and Radiographer

The decision as to whether an X-ray examination is justified (whether taken by the referring physician or the radiologist) must be based on a correct assessment of the indications for the examination, the expected diagnostic yield, and the way in which the results are likely to influence the diagnosis and subsequent management of the patient. This assessment should be made against a background of knowledge of the physical properties and biological effects of radiation. Methods of making evaluations of the cost-benefit aspects of diagnostic procedures are improving, and new techniques have been developed to improve efficacy and decision-making. Retrospective analysis of the correctness of the decisions will refine the indications and non-indications for future patients.

The referring physician's understanding of the benefits and risks of X-ray diagnosis is often incomplete. Before prescribing an X-ray examination he should be satisfied that the necessary information is not available, either from an earlier X-ray examination or from other tests. He should provide a clear request describing the patient's problem and indicating the clinical objectives, so that the radiologist can carry out the correct examination.

To achieve the necessary overall clinical judgement the radiologist may need to consult the referring physician. This practice is to be encouraged. The radiologist has the responsibility for the control of all aspects of the conduct of the X-ray examinations, including the technique to be used. The sequence in which the examinations are to be conducted should be determined in each case. The results of each examination in the sequence should be assessed before the next one is performed.

The radiographer carries out the X-ray examinations under the supervision of the radiologist. He is in a key position regarding the amount of radiation administered, the optimal use of imaging equipment and the recognition of equipment malfunctions. Where the physician or dentist performs his own radiological procedures, this could result in unnecessary examinations being performed.

Three major developments in diagnostic imaging have occurred in recent times, viz., computed tomography, ultrasonic techniques, and nuclear medicine procedures. Ultrasonic diagnosis, which appears to have minimal or no risk, provides information without the potential hazards of diagnostic doses of ionizing radiation. The radiologist should have enough knowledge of the relative merits of these techniques to decide upon the most appropriate technique in any particular case.

It is desirable that all radiological equipment and procedures be placed under the control of the chief radiologist, even though some equipment may not be located in the radiology department.

Education in protection should form part of the educational programme of radiologists and radiographers. There should be short courses to demonstrate radiation safety organized by radiologists and radiological physicists for medical students. Concepts of efficacy and safety should be interwoven in clinical radiological presentations. National bodies should give consideration to evolving programmes for training and continuing education of radiographers.

In developing countries it may not be possible to employ a trained radiographer or a qualified radiologist in many places where X-ray services are required. In such cases there should be arrangements for peripheral units (e.g., at the level of a health centre) to get advice from higher level radiological centres nearby.

When a patient is transferred from one hospital to another, radiographs and copies of records should be made available when practicable.

39.3 PLANNING AND DESIGN

Physicians and ancillary staff should have received appropriate training and been authorized by the national regulatory authority. Patients awaiting radiological procedures shall wait outside the X-ray room. Regarding the technical aspects, in each X-ray room there is normally a shielded area (protective cubicle) where the workers stand and where the control console of the X-ray machine is located. X-rays of 50-200 kVp are attenuated a thousand-fold by shields of lead equivalent thickness of a fraction of 1-2 mm. In non-fluoroscopic investigations the exposure switch should be so mounted that it is impossible to make an exposure from outside the protective cubicle.

If in special cases (e.g., rapid film changing, angiography) it is not possible to retreat behind the protective screen, the room size should allow for additional protective barriers and adequate distance of the operator from the X-ray tube. The degree of shielding will depend on: direction of the primary beam, anticipated secondary/leakage radiation, workload and type of examination, and occupancy of X-ray room (present and future).

In rooms where fluoroscopy is undertaken, some workers will be outside the shielded area, and design should allow for additional shielding (like protective drapes suspended from the ceiling). The workers should also be wearing protective clothing.

The protective screen at the control console should contain lead glass windows of the same lead equivalence as the screen to enable a clear view of the patient and of any other worker outside the shielded area.

ICRP 33 has an extensive appendix containing data on output of X-ray generators and gamma sources, scattering of X- and gamma rays, broad beam transmission through shields of various materials, half value and tenth value layer thicknesses, transmission of scattered and leakage radiation through shields, and range-energy curves for electrons. It also provides some indication of the cost of sheilding and has an illustration of how the optimization process can be applied to the design of sheilding. ICRP 57 has also an appendix giving information of a related nature, including dose rates in fluoroscopy rooms. Table 39.1 (based on ICRP 33) gives a very condensed summary of HVLs for broad beam geometry.

The primary beam should not be directed towards the protective cubicle or any entrance to the room. The natural boundaries of the room like walls, floors and ceiling should provide protection in adjacent areas. The area within the boundaries will normally be considered as a Controlled Area. (In the case of mobile/portable equipment it may be difficult to demarcate the Controlled Area, and the need for additional temporary shielding should be evaluated.) Secondary and leakage radiation can be reduced by proper design of X-ray beam collimators and tube housings. Doors and door frames, as well as wall penetrations for ductwork should have appropriate shielding. Entrance to X-ray rooms should be wide enough for beds to pass through.

A radiation warning sign should be prominently displayed at all entrances to the X-ray room. At the main entrance (particularly in fluoroscopy rooms) a warning light should be installed at eye level and should indicate that the X-ray machine is on or about to be on. An interlocking system will provide extra protection to prevent entry when fluoroscopy is

on. An indication that X-rays are being generated shall be provided at the control panel. When there is more than one X-ray tube capable of being selected from a single location (an undesirable practice, to be eliminated wherever possible), a warning light should indicate which tube has been selected. The patient shall be observable from the control location. Means shall be provided on the control panel to indicate the tube potential, current and exposure time; alternatively, a meter to indicate the product of current and time (mAs-meter) may be used.

Provision for absorbing the primary radiation (after it has passed through the patient) and scattered radiation should be as close to the patient as possible.

Wherever possible, X-ray examinations should be conducted in the X-ray department, and mobile X-ray examinations in wards and operating theatre should be reduced to a minimum.

Access to controlled areas should be restricted only to the authorized persons. Workers should not expose parts of their body to the primary beam. If workers cannot remain in the protective area when the machine is operated, they shall wear a protective apron of at least 0.25 mm lead equivalence. The minimum distance of the operator from the tube and the patient shall be 2 metres for mobile equipment. No person shall normally hold the patient during diagnostic examinations. Where it is unavoidable, the person holding the patient should wear a protective apron (of at least 0.25 mm lead equivalent) and, in special cases, gloves (of 0.35 mm lead equivalent). The patient should not be held by the worker or a person below 18 years of age or a pregnant woman. Immobilization devices should be used for children and old patients. Care should be taken to ensure that those awaiting examinations are adequately protected.

A quality assurance programme should be implemented that applies to all the major components of the total system (including acceptance tests on new equipment). Equipment should be used only when it has been checked that it is functioning correctly. Equipment that does not meet standards should not normally be used against the advice of experts. No equipment should normally be used for purposes other than that for which it has been designed.

39.4 TECHNICAL AND PHYSICAL FACTORS

39.4.1 General: Avoidance of Unnecessary Doses

(a) Reduce the doses to the organs examined to the minimum compatible with the necessary diagnostic information;

(b) Limit irradiation of other parts of the body; and

(c) Reduce the frequency of unnecessary repeat examinations.

39.4.2 X-ray Tube Housing

Every X-ray tube shall be enclosed in a housing such that the air kerma from the leakage radiation at 1 m from the focus does not exceed 1 mGy/h at every tube rating.

39.4.3 Field Size

The smallest practicable field should be used and it should be accurately positioned on the patient. Decrease in field size reduces patient dose, reduces scatter radiation reaching the image receptor, and improves image quality. Ideally, the edges of the beam should be seen on the film.

An adjustable rectangular collimator (specially a multi-leaf diaphragm with light indication) is better than a circular one. Means should be provided to verify that the film is aligned with the beam. Non-adjustable collimators should be marked with the appropriate field size.

The gonads should be kept outside the direct beam. Beam limiting devices are available which automatically restrict the size of the beam to the size of the cassette. But when the cassette is larger than the area of interest, further beam limiting should be done; this is particularly important for children, where total body irradiation can otherwise result even from a chest radiograph.

39.4.4 Shielding of Organs

In most radiographic examinations of the abdomen, male gonads can be shielded; in the female this is not possible since the gonadal area is often diagnostically important as it contains structures like ureters and colon. Gonad shielding reduces dose to testes by 95% and dose to ovaries by 50% when the gonads are in the primary beam. Eye shields reduce eye doses by 50-75%; PA projection reduces eye doses by 95% compared to the AP view. Thyroid protection, if deemed necessary, can be achieved by a lead equivalent collar or a protective apron with a high neck line. In dental radiography the primary beam should be directed away from the gonads.

39.4.5 Distance of the Focal Spot to the Skin (FSD) or Image Receptor

For the same radiation intensity at the image plane the entry dose at the patient surface increases sharply with decreasing FSD. Moreover, for focal spot-to-image receptor distances of less than 1 m, the quality of the diagnostic information becomes poorer as this distance becomes shorter. On both these counts, it is desirable to have long focal spot-to-image receptor distances. For stationary radiography and fluoroscopy equipment the FSD should not be less than 45 cm and shall not be less than 30 cm. In mobile radiography/fluoroscopy equipment, the FSD should not be less than 30 cm. Photofluoroscopy and radiography of the chest should be performed with focal spot-to-image receptor distance of at least 120 cm.

39.4.6 Tube Voltage

The higher the tube voltage, the more penetrating the radiation and the less the surface dose. But the contrast between soft tissue and bone decreases and more scattered radiation reaches organs outside the primary beam. Increases in peak voltages up to 100 kV give substantial reduction of skin dose for a constant exit dose.

39.4.7 Voltage Waveform

Three phase supplies and higher frequency generators produce less ripple (which generate a higher proportion of radiation of lower penetration) than single phase supplies and hence lower doses.

39.4.8 Filtration

Filtration preferentially attenuates unwanted components of the beam (usually those with lower energy) which otherwise would be absorbed by the patient and provide little diagnostic information. The use of filters results in a more penetrating beam and lower skin doses. Total filtration in the useful beam shall be equivalent to not less than 2.5 mm Al equivalent, of which 1.5 mm should be permanent. For dental equipment and X-rays below 70 kV, the total permanent filtration shall be not less than 1.5 mm Al. (For mammography see later.) The aluminium equivalent of the table top or front panel of the vertical cassette holder shall not be more than 1 mm Al at 100 kV.

39.4.9 Carbon Fibre Materials

The use of carbon fibre materials for the patient support, in anti-scatter grids and for the cassette face allows transmission of a larger proportion of the beam. Dose reduction can be 30-50%.

39.4.10 Control of Irradiation and Recording Time

A device shall be provided to terminate the exposure after a pre-set time or exposure. Switches should be such that irradiation can be terminated manually at any time, and shall be so arranged that accidental exposure is unlikely. Timers should be checked periodically, particularly in mobile and conventional dental radiographic equipment. Details regarding control of timings in fluoroscopy are given later.

39.4.11 Control of Scatter to the Recording System

Reduction of scatter enhances image quality. This is done by grids (parallel or focused) interposed between the patient and the recording device. Since grids absorb some radiation leaving the patient, their use increases exposure factors and the patient dose. Care is needed to ensure that the X-ray focus is properly aligned with the grid; otherwise a retake may be necessary. In fluoroscopy and in radiography of infants the use of grids is not necessary and doses can be decreased by a factor of 2-4. An air gap can be used in place of the grid, particularly in chest radiography. Moving slit radiography gives better radiographic contrast than with a grid and patient dose is less.

39.4.12 Intensifying Screens and Radiographic Films

Intensifying screens containing high efficiency phosphorescent materials like rare earths, barium or tantalum require less radiation than conventional intensifying screens to produce radiographs of the same quality; they are faster than calcium tungstate films but have the same resolution. Non-screen radiographic films have no place in diagnostic radiology.

39.4.13 Film Processing

Processing techniques are important. Over-exposure and under-development of films is a common undesirable procedure leading to over-exposure of the patient and also a poor quality film. Periodic checks

should be made that the dark room is light-tight and that correct safe lights are used.

In manual processing, correct developer, fixer and processing temperature should be determined. Developer must be replenished and replaced at regular intervals. Adequate fixing, washing and hardening are necessary for a permanent record. Drying should be carried out in a special cabinet. Routine control of film density by inspection during development is strongly deprecated.

Automatic processing is preferred to manual processing, provided that the equipment is properly operated and maintained. Quality control is particularly important in this case. Control is readily done by the daily use of film strips exposed to a sensitometer shortly before processing and evaluation by a densitometer.

39.4.14 Reduction in Repeat Exposures

Rate of retake of radiographs varies from 3 to 15%. Major causes are either errors in patient positioning or radiographs that are either too light or too dark. Use of a reference list of technical factors (kVp, mAs) or automatic control, as well as exposure tables based on patient size, is recommended. Suitable preparation of the patient is necessary in some examinations such as excretory urogram or barium enema. There should be a regular review of rejected films aimed at detection and elimination of causes for rejection.

39.4.15 Fluoroscopy

Fluoroscopy gives 10 times the dose compared to radiography. Perception of contrast and details is poor. Doses to workers can also be high, particularly in interventional radiology. Fluoroscopy should only be done if the information cannot be obtained by radiography. It should be used primarily to study dynamic phenomena rather than to evaluate anatomical detail. The dose rate at the entrance surface of the patient should be much less than 50 mGy/min. Dark adaptation of the eyes by the radiologist (for at least 10 minutes) and use of sensitive fluorescent screens are recommended.

Wherever practicable, fluoroscopy should be performed with image intensifiers. Fluoroscopy should not be performed with mobile equipment unless an image intensifier is employed. Image intensification reduces doses three-fold.

An adjustable collimator shall be provided to define the useful beam. The X-ray tube, adjustable beam-limiting device and fluoroscopic screen or image intensifier should be linked together in such a way that the beam will not fall outside the screen. In case of failure, an indicator should warn the operator.

The fluorescent screen shall be covered with a protective glass sheet having a lead equivalent of not less than 1.5 mm for tube voltages up to 70 kV, 2.0 mm for 70-100 kV, and an additional 0.01 mm per kV above 100 kV.

It is valuable to record the whole of a fluoroscopic examination in a video recorder, where the image can be viewed several times without repeating the examination.

The operator should be aware of the exposure time. There should be an integrating timer (with a maximum setting of 10 minutes) which terminates the exposure after a preset time after having given an audible warning for a few seconds. Switches should be spring-loaded ('dead-man') type, whether operated by hand or foot, i.e., the circuit-closing contact is maintained only by continuous pressure on the switch.

The fluoroscopist shall be protected by an apron or drape with a lead equivalent of not less than 0.5 mm. Wherever possible, members of the staff shall remain behind protective screens during fluoroscopy. If not, protective apron of lead equivalent of at least 0.25 mm shall be worn. Protective gloves with a lead equivalent of not less than 0.25 mm should be worn when any fluoroscopic examination may involve placing the hands in or near the useful beam.

It is preferable (though less convenient) to have the image intensifier above the X-ray tube (with undercouch tube) as it gives 2-3 times less potential exposure to workers than an overcouch tube and the image intensifier below; in the latter case the system should be operated by remote control. For lengthy procedures additional shielding may be required.

Dose reduction can be achieved by temporary removal of anti-scatter grid, use of pulsed systems, image storage, carbon fibre products, display of fluoroscopic time on the image monitor and automatic brightness control.

Fluoroscopy is finding increasing use in applications that involve long screening times (e.g. coronary angioplasty, coronary arteriography, pacemaker implant) with the potential for high patient doses. Such procedures should be carried out with dedicated equipment by qualified specialists.

39.4.16 Mobile Radiography

The head of the X-ray department should be responsible for safety of operation. Only trained persons should be allowed to operate, and access to the area should be restricted. The workers shall wear a protective apron, and wherever possible, should be at least 2 metres from the tube or the patient.

39.4.17 Mobile Fluoroscopy

A radiologist or radiographer should always be present. Radiography in conjunction with mobile fluoroscopy should be avoided (as it gives poor quality images). Persons within 2 metres of the X-ray tube or patient should wear a protective apron and dosimeter.

39.4.18 Photofluorography

Mass survey photofluorography utilizing a camera with miniature film to photograph the image produced on a fluorescent screen has, in the past, been widely used in chest surveys. The patient dose was many times higher than for full-size radiography. Where this system is still used, a wide aperture optical system, and correct film-screen combination should be achieved; it should be possible to get a satisfactory image with an entrance air kerma not exceeding 1 mGy. A combination with image intensification would be desirable. Mass survey photofluorographic equipment shall be so arranged that workers and waiting patients are protected without the necessity for protective clothing.

39.4.19 High Technology Equipment

Newer modalities like computed tomography and digital radiography provide improved image quality capabilities, provided that the modalities are used by experts. There is potential for increasingly higher doses with limited further improvement in image quality or diagnostic information.

39.5 SPECIFIC TYPES OF X-RAY EXAMINATIONS

39.5.1 Chest Examinations

Chest examinations form a large proportion of all radiological examinations and contribute a substantial fraction of population dose from diagnostic

radiology. In many cases such examinations are unjustified (e.g. young patients without respiratory or cardiac symptoms).

Often large field sizes (which include even adult gonads in the primary beam) are used unnecessarily. An adjustable light beam diaphragm is useful to ensure that a child is not irradiated with a beam size suitable for an adult. If a fixed aperture diaphragm is used, it is necessary to have one or two smaller apertures or a protective screen of adjustable height. High voltage air gap techniques are recommended in chest radiography.

Fluoroscopy should be done only with image intensification. Photofluoroscopy for mass examination of chest should be restricted.

39.5.2 X-ray Examinations during Pregnancy

The possibility of pregnancy is one of the factors to be considered in deciding to conduct an X-ray examination involving the lower abdomen in women of reproductive capacity. As already discussed earlier, the risk to the child who has been irradiated *in utero* during the first two weeks after conception is likely to be so small that there need be no special limitation on X-ray examinations required within that time period. Fetal irradiation from a diagnostic procedure during the time pregnancy was unrecognized very rarely justifies termination of pregnancy; after an estimate of the dose and associated risk by a qualified expert, the patient should be given balanced advice couched in careful terms so that she can take a decision about abortion.

39.5.3 X-ray Examination of Women of Reproductive Capacity

It is prudent to assume that any woman presenting herself for radiography at a time when a menstrual period has been overdue or missed could be pregnant. To minimize unintentional irradiation of the fetus, it is recommended that notices like the one given below be posted conspicuously in the department:

IF YOU THINK YOU MIGHT BE PREGNANT PLEASE NOTIFY THE PHYSICIAN OR RADIOGRAPHER BEFORE YOUR X-RAY EXAMINATION.

39.5.4 Obstetric Radiography

In many cases, especially for evaluation of fetal maturation and placental localization, ultrasonic methods are to be preferred. Radiographic

pelvimetry should not be done routinely. The superior-inferior projection for the pelvic outlet (brim view) should not be used in view of high fetal doses. Strict collimation, high speed films and screens, gridless techniques, and partial shielding of fetus on AP and lateral views are some of the procedures that help to reduce fetal dose.

39.5.5 Other Examinations during Pregnancy

Special care is to be taken to ascertain that the examination is indeed needed in types of examinations that may irradiate the fetus directly. Greater than usual care should be taken to minimize the irradiation time or number of radiographs. Radiography of areas remote from the fetus (e.g., chest, skull, extremities) can be done safely at any time during pregnancy under proper conditions.

39.5.6 Paediatric Radiology

Radiographers should be specially trained in paediatric methods. Mechanical means of immobilization are useful. Shortest exposure times should be used. Grids are not necessary for infants and doses can be reduced 3-4 fold. The areas of the body examined in infants are often smaller than the available film; collimation must be adjusted to the size of the area in question rather than to size of the film.

39.5.7 Mammography

The preferred techniques use either a molybdenum target and molybdenum filter with a rare earth intensifying screen and matching film, or a tungsten target and aluminium filter with xerographic plate.

Mammography should be carried out only with dedicated equipment and not with conventional equipment. Under no circumstance should the total permanent filtration be less than 0.03 mm Mo for screen-film mammography or 0.5 mm Al for xeromammography.

39.5.8 Dental Radiography

Dental radiology is carried out widely by non-radiologists. Most recommendations for general radiology apply here also. Dental radiographs should not be routine at every visit but be based on definite indications.

The room should be large enough with space for protective screen and a distance of 2 metres from the tube to the operator. Adequate shielding is to be provided to ensure safety of persons in adjacent rooms. The primary beam should not be directed towards a window or door. If there is a separate room for X-ray examination, it should be classified as a Controlled Area. In dental surgery the designation of a Controlled Area may not be helpful and extra shielding (beyond that provided by the building) should be considered for ensuring safety of persons in adjacent rooms.

The total permanent filtration for equipment shall be equivalent to not less than 1.5 mm Al for tube voltages not exceeding 70 kV.

Exposure of workers in dental radiology is very low if proper equipment is used. Personal dosimeters need not be worn (unless, for example, workload exceeds 150 intra-oral films per week). Protective clothing should be of at least 0.25 mm lead equivalence if a patient is to be held. A quality assurance programme should be followed.

Only dedicated equipment should be used. In particular, the use of high speed film and proper filtration will help to reduce skin dose. The beam should be directed away from the trunk and gonads. If a dental film cannot be kept in position, it should be held by the patient, and not by anyone else.

For intra-oral films, a field-defining spacer cone should be employed which provides a minimum FSD of 20 cm for equipment above 60 kV and 10 cm below 60 kV. Open-ended cylinders or divergent cones are preferable to pointed ones. The field diameter at the cone should not exceed 6 cm and shall not exceed 7.5 cm. Rectangular collimators are better than circular ones because they reduce the area exposed. The maximum range of the exposure switch should not exceed 5 seconds. The exposure should have a circuit-closing contact which can be maintained only by continuous pressure. It shall not be possible to make repeat exposures without release of the switch. The greatest concern is thyroid irradiation. Thyroid shielding is effective. Special equipment should be used for panoramic radiographs. Pantomography and cephalometry present special problems.

39.5.9 Examinations with Mobile Equipment in Wards and Operation Theatre

The principal difficulty is the uncertainty in the relative positions of the tube and the film. A light beam localizer (or some form of beam-pointing device) should be used. 'Hand fluoroscope' or 'head fluoroscope'

should never be used. Fluoroscopy with mobile equipment should not be carried out unless an image intensifier is employed. The tendency to shorten FSD and to remove filters in the case of low output mobile equipment should be avoided.

39.5.10 Low Yield Examinations

There are several examinations (called 'low yield') where reduction in frequency of the examinations may be warranted. Here a balance has to be made between the health costs of missing a diagnosis or making an incorrect diagnosis against the influence the diagnostic examination is going to have on the course of the treatment. Examples of low yield examinations are:

Excretory urography in children for evaluation of failure to thrive when there are no additional findings suggesting urinary tract abnormalities;

Fluoroscopy of the heart without special indications;

Fluoroscopy during reduction of uncomplicated fractures;

Radiography of the paranasal sinuses for evaluation of fever when there are no localizing synus symptoms;

Pre-operative chest radiography without special indications;

Chest radiography in pregnancy without special indications;

Pelvimetry in pregnancy without special indications;

Excretory urography in hypertension without special indications;

Barium enema in the absence of specific indications.

39.6 X-RAY EXAMINATIONS NOT DIRECTLY ASSOCIATED WITH ILLNESS

39.6.1 X-ray Examinations in Health Assessment

Chest radiography is often a part of an annual health examination and sometimes is part of the procedure for hospital admission. In many cases these may be unjustified. Dental radiography is widely carried out (often on children) by non-radiologists; they should not be routine but be based on definite indications.

39.6.2 X-ray Examinations in Screening for Specific Diseases

The justification should be based on a balance between the implied benefit to the individual and the population against the risk. The benefits will depend on the diagnostic yield of the screening procedures, the possibility

of treatment of diseases detected, and the advantage to the community from the control of the disease. The screening programmes should be evaluated frequently for their utility.

In countries where tuberculosis is a major health problem, sputum examination or tuberculin test should be the first screening procedure; it is also cost-effective compared to chest radiography of large groups. In the case of annual mammograms for early detection of breast cancer, it has been concluded that with the techniques now available, the number of early detection cases (in women above 50) that can be successfully treated is significantly higher than the likely number of radiation-induced breast cancers.

39.6.3 X-ray Examinations for Occupational, Medico-legal or Insurance Purposes

Such examinations may be for assessing fitness of an individual for work, medico-legal or insurance purposes (e.g., annual chest radiographs for teachers, food-handlers, hospital personnel; low-back radiographs for manual workers). Continuing evaluation is necessary whether the diagnostic yield of the routine examination is sufficient to justify the cost and health risk.

39.7 ABSORBED DOSES IN BODY TISSUES

The dose received from a given examination will vary widely throughout the body, the maximum being to the skin in the primary beam. Typical absorbed doses in tissues per examination range from a fraction of a mGy in some examinations to hundreds of mGy for specialized fluoroscopic examinations. Mean values of absorbed doses for common diagnostic X-ray examinations observed in recent surveys in UK are given in Table 39.2 (NRPB,1986,1991, as quoted in ICRP 62). An appendix in ICRP 34 gives more detailed information on the organ doses for various conditions of beam quality and field size for phantoms of adult, 5-y old, 1-y old and newborn.

The tissue doses are highly dependent on the technical factors employed in radiography and fluoroscopy, the characteristics of the equipment, the number of films taken, and the fluoroscopy time per examination. The doses for any particular type of examination vary between countries, between different institutions in one country, and even within the institution itself. It has been found that the dose may vary between hospitals by a factor of

2-10. The range for gonad exposures is as much as three orders of magnitude.

The incident skin dose gives an indication of the maximum dose received by any cell population of the body and ranges from less than 100 μGy for a large-film examination of the chest to 1 Gy for cardiac catheterization. Table 39.3 shows typical skin doses in the primary beam classified into 3 groups according to the magnitude of the dose. In fluoroscopy dose rates to the skin are around 2-6 R/min.

Many common examinations give gonad doses of less than 100 μGy. In examinations of the lower trunk in which the gonads are directly irradiated, the doses are 5-20 mGy. Large doses may also be given to the fetal gonads in abdominal examinations of pregnant women.

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Table 39.1: Approximate HVL in lead and concrete for broad beams of X-rays and gamma rays
(based on ICRP 33)

X-ray voltage/ radionuclide		HVL, cm	
		Lead	Concrete
X-rays			
	50 kV	0.005	0.4
	75 kV	0.015	1.0
	100 kV	0.025	1.6
	150 kV	0.029	2.2
	200 kV	0.042	2.6
	250 kV	0.086	2.8
	300 kV	0.17	3.0
	500 kV	0.31	3.6
	1 MV	0.76	4.6
	2 MV	1.15	6.1
	6 MV	1.54	10.2
	10 MV	1.69	11.7
Radionuclide			
	^{60}Co	1.2	6.1
	^{137}Cs	0.7	4.9
	^{182}Ta	1.2	-
	^{192}Ir	0.6	4.1
	^{198}Au	1.1	4.1
	^{226}Ra	1.3	7.0

HVL: half value layer thickness

Table 39.2: Mean values of absorbed doses and effective doses for common X-ray diagnostic examinations: UK survey data
(based on NRPB, 1986, 1991, quoted in ICRP 62)

X-ray examination	Absorbed dose (mGy)					Effective dose (mSv)
	Active bone marrow	Breasts	Uterus (embryo; fetus)	Thyroid	Gonads[a]	
Chest	0.04	0.09	*	0.02	*	0.04
CT chest	5.9	21	0.06	2.3	0.08	7.8
Skull	0.2	*	*	0.4	*	0.1
CT head	2.7	0.03	*	1.9	*	1.8
Abdomen	0.4	0.03	2.9	*	2.2,0.4	1.2
CT abdomen	5.6	0.7	8.0	0.05	8.0,0.7	7.6
Thoracic spine	0.7	1.3	*	1.5	*	1.0
Lumbar spine	1.4	0.07	3.5	*	4.3,0.06	2.1
Pelvis	0.2	*	1.7	*	1.2,4.6	1.1
CT pelvis	5.6	0.03	26	*	23,1.7	7.1
Intravenous urography	1.9	3.9	3.6	0.4	3.6,4.3	4.2
Barium enema (including fluoroscopy)	8.2	0.7	16	0.2	16,3.4	8.7
Mammography (screen-film)	*	2	*	*	*	0.1

* Less than 0.01 mGy; a: when two values are given for the gonads, the first value is for the ovaries, the second value is for the testes.

Table 39.3: Typical skin doses in primary beam in diagnostic radiology (median values in cGy per examination)
(based on UNSCEAR, 1977, quoted in ICRP 34)

A.	*High*
(a)	Fluoroscopy: Ba enema - 20; whole chest - 12; Ba swallow - 9; Ba meal-2.
(b)	Radiography: Cardiac catheterization - 47; mammography, pelvimetry, lumbosacral spine - 5 to 8.
B.	*Medium*
(a)	Pelvis: Angiography of abdomen; obstetric abdomen: descending urography, cystography; placentography; pelvis, dorsal spine; retrograde urography; dental; cholecystography - around 2-3.
(b)	Head; angiography of head; cervical spine, hip and upper femur; abdomen: salpingiography; tomography of chest; mass survey chest; thorax - around 1-2.
C.	*Low*
	Femur (lower two-thirds), leg and foot, arm and hand, chest - less than 0.4

CHAPTER 40

RADIATION PROTECTION IN RADIOTHERAPY

The material presented here is based on ICRP publication 33, 'Protection against ionizing radiation from external sources' (1978); ICRP publication 44, 'Protection of the patient in radiotherapy' (1985); and ICRP publication 57, 'Radiological protection of the worker in medicine and dentistry' (1989).

40.1 INTRODUCTION

Significant advances in cancer management have taken place in recent years, including improved diagnosis, improved supportive care and better understanding of radiobiological effects on cancer and normal tissues. The availability of a great variety of radiotherapy equipment, improved treatment planning, utilization of filters, protective blocks, rotational techniques, and multiple beams, have all made it possible to deliver a higher radiation dose to the target volume than previously achieved, while also reducing considerably the dose outside the target volume. These developments have been accompanied by an increase in survival rates for several types of cancer and a reduction in the incidence of complications.

With increasing dose, a progressively higher proportion of both tumour and normal cells die; the dose-response curve is steeply sigmoid in shape (Fig. 40.1). In radiotherapy the risk to the patient is twofold: failure to control the disease and risk to normal tissue from irradiation. The acceptable level of normal tissue damage will depend on the natural course of the disease if left untreated, and upon how well normal structures can be excluded from the target volume. (A greater degree of normal tissue damage is acceptable when the treatment aims at cure rather than palliation.)

For low LET radiations like X-rays and gamma rays, fractionation or protraction of exposures increases the total dose which must be given to produce a specified level of damage to either tumour or normal tissue,

because some radiation damage can be repaired. From clinical experience approximate empirical formulae of limited validity have been developed to predict cumulation of dose, fractionation and overall time that will produce comparable levels of acute radiation damage to specified tissues.

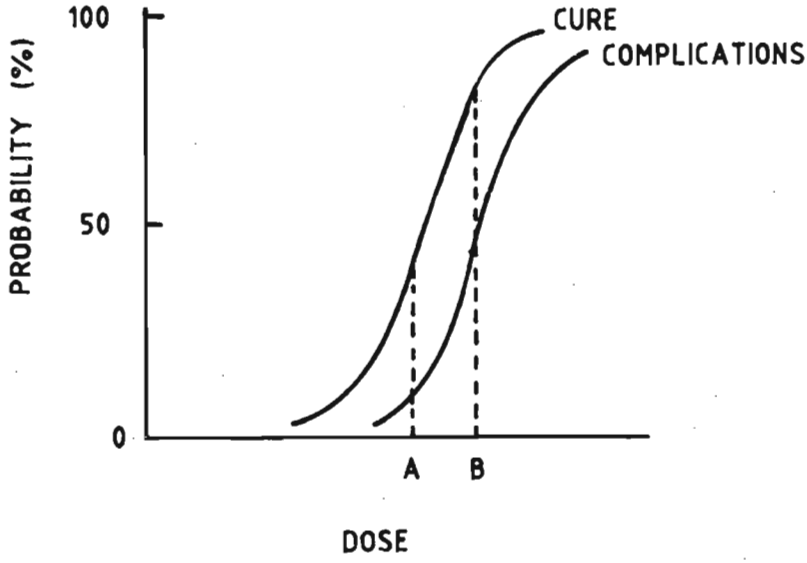

Figure 40.1. Hypothetical relationship between radiation dose and tumour cure/production of normal tissue damage (from ICRP44).

In the annual incidence of cancer, there is a tenfold variation range among different populations. In more industrialized countries the reported incidence is 200-400 new cases per 10^5 population per year, and about 50% of these receive radiotherapy at some time. One in four or five deaths in these countries results from malignant disease. In Bombay the reported incidence is 70 per 10^5 per year. Palliative treatment involves the use of lower doses than curative treatment and should be associated with minimal side effects.

The use of radiation therapy for benign diseases (e.g. ankylosing spondylitis, thyrotoxicosis, rheumatoid arthritis) differs widely between different countries, and is declining sharply in recent times; only 1-4% of radiation treatments are for non-malignant conditions. Benign tumours

are often well differentiated and respond poorly to radiation. However, radiation is used in conjunction with surgery in some cases (e.g. pituitary adenoma). Treatment of benign diseases by radiotherapy carries a small risk of carcinogenesis and hereditary detriment, which must be balanced against the potential value of the treatment.

40.2 JUSTIFICATION, OPTIMIZATION AND DOSE LIMITATION IN THE CONTEXT OF RADIOTHERAPY

In the context of application of the ICRP principles of justification, optimization and dose limitation to patient protection in radiotherapy, the professional judgement of the radiation oncologist that a proposed treatment by radiation will be of net benefit to the patient will normally constitute justification. Optimization acquires special meaning here. The complex interrelationship between ensuring adequate dose to the diseased tissues and reducing unwanted radiation dose to other tissues to as low as reasonably achievable means that the process of protecting the patient requires optimization of the treatment as a whole. Optimization of protection alone is not enough. For medical exposures dose limits do not apply.

Patient protection requires, uniquely, not the avoidance of exposure or even the avoidance of risk of severe damage to some tissues. Rather, once the choice is made that radiation is the appropriate treatment, it involves the optimal balance between the efficacy of irradiation in achieving sterilization of the malignant growth and the production of the minimum treatment-related complications. The latter involves making unwanted doses as low as reasonably achievable, as well as sparing critical normal tissues from receiving unacceptably high doses. The task of protection of the patient, in the broader sense, includes the need for proper medical training, sound clinical judgement, proper design and use of radiation-producing equipment and protective materials by suitably trained staff, and careful recording of the results of treatment so that future patients can be treated even better.

ICRP makes it clear that, where because of limitation of resources - both in material and personnel - the recommendations cannot yet be met, patients should not be denied necessary treatment.

40.3 CATEGORIES OF RADIOTHERAPY: BRACHYTHERAPY AND EXTERNAL BEAM THERAPY

Radiotherapy is broadly divided into two major categories, viz. brachytherapy and external beam therapy.

Radiation sources used for external beam therapy are usually classified according to the energy of the radiation as follows:

Superficial or contact therapy	10-50 keV max energy
Medium kilovoltage therapy	50-140 keV max energy
Orthovoltage therapy	100-400 keV max energy
Cesium-137 therapy	600 keV initial energy
Cobalt-60 therapy	1.2 MeV initial energy
High energy electron accelerators (for photons and electron beams)	4-50 MeV max energy

A well-equipped radiation therapy centre will have a range of radiation sources. In addition to radiation sources, many other items of equipment are necessary for the proper conduct of radiation therapy. (These are discussed later.)

External beam irradiation, by photons or high energy electrons, may be administered during an operative procedure when the area to be irradiated is exposed directly to the radiation beam in a single large dose. It is a highly specialized procedure.

Brachytherapy is divided into two sub-categories, viz., intracavitary and interstitial.

40.4 BRACHYTHERAPY

Radium sources, usually sealed within an inactive metal container or 'tube', have been used since the earliest days of radiotherapy for intracavitary insertion, particularly in the treatment of carcinoma of the cervix. Due to its great potential hazards, it has been strongly recommended that, as soon as practicable, radium sources be disposed of and replaced by less hazardous radionuclides such as ^{60}Co, ^{137}Cs or ^{192}Ir.

Previously it was common practice to position the applicator in the patient while it contained the radioactive source. This leads to high staff exposures. In many institutions this technique has been largely replaced

by 'afterloading', in which an empty container or guide is first introduced into the patient and its position then determined radiographically using dummy sources. Only when the position is satisfactory and the patient is in an isolated or shielded room is the actual radioactive source inserted. Afterloading may be either manual or mechanical. ICRP strongly recommends that, as soon as practicable without interruption of services, afterloading techniques be adopted.

In interstitial therapy, needles, seeds or wires containing radioactive sources are inserted directly into tumours in a geometrical arrangement designed to deliver a relatively uniform dose to the target volume or applied to the surface of tumours. These may be either removable or permanent. Removable implants are usually rigid needles containing ^{60}Co or ^{137}Cs inserted directly, or flexible wires containing ^{182}Ta or ^{192}Ir (either inserted into the tumour or passed through hollow plastic tubes previously inserted into the tumour). Permanent implants are usually seeds of short-lived nuclides of ^{198}Au or ^{125}I. For direct implantation of needles afterloading may not be possible; implantation of flexible or rigid guides into which the sources can be afterloaded is more satisfactory. Permanent implants should be done by experts since, once the sources have been implanted, errors of position can be corrected only with great difficulty.

In high dose-rate afterloading, high activity sources (which are mechanically moved from a safe container into flexible or rigid guides which have been placed in the patient) are used; treatment time is only minutes. The activities are likely to be 100 times higher than those used for conventional brachytherapy.

40.4.1 Occupational Exposures in Brachytherapy

It is worth noting that workers in brachytherapy departments get substantial doses. The exposure is relatively high among workers who prepare the sources for use. The degree of exposure of those who implant the sources into the patients depends on the manual skill of the operator, and, more importantly, on whether afterloading techniques are used. The exposure of nursing staff and staff in ward area is not reduced in manual after-loading but lessened in automatic afterloading. Doses to staff in operation theatre and the radiology department are reduced in manual afterloading. Table 40.1 gives the average staff doses (UK data) in a radiotherapy department.

40.4.2 Planning, Design and Operational Procedures in Brachytherapy

Attention has to be given to source preparation area, wards, treatment rooms, operation theatres, diagnostic X-ray facility for determining position of sources in patients, and safe transport of sources and patients.

40.4.2.1 Radioactive Source Laboratory

This is a Controlled Area with restricted access and provided with a monitoring device. Work benches should have impervious, cleanable surfaces. Benches and bench tops, handling area and room enclosure should have appropriate shielding (attenuation ~ 1000). There should be adequate ventilation and filtration of the exhaust air. A register shall be kept of all sources as well as their movements.

Direct viewing through a 5 cm lead glass in a well illuminated area and with means of viewing sources by magnification is preferable to use of mirrors. The provision of an ionization chamber for activity measurement is highly recommended.

There should be provision for storage of sources with partitioned built-in shielding and locks. Where radium is still in use, the storage facility should have proper ventilation to the building exterior to extract leaking radon (with an optional radiation detector alarm in the system). Remote handling devices should be available.

The laboratory should be close to the wards (and to the operation theatre). The storage safe should be close to the work benches. There shall be provision in the lab and/or the operation theatre for the sterilization of sources behind protective screens and for their temporary storage.

In the preparation and cutting of sources like indium wire, small radioactive fragments may be produced and handling instruments contaminated. Provision for safe collection of the fragments and monitoring of the area should be available.

The source containers must be clearly and permanently labelled so that the type and amount of radionuclide can be readily ascertained. Identification of needles and capsules of the same appearance but containing a different activity or nuclide should be facilitated, for example, by coloured beads or threads. Radium sources must be tested for integrity at least once a year. In interstitial therapy care must be taken to ensure that heat sterilization or mechanical handling does not damage the source. Con-

tamination checks on handling tools and transfer tubing of afterloading equipment should be carried out regularly. Shielded containers with a mechanical hoist to aid lifting shall be used during transport of sources.

40.4.2.2 Operation Theatre Design

Adequate shielding should be provided with attention to occupancy of adjacent rooms. The operation theatre should have shielded trolleys for source storage/dispensing and sterilizing facilities.

40.4.2.3 Dosimetry and Treatment Planning

The dose rate from individual applicators should be known correct to ± 5%. Correction for radioactive decay should be made at frequent intervals. Apart from the calibration certificate provided by the manufacturer, an independent measurement of the activity at the time of source receipt should be made. For multiple small sources the total activity of the batch should be known within ± 5% and the activity of the individual sources should not differ by more than 15%.

Treatment planning for brachytherapy should have the same degree of accuracy as external beam therapy (see Section 40.5). If pre-loaded applicators are used, the time required for dose calculation should be less than 5% of the treatment duration (to enable change in configuration if required). The use of a computer is of value here.

40.4.2.4 Insertion of Sources into the Patient

Appropriate handling tools and implant instruments which provide adequate distance and with shielding compatible with effective manipulation must be used. All operators must undergo training with dummy sources. Applicators should be appropriate both for the treatment plan and the anatomical characteristics of the area to be treated. A record of the number, type and position of sources in the patient and the date and time of the insertion should be kept.

The three-dimensional position of the sources must be determined immediately after implant. The dose rates to the target volumes and to critical normal tissues must be calculated as soon as possible. If placement is unsatisfactory, repositioning must be done in the operation theatre itself. The treatment duration should then be calculated. The position of

the sources should be checked during the course of the treatment. If the source fails to return to its safe position at the end of the treatment, this should be indicated by a visible or audible alarm.

In high dose rate brachytherapy, the timer (including a back-up timer) should be accurate within ± 0.5%. The source travel time from safe-to-exposed position should be determined, and allowed for in dose calculations if it is more than 2-3% of the treatment time. The equipment should be checked regularly. There should be means for indication of the source position.

An X-ray service facility for check of source position in patient may be provided in the diagnostic radiology department or by a simulator in the radiotherapy department.

40.4.2.5 Ward Design

Patients with sources should be kept in a separate specifically designated area (with barrier shielding in room enclosure, additional mobile shields, limitation of time and distance spent near the patient). In high dose rate applications a special shielded room is necessary in view of the high activities. A maze can be incorporated in the room design. Access to area outside windows should be restricted or mobile barriers used. Mobile bedside shields of 2.5 mm lead are useful.

CCTV surveillance of patients reduces worker exposure. An audible alarm should be provided at the room exit to check whether sources have been inadvertently removed or when the patient leaves the room. Separate toilets should be provided for the patients, care being taken that any source lost down the drain will be trapped and detected.

The dose rate at 1 m from the patient (typically 80-800 μSv/h) should be measured. A label giving details (activity administered, date and time, dose rate at 1 m, etc.) should be attached to the patient. Patients should be transported with minimum exposure to the workers. Crowded waiting areas and corridors should be avoided. The patient should never be left unattended during transport. Patients should be cared for by properly trained personnel.

Sources should be removed from the patient with appropriate tools. It should be ensured (by manual counting of the sources and by monitoring of the patient) that no source has been overlooked.

40.4.2.6 Operational Procedures

The source preparation area, operation theatre (when sources are present) and the brachytherapy wards are Controlled Areas which should have warning signs, radiation monitors and restricted access. Areas immediately surrounding a Controlled Area may need to be designated as Supervised Areas.

Records should be maintained of the location and movement of each source. Sources shall not be left unattended when in transport. All workers shall wear individual dosimeters. Finger dosimeters should be worn by workers who prepare sources. There should be an optimum balance between encumbering the operator with protective devices which provide shielding and the likely increase in exposure time.

Area monitoring around patients in nursing area, and monitoring of all material (e.g. linen, dressings) should be carried out. On completion of treatment, the patient, the operation theatre area, items handled by the patient, and nursing area should be monitored to ensure absence of any activity.

40.4.2.7 Emergency Procedures

In the event of an emergency the radiotherapist and the Radiation Safety Officer should be informed immediately. A storage container shall be available near an implant patient to permit source removal in an emergency. A list of emergency telephone numbers shall be readily available in the area. Written safety instructions on emergency procedures should be available in the area; these include action to be taken in the event of a source being lodged in the tubing during transit between the storage container and the patient.

A standard procedure should be followed whenever a source cannot be accounted for (e.g., monitoring of the patient and the area, source inventory check, checks for malfunctioning of monitor, hermetic sealing of suspected leaking source, monitoring of workers, etc.)

In the event of death of a patient, sources should be removed and returned for storage. If this is not possible, no person should work nearby without being informed of precautions to be taken. Prior to autopsy all removable implants should be removed and returned for storage. Permanent implants should be removed by surgical autopsy technique and transferred to a shielded container.

In the event of a fire, the protection of the patient takes precedence over the radiation protection of the worker. After evacuation, patients should be segregated and monitored, and stock taking of sources done. There shall be prior liaison with the local fire station for fire fighting arrangements. After the emergency is ended, access to the area should be controlled and the area decontaminated.

40.4.2.8 Quality Assurance

The following checks should be undertaken at regular intervals: Source inventory, leak tests for sources, functioning of radiation measuring and alarm instruments, and of automatic afterloading mechanisms. Timers controlling exposures from high dose rate afterloading equiment, including a back-up timer, should be accurate to $\pm$ 0.5%.

40.5 EXTERNAL BEAM THERAPY

40.5.1 Planning and Design

Safety is achieved by structural shielding, interlocks and radiation barriers. When a telecobalt or telecesium machine is in the OFF position radiation levels are low (around 10 μSv/h).

For planning purposes, dose rates outside treatment rooms should be time-averaged, so that this area (and the floors above and below) would be classified as neither a Controlled nor a Supervised Area. In intra-operative therapy the theatre should have similar shielding. Provision should be kept for shielding adequacy in case of future expansion (say, with a machine of a higher energy). It is cheaper to provide such shielding at the start itself rather than to modify the design later.

Entrance to the treatment room should be through a maze or shielded door, taking into account possible directions of primary beam, secondary and scattered radiation. A radiation warning sign at the entrance should be connected to the control console so that it is ON several seconds prior to switching on the machines. There should be a fail-safe interlock (e.g. photoelectric sensor) to prevent equipment being switched on when the door is open, or when someone enters the maze unintentionally; the equipment should automatically be switched off when the door is opened inadvertently. When structural shielding is inadequate in a particular direction, a beam interceptor (stopper) of adequate shielding can be introduced.

Inside the treatment room there shall be a visible/audible alarm to indicate that radiation is being or is about to be emitted. There shall be an emergency shut-off system inside the room when the beam is on. There should be monitors to give warning inside and outside the room if a source has failed to go back to the safe position. The machine shall be equipped with a locking device to prevent its unauthorized use. It shall be possible to open the treatment door from the inside.

The treatment room is a Controlled Area in which no one except the patient is present when the beam is on. Access to the treatment room should be controlled even when the machine is off. (For linear accelerators the hazard exists only when the machine is energized.)

For voltages below 140 kV, the worker can be present in the room taking the same precautions as in diagnostic radiology.

40.5.2 Emergency Procedures

An emergency plan should be permanently displayed both within the treatment room and at the control console. Workers should be familiar with measures to be taken in the event of the source failing to return automatically to its safe position. In such an emergency, the radiation field size should be reduced by closure of diaphragms, the patient removed from the room, and the source returned to the safe position manually with suitable tools. Workers should ensure that they do not move across the line of primary radiation when carrying out emergency procedures. If the source cannot be removed to the safe position quickly, the room should be evacuated immediately and expert assistance summoned.

40.5.3 Equipment Specification in External Beam Therapy

A device shall be provided which will automatically terminate the exposure after the preset time or exposure has elapsed. To ensure that the correct dose has been delivered, the constancy of the radiation output should be checked by a transmission or other device.

All beam therapy equipment shall be tested for performance and calibrated by an expert before it is first put into use and at regular intervals thereafter.

40.5.3.1 Superficial and Orthovoltage X-ray Therapy

The equipment shall be so designed as to prevent unintentional combination of tube voltage and filtration. Means shall be provided at the control panel to indicate tube voltage and current and for easy recognition of filtration being used. Whenever practicable, equipment which provides set combinations of tube voltage, current and filtration should be employed. The tube shall not be held by hand and it shall be fixed in correct position through mechanical devices.

Due to the low inherent filtration and short focus-window distance, the kerma rate close to the window of a low voltage tube for superficial therapy can be very high (around 10 Gy/s).

For X-ray therapy apparatus at voltages up to 500 kV, each tube shall be enclosed in a housing such that the air kerma rate measured at 1 m from the focus does not exceed 10 mGy/h, nor 300 mGy/h at any accessible position at a distance of 5 cm from the surface of the housing or its accessory equipment. (For 5-50 kV, the figure is 1 mGy/h at 5 cm.) Measurements can be averaged over 100 cm^2 at a source distance of 1 m or 10 cm^2 at 5 cm from the tube or source housing.

In addition to permanent diaphragms or cones, additional cones or adjustable diaphragms should be constructed so as to reduce the integral dose to the patient. They shall not transmit more than 2% of the useful beam. Where cones are not used, the diaphragm shall include a light beam localizer.

40.5.3.2 Sealed Source Beam Therapy (^{60}Co Units)

The beam mechanism shall return automatically to the OFF position at the end of the exposure or in the event of any breakdown of the force holding the beam control mechanism in the ON position. The OFF position shall be maintained until the mechanism is operated from the control panel. Additionally, in case of failure of the automatic return system, the exposure shall be capable of being interrupted by other means, e.g.manually.

An indicator shall be provided at the control panel (and, when practicable, also in the source housing) to show whether the source is in the ON or OFF position. Signals should be displayed at the entrance to the treatment room, when appropriate. The source shall be enclosed in a housing such that with the beam control in the OFF position leakage radiation

at 1 m from the source does not exceed 10 μGy/h. At any readily accessible position 5 cm from the housing surface, it shall not exceed 200 μGy/h.

In the ON position the leakage radiation at 1 m from the source shall not exceed 10 mGy/h or 0.1% of the useful beam air kerma rate at 1 m from the source, whichever is the greater.

Permanment diaphragms and cones shall afford the same degree of protection as the source housing. Adjustable beam limiting devices shall satisfy the requirements stipulated in the section on superficial and orthovoltage therapy.

Source housings should be fire resistant and shall be tested for leakage of radioactive material at least every year. (Free activity of more than 2 kBq is an indication of leakage and the equipment should be withdrawn from use till it is repaired and decontaminated.)

The adjustable beam-limiting device should be symmetrical about the beam axis within ± 0.5 mm. The central beam of the light beam localizer should coincide with the radiation beam axis. The field defined by the light beam should coincide with the borderlines of the radiation field defined at the position of the 50% isodose curve at a depth corresponding to the maximum dose, coinciding within 2 mm at a field size of 10 cm x 10 cm at the source-field distance normally used.

The mechanical and radiation beam isocentres should be contained within a maximum 2 mm diameter sphere. Homogeneity of the dose within the beam should be verified (especially for linear accelerators) for all orientations of the beam. The field size indicators should agree, within 2 mm, with the actual field size. Individual field-shaping blocks should have at least 5 HVL thickness.

40.5.3.3 High Energy Accelerators

The choice and arrangement of absorbers are important to minimize secondary radiation (which includes X-rays produced when electrons are absorbed). The adjustable beam-limiting device shall be so constructed that the leakage radiation imparts less energy to the patient than is imparted by a field of size 10 cm^2. This implies that for a field size of 10 cm x 10 cm the leakage radiation shall contribute less than 10% of energy imparted by the useful beam; for a 35 cm x 40 cm field the transmission figure is 0.8%. A similar recommendation applies to telecobalt units also.

The accelerator shall be provided with mutually non-interfering independent dose monitors, whose detectors shall be provided inside the radiation head. Each should be able to independently terminate irradiation. In case the 'master system' fails, the other system shall terminate the radiation after an additional dose of 0.4 Gy.

The system of interlocks should prevent mistakes in selection of type and energy of radiation, wedge field, scattering foils, etc.

For megavoltage and electron beam therapy, the kerma rate due to leakage radiation at any point outside the useful beam but inside a plane circular area of radius 2 m centred around and perpendicular to the central beam axis at the normal distance of treatment shall not exceed 0.2% of the air kerma rate on the axis at the same distance. Beyond this area the kerma rate of leakage radiation (excluding neutrons) at 1 m from the path of the elctrons between their origin and the target or the electron window shall not exceed 0.5% of the air kerma rate on the central axis at the normal distance.

The contribution of neutrons to the dose inside and outside the area should be kept as low as practicable (< 1% of the X-ray kerma inside the field).

40.5.3.4 Neutron Therapy

Working procedures in neutron therapy are similar to those in beam therapy. The equivalent dose rate from neutron radiation should be less than 1 μSv/h outside the treatment room and less than 2.5 μSv/h at the entrance to the labyrinth (Bonnett, 1983).

Induced activity may be present in the neutron generator equipment as well as in other locations. Dose rates may be around 370 μSv/h around the unit at 5 min after switching off and 5-33 μSv/h after a week-end break. Both individual and environmental monitoring for neutrons are required in addition to beta/gamma monitoring. The highest doses are likely to be received by maintenance/servicing staff.

40.5.3.5 Quality Assurance Programme in External Beam Therapy

A quality assurance programme shall be carried out involving acceptance tests for new equipment and periodic performance tests which include main equipment, simulator, accessories, recording and processing systems.

The frequency of the various check schedules include daily check on interlock system; daily check on emergency system; three-monthly dosimetric checks for telecobalt units in the first year after a new source has been installed to detect possible radioactive impurities in the source (using a dosimeter with a reproducibility of ± 0.5%); periodic check on warning systems and timers; periodic checks on contamination on external surfaces of sealed equipment and of leakage radiation levels; periodic checks on beam output, beam energy, uniformity, accuracy of beam direction, dose monitors, field-determining devices. (Monitors may be affected by RF interference from nearby high energy electric generators or due to pulsed nature of radiation from accelerators.)

40.6 OPERATIONAL PROCEDURES

40.6.1 Patient Care

After clinical diagnosis, the optimum form of therapy should be determined by the specialists. If the chosen treatment includes radiotherapy, the radiation oncologist, in conjunction with his staff, must prepare the optimum treatment plan. The radiation oncologist should be present in the radiotherapy department whenever treatments are being given and must personally supervise the initial setup for treatment and any subsequent alterations. The patient should be examined at least once a week by the oncologist during treatment and subsequently followed up at intervals. Detailed records should be kept and reviewed periodically to assess the results of the treatment for the benefit of future patients.

40.6.2 Workers

Workers should wear personal dosimeters and their doses recorded. (In modern external radiotherapy exposure of workers is generally very low.) The last worker to leave the room prior to start of treatment should visually check the area that no one except the patient is inside the room. Similarly, before entering the room he should confirm that the source is within the housing.

40.7 ANCILLARY EQUIPMENT FOR RADIOTHERAPY

Equipment for radiography and special imaging techniques (like ultrasound, CT) must be available. It is now possible to input CT data directly into treatment planning computers. Simulators allow the produc-

tion of radiographs for the precise localization of the radiation field. It is useful if the simulator can also do fluoroscopy with image intensification. Simulators provide better images than high energy beams, and do not also involve setting up of the patient on the busy therapy machine. For treatment planning involving complex calculations, the use of a computer is suggested.

To protect normal tissue, blocks individually designed for each patient provide the best protection. The slope of the edges of the beam-shaping blocks should coincide with the divergence angle of the beam. Wedge filters are used to shape the isodose curves to the desired treatment volume and to correct for oblique incidence or sloping body surfaces. Compensating filters, individually designed, are used to offset the effect of tissue inhomogeneities and irregular surfaces. A suitable range of wedge and compensating filters should be available.

Attachments for the wedge filters, compensating filters, beam-shaping blocks, etc. must be securely mounted so that the planned dose distribution is not altered accidentally by change of position and that no device can accidentally fall on the patient.

Various types of dosimeters should be available. There must be means for producing directly a radiograph (treatment port film) to verify the position of the beam. Patient positioning devices (like 3 or 4 light sources or laser beams, 2 horizontal, and 1 or 2 vertical, which intersect at the isocentre) must be available. When only one beam is used, a simple mechanical front pointer may suffice. There must be means for observing the patient continuously during treatment (lead glass window or CCTV). Where necessary, patients may have to be immobilized suitably.

40.8 ACCURACY OF DOSE DELIVERY

There is greater need for precise dose delivery in cases of high dose curative treatment which approaches normal tissue tolerance than for palliative treatment. Greater accuracy is required for high dose treatment of regions adjacent to critical normal tissues.

Deviations of 5% or more can be expected in the cumulative dose for a complete course in 3-4% patients due to random and systematic errors even in the best of institutions (ICRU Report 24, 1976). There are also uncertainties associated with tumour localization, dose distribution problems due to surface heterogeneity, movement of organs during treatment, etc. Mistakes due to human error or unforeseen equipment malfunction-

ing can never be completely avoided; their occurrence can only be minimized by staff training and a good QA programme.

40.8.1 Dosimetric Considerations

For external beam therapy doses should be capable of being determined with an accuracy of ± 3%. Measurement of depth doses, wedge and tray factors should be performed with a precision of 0.5-2%. Dosimeters should be calibrated at regular intervals against a secondary standard dosimeter which itself should have been calibrated against a recognized standard. The IAEA/WHO Network of Secondary Standard Dosimetry Laboratories and the IAEA/WHO TLD Service for Intercomparison of Dosimetry in Radiotherapy offer calibration facilities.

40.8.2 Treatment Planning and Optimization

The dose to the target volume should be within ± 5% of the prescribed dose. When treatment planning is made manually care should be taken to place the isodose charts in the correct position. If a computer is used, the programme should be verified initially by phantom measurements. In addition a QA programme should be developed for periodic checks of computer programmes. Isodose charts supplied by the manufacturers should be confirmed independently.

With CT, tumour volumes can be localized accurately. Anatomical parameters for the individual patient for treatment planning should be determined with the patient in the same position as that during treatment. If significant changes in body contours may occur during treatment they should be remeasured and the treatment plan corrected.

40.8.3 Performance of Treatment

Before starting treatment all computations should be checked independently by a second individual (including maximum and minimum dose to the target tissue, and dose to vital structures). Cumulated doses should be recorded at each treatment and checked independently at regular intervals. The pre-set time or monitor values should be calculated and independently checked.

It may be advisable to make dose measurements on the patient during the first treatment to compare actual doses with those indicated by the treatment plan. Verification radiographs should be obtained at the onset

and periodically during treatment. For external beam therapy the patient should be positioned accurately and observed constantly during treatment.

40.9 ABSORBED DOSES INSIDE AND OUTSIDE THE USEFUL BEAM

In general, radiation therapy makes use of individual treatment plans to deliver the desired dose to the target volume while the dose to tissues outside is kept as low as possible. The tissues outside the target volume may be separated into those within the useful beam, where the dose is high, and those outside where it is r ' 'ively low. [The useful beam is usually defined as being outlined by t ı J% isodose curve at the depth of the maximum value of the absorbed dose at the isocentre, if one exists (ICRU Report 24, 1976)].

40.9.1 Doses to Tissues Within the Useful Beam

For external beam therapy, fixed beams (single or multiple) alone, partial or full rotational beams are used. Single beams are used for treatment of tumours near the surface. For deeper tumours an acceptable dose distribution can only be achieved by using multiple or moving beams; treatment plans are obtained by superposing isodose charts or by computer. Patient contours, position of tumour and critical organs are determined by measurements on the patient and from radiographs, ultrasonic or CT scan. Multiple beam therapy requires more time for treatment planning, simulation, setup and treatment, but improves dose distribution. Moving beam therapy may involve rotation (from small angles to 360 degrees) around equipment isocentre or translation of beam with respect to the patient; these produce smoother contours than multiple fixed beams.

Figures 40.2, 40.3 and 40.4 (taken from ICRP 44) illustrate some isodose distributions.

40.9.2 Doses to Tissues Outside the Direct Beam

This consists of leakage radiation (which penetrates the shielding of the therapy unit), and scattered radiation which may be internal (from within the patient) or external (from beam-limiting devices, filters, couch,

etc.). Internal scatter can be measured or calculated by Monte Carlo methods for a phantom. ICRP 44 gives tables and figures of the internal scatter contribution for various cases; it may generally vary from a fraction of 1% to less than 10%. The higher the photon energy, the smaller this contribution. Both leakage and scatter components should be reduced. Permissible leakage levels have already been discussed.

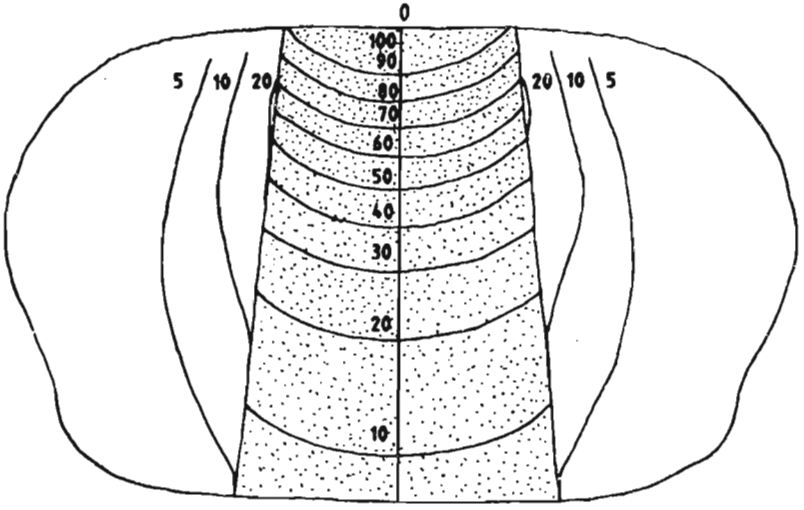

Figure 40.2. Isodose distributions for ^{60}Co single beam (from ICRP 44).

40.10 RADIATION RESPONSE

ICRP 44 gives a brief description of the effects of dose fractionation. With low LET radiations repair of sub-lethal damage occurs largely within 3-4 hours. In fractionated exposure the net damage is determined by the radiation-induced cell killing offset by cell proliferation that occurs during the overall treatment. The commonest treatment modality in external beam therapy is to give one fraction of 2 Gy to the target volume per day, 4-5 days a week over a total time of 4-6 weeks. The 'therapeutic ratio', i.e. the ratio of local tumour control rate to normal tissue complication rate, should be optimized.

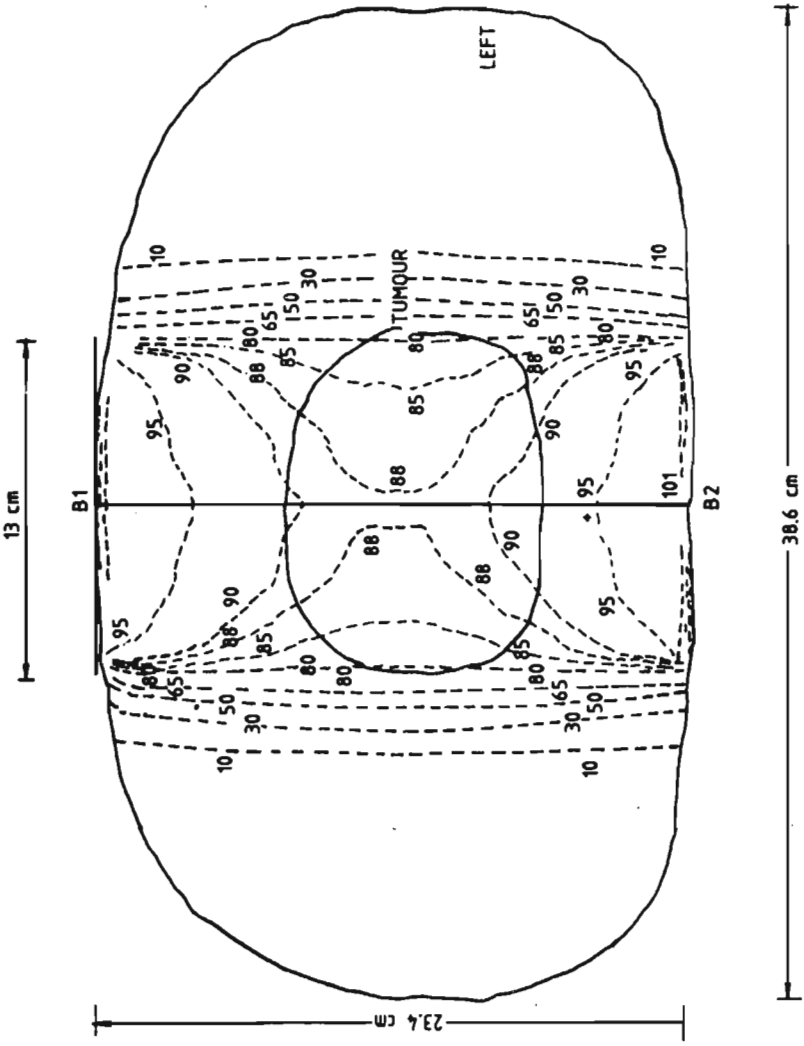

Figure 40.3. Isodose distributions for ^{60}Co coaxial opposed beams including contours of an assumed target volume (from ICRP 44).

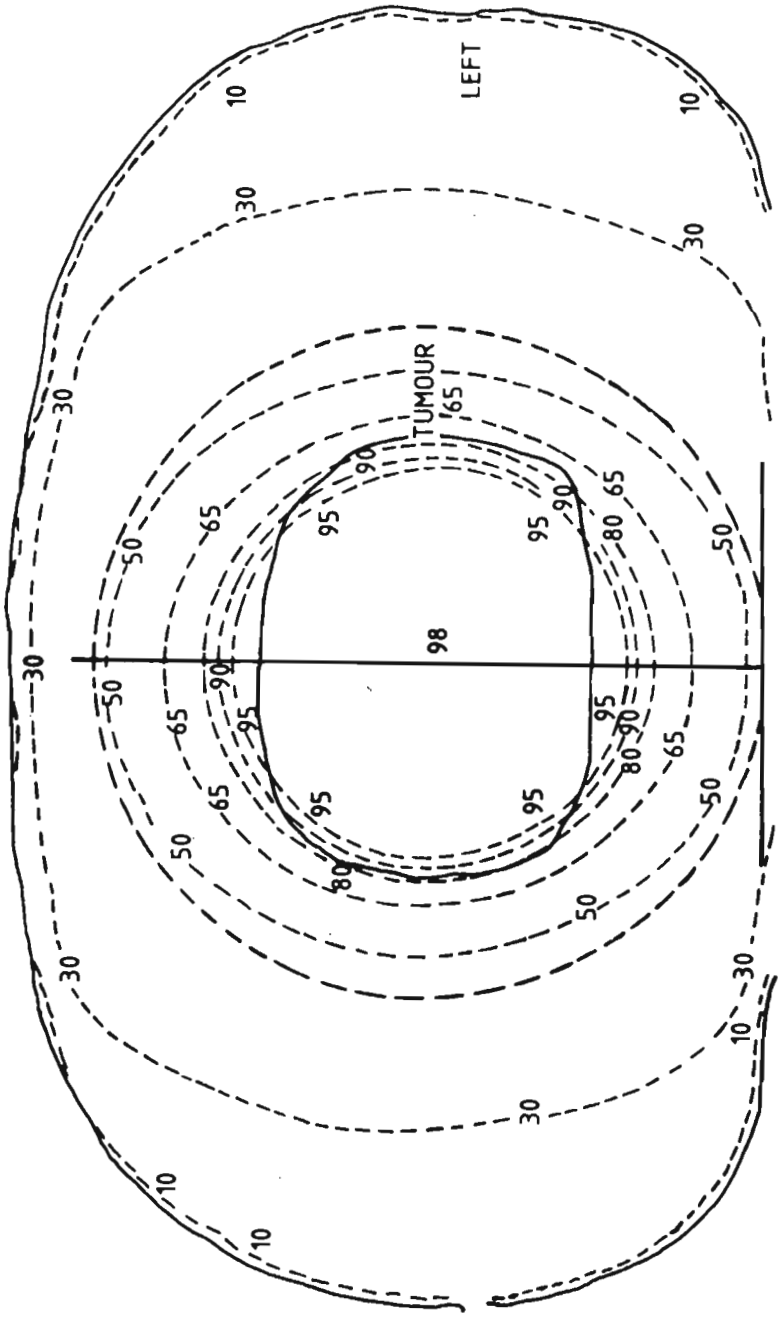

Figure 40.4. Isodose distributions for 8 MV photons from linear accelerator using 360° moving beam isocentric technique including contours of an assumed target volume (from ICRP 44).

Special techniques include:

(a) Irradiation while the patient breathes pure oxygen under hyperbaric conditions. Radiation sensitivity of formerly hypoxic cells in tumours is increased with little effect on normal tissues;

(b) Use of electron-affinic hypoxic cell sensitizers like misonidazole;

(c) Use of radioprotectors like WR-2721 which raise by about 30% the dose necessary to produce a given type of damage; and

(d) Hyperthermia (with raising temperatures in the tumour area to above 42 °C) in conjunction with radiation.

40.11 RISKS TO SPECIFIC ORGANS AND TISSUES FROM RADIOTHERAPY

ICRP 44 summarizes the expected risks (mainly deterministic) to specific normal organs and tissues during radiotherapy and indicates the threshold doses for these effects under the fractionated irradiation regimes used in radiotherapy.

Early, generally transient, reactions may occur during or within a few weeks following completion of radiotherapy. Later manifestations of injury include lasting cosmetic and/or functional deficit. Irradiation in childhood may interfere with subsequent growth and development, e.g., radiation-induced hypoplasia of breast, cartilage, bone, muscle, etc. For a summary of deterministic injury to various organs and tissues at various doses (including thresholds) for acute exposures as well as for conventional fractionated exposure regimes generally used in radiotherapy, reference may be made to Chapter 10, Section 1 and Chapter 11, Section 4.

40.12 ORGANIZATION AND PLANNING OF RADIATION ONCOLOGY SERVICES

Since cancer is treated by a variety of modalities including radiotherapy, the approach should be multidisciplinary. While radiation oncology should be an independent clinical speciality, it should be closely associated with a comprehensive facility (which includes medical oncology, surgery, pathology and medical imaging). The services should be centralized in comparatively few centres to make it economically feasible to provide variety of equipemnt and staff. The workload will depend on the epidemiological pattern of cancer incidence in the area.

The suggested goals for staffing and equipment (which are unlikely to be achieved in the foreseeable future even in industrialized countries) are:

Radiation oncologist, medical physicist, dosimetrist, and nurse - one each per 200-400 new patients treated per year;

Radiographer - one or two each per equipment including simulator; one or more for brachytherapy;

Megavoltage or telecobalt machine - four per million of population; and

Simulator - at least one per department.

Recommendations (which again represent goals as of to-day) are given in ICRP 44 for the qualifications, training and duties of the radiation oncologist, medical physicist and radiographer.

40.13 MEDICAL RESEARCH INVOLVING RADIOTHERAPY

It should conform to the principles of the Declaration of Helsinki of the World Medical Association and related international guidelines.

REFERENCES

Bonett, D.E. (1983). Fast neutron therapy at Edinburgh: staff protection. *Brit. J. Radiol.*, **56**, 665-72.

ICRU Report No. 24 (1976). *Determination of absorbed doses in a patient irradiated by beams of X or gamma rays in radiotherapy procedures*. International Commission on Radiation Units and Measurements, Bethesda, Marlyand.

Table 40.1: Average annual doses in a radiotherapy department before and after replacement of radium by automatic after-loading cesium sources (UK data, 1985)

(from ICRP 57)

Category of staff	Average annual dose, mSv	
	Before	After
Radiotherapists	1.30	1.30
Radiographers	1.00	0.80
Mould room technicians	2.20	0.60
Physics staff	0.50	1.20
Radium custodians	4.00	1.00
Nursing staff	4.00	0.90

APPENDIX 40.1

PROTECTION OF THE WORKER IN BALNEOTHERAPY

In many countries radon and its daughters in water and air are used for the treatment of non-malignant conditions. These practices should be justified as per ICRP guidelines. Staff exposure is mainly due to radon from deep well water and is exclusively from inhalation. Attendants may get exposures from around 10% to 100% of the occupational dose limit (4.8 WLM per year). Service engineers may get 2-4 times the dose limit. There is no justification for exposure above the prescribed dose limits. In certain countries radon daughters are required to be filtered out, which will reduce doses to both patients and bath attendants. Individual dosimetry should be introduced in appropriate cases.

CHAPTER 41

RADIATION PROTECTION IN NUCLEAR MEDICINE

The material presented in this chapter is based on ICRP publication 52, 'Protection of the patient in nuclear medicine' (1987), ICRP publication 53, 'Radiation dose to patients from radiopharmaceuticals' (1987), ICRP publication 57, 'Radiological protection of the worker in medicine and dentistry' (1989), and an addendum to ICRP 53 given in ICRP publication 62, 'Radiological protection in biomedical research' (1992).

41.1 INTRODUCTION

In nuclear medicine unsealed radioactive isotopes are used for diagnosis, therapy and clinical research. The major part of the discipline, diagnosis, involves *in vivo* and *in vitro* applications. No radioactivity is administered to the patient in *in vitro* applications. Diagnostic nuclear medicine employs, as a rule, radionuclides with short half-lives, the most common beings compounds of technetium (^{99m}Tc) which is a pure gamma emitter with a half-life of 6 hours. This allows one to use activities of up to 1000 MBq which give only moderate doses to the patient.

The annual frequency of diagnostic nuclear medicine procedures in developed countries is 10-40 per 1000 of the population while in developing countries it is 0.2-2 per 1000, as per the 1982 Report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR, 1982).

41.2 GUIDELINES TO GOOD CLINICAL PRACTICE

The professional judgement of the nuclear medicine physician and of the referring physician that a proposed use of radiation will be of net benefit to the patient will normally constitute justification of the patient's exposure. Methods of determining the value of specific nuclear medicine procedures are improving, and techniques are becoming available for determining efficacy and improving decision making (e.g., decision making matrices, receiver-operator characteristics, and information theory). An

analysis should be made of the possibilities of alternatives to *in vivo* nuclear medicine procedures (such as *in vitro* procedures, ultrasound, thermography, etc.). In nuclear medicine, optimization means that the patient dose (which is related to the administered activity) should not exceed that required to provide the necessary medical information. There is an optimum value for the activity below which much diagnostic information is lost and above which additional information acquired is marginal. Quality control of radiopharmaceuticals and equipment as well as techniques for improving the signal-to-noise ratio (e.g., by electronic means) will help in optimizing performance.

The referring physician should have some familiarity with basic aspects of nuclear medicine and its capabilities to enable him to decide about the worthwhileness of a nuclear medicine test. When referring to the nuclear medicine physician he should describe the patient's condition and indicate the medical objectives. The nuclear medicine physician will then decide as to whether an *in vitro* or *in vivo* procedure would be useful, and, if so, what type of procedure. He has the ultimate responsibility for the control of all aspects of nuclear medicine examinations, including the radiation protection of the patient. The medical physicist is responsible for the physical and technical aspects of dosimetry, protection, quality control, data handling, advice on choice of equipment, laboratory planning, as well as teaching. Qualified technologists should assist the physician in carrying out the tests. The expert advice of the Radiation Safety Officer should be available to all the staff of the department. It will be useful to have a Radiation Safety Committee to provide advice on protection to the patient, workers, and the public, and on maintenance of appropriate records, to take care of regulatory requirements, and prepare written policy instructions.

41.3 GENERAL PRINCIPLES OF PLANNING AND DESIGN

Radiation protection in nuclear medicine involves control of both external exposure and contamination. As the radiopharmaceuticals are used mainly as non-volatile solutions or colloids (except in the case of certain radioiodination procedures in the radiopharmacy), the hazard of internal contamination, if good practices are followed, is minimal. Contamination control is achieved by defence in depth - proper laboratory design, correct operating procedures, clean working conditions, and containment of radioactive materials.

41.3.1 Radiotoxicity Classification

The degree of safety to be incorporated in the planning of facilities will depend on a combination of activity to be handled, nature of radionuclide, and type of operation. The concept of *weighted activity* is useful here.

Radionuclides are classified into three broad categories in decreasing order of radiotoxicity, with corresponding weights of 100, 1 and 0.01.

Category A: Examples are ^{125}I, ^{131}I.
Category B: Examples are positron emitters, ^{51}Cr, ^{67}Ga, ^{99m}Tc, ^{111}In, ^{113m}In, ^{210}Tl.
Category C: Examples are ^{3}H, ^{81m}Kr, ^{127}Xe, ^{133}Xe.

Types of operation are similarly given weights:

Storage:	0.01
Scintigraphy, patient waiting area, diagnostic bed area waste handling:	0.1
Local dispensing, radionuclide administration, simple radiopharmaceutical preparations, therapy patient bed area:	1.0
Complex radiopharmaceutical preparations:	10

The final figure obtained by multiplying the above two is the weighted activity, which determines the category of hazard:

Hazard category	*Weighted activity*
Low	< 50 MBq
Medium	50-50,000 MBq
High	> 50,000 MBq

41.4 BASIC PRINCIPLES OF DESIGN

41.4.1 Requirements and General Principles of Design

The following are the requirements and principles of design:

Proper environmental characteristics (temperature, humidity, clean air, stabilized electric power supplies) for reliable operation of instruments;
Adequate space in radionuclide handling area;
Bright and shadow-free work areas;
Materials resistant to chemical corrosion, heat and fire;

Non-absorbable, easily decontaminable floors, walls, ceilings and work surfaces;

Hand-washing facilities, preferably operable without use of hands;

Shower for emergency use;

Sinks with taps that can be operated without use of hands;

Drains connected directly to main sewer (with accessible traps for periodic monitoring in medium/high hazard labs);

Fume hood operating under negative pressure for complex radiopharmaceutical preparations;

Fully exhausted vertical laminar flow system under positive pressure with discharge outside the building if sterile procedures are required (Additional charcoal filter to remove iodine desirable);

Monitoring facilities; and

Segretation of areas from the point of view of external radiation levels and potential contamination;

Activities should progressively increase in consonance with work pattern:

Clean area - waiting area - *in vitro* counting - *in vivo* counting and imaging - radiopharmaceutical preparation - waste storage - therapy ward.

41.4.2 Technical Details of Design

The following sections give some more detailed notes on specific aspects of operation and design.

41.4.2.1 Design of Medium and High Hazard Radionuclide Laboratories

Classification as 'controlled area';

Control of entry; minimum stay time;

Adequate (~ 10 HVL) shielding for benches, bench tops, handling areas, room enclosures;

Direct viewing through brightly illuminated lead glass (and with magnification facility); this is preferable to use of mirror during isotope handling;

Use of remote handling devices for manipulation of high activity gamma sources;

Shielded and partitioned storage containers; and

Provision of device for measuring source strength.

41.4.2.2 Radiopharmacy

The greatest amount and variety of radionuclides are handled in this area. The basic requirements include: shielded storage space; ventilated

laminar flow facilities for maintaining aseptic conditions; and fume hood.

41.4.2.3 Radioactive Waste Storage

Easy accessibility for short/long-term storage;
Good ventilation and plumbing; and
Restricted entry.

41.4.2.4 Toilets

Separate toilets for workers and patients.

41.4.2.5 Classification of Areas and Monitoring Procedures

In view of possible contamination hazards, it is useful to demarcate, in addition to 'controlled' and 'supervised' areas, a 'controlled contamination area'; contamination control is also important to minimize interference with radionuclide measuring and imaging devices;

Area monitors for external monitoring of controlled areas;

Wipe tests to check contamination levels in areas of high external background;

Routine air monitoring not normally necessary;

Checking of skin contamination (averaged over an area of 100 cm^2) and prompt decontamination where required (see later for methods of decontamination); and

Facilities for internal monitoring (whole/partial body counting, urine analysis) for workers handling large amounts of volatile materials.

41.4.2.6 Storage and Movement of Radioactive Materials

Prompt recording of entry/dispensing/exit of sources;
Periodic inspection and stock-taking; and
Shielded, contamination-free transport containers with radiation sign.

41.4.2.7 Operational Procedures

Controlled entry;

No eating/drinking/smoking/application of cosmetics in 'controlled' and 'supervised' areas;

Containment of contamination through manipulations in trays lined with absorbent materials and disposable absorbent coverings on bench tops;

Contamination checks on outside of isotope containers;
No wounds or breaks in skin of workers;
Use of impervious gloves, lab coats, aprons;
Container for used gloves; facilities for glove decontamination;
Proper area for radiopharmaceutical administration; use of shielded syringes; control of spills during injection;
Washing and monitoring of hands; decontamination where necessary;
Ready availability of personal protective devices in controlled/supervised areas (gloves, overshoes, protective gowns);
Monitoring of items leaving controlled area;
Routine periodic monitoring for contamination in accessible areas;
Control of surface contamination levels to below those given in Table 41.1;
Record maintenance; and
Periodic inspection by the Radiation Safety Officer.

41.4.2.8 Preparation and Quality Control of Radio-pharmaceuticals

Quality control and quality assurance ensure that the radiopharmaceuticals are effective, toxicologically safe, contain known ingredients of known quality in known proportions and predetermined purity.

Quality control specifications for a radiopharmaceutical include radionuclide and radiochemical purity, specific activity, chemical purity, sterility, apyrogenicity, stability and toxicity. Radionuclide impurities increase patient dose and also degrade image quality; some impurities may change with time. Basic checks to be invariably made include: package documentation, surface contamination, leakage of activity, and expiry date.

Radiopharmaceuticals can be divided into the following four general classes having different requirements for quality control (QC):

(a) Ready-to-use radiopharmaceuticals supplied as such;

(b) Radionuclides derived from generators (e.g., ^{99}Mo-^{99m}Tc) and kits which are delivered to the department by the manufacturers. The final product for administration requires further preparation by the user;

(c) Facility-produced radiopharmaceuticals (e.g., cyclotron produced radionuclides, laboratory produced pharmaceuticals such as colloids);

(d) Autologous radiopharmaceuticals produced by labelling of substances from the patient for readministration (e.g., ^{51}Cr-labelled RBC).

The quality control system appropriate to each class of radiopharmaceuticals is discussed in some detail in ICRP 52. Particularly for generator-produced pharmaceuticals, breakthrough of parent during elution of generator, and for kit prepared compounds, rapid check of radiochemical purity by chromatography are recommended.

An essential part of a QC programme is to maintain appropriate records. These should include protocols for radiopharmaceutical preparation and testing. Drug defects and adverse reactions should be systematically recorded and reported.

41.4.2.9 Iodination of Radiopharmaceuticals

Operation in fume hoods;
Periodic thyroid monitoring of workers; and
Special attention to female workers (iodine crosses the placental barrier to reach fetal thyroid).

41.4.2.10 Decontamination of Workers

Monitor body surfaces and clothing to identify area of contamination; cover wounds, if any, with adhesive water-proof covering;
Wash contaminated area with mild soap and water using a soft nail brush; pay special attention to cleaning under finger nails;
First remove contamination from nose and mouth;
Start from periphery of contaminated area and work gently towards centre;
If mild soap and water fail, use weak detergent solution;
Shower if necessary;
For persistent contamination apply potassium permanganate solution (but not near eyes or in hair) for a few minutes, wash off and dry; then wash pigmented area with 10% sodium metabisulphite solution to remove coloration;
Irrigate wounds immediately with sterile water or saline; encourage free bleeding for about a minute;
Perform bioassay for accurate estimation of intake; and
Train workers in methods of mouth and eye decontamination.

41.4.2.11 Handling of Radioactive Waste

Remove wastes from working areas without delay;
Use leak-proof strong containers for solid wastes;

Seal and label bags/solid waste containers before transfer; and

Dispose off liquid wastes down special sinks in controlled and supervised areas.

41.4.2.12 Emergency Procedures

Radiation incidents are unplanned events during which higher than normal potential/actual exposures are likely. An accident is one where doses can exceed recommended dose limits. They may arise due to improper handling, mislaying of sources, incorrect administration, fire or explosion. Safeguarding of life is the most immediate concern; next is contamination control. Such events should be recognized and acted upon quickly. Emergency plans should be prepared in advance, delineating lines of responsibility. Training (with periodic rehearsals) in emergency management is necessary. Expert radiation protection and medical advice should be available readily.

Each room should have instructions on emergency management including persons to be contacted. Emergency equipment (including warning signs, first aid kit, decontamination kit, tools and portable monitoring equipment) should be readily available.

41.4.2.13 Fire or Explosion

The first concern is safety of patients and staff; radiation hazards would be relatively small. Where high levels of activity are handled, training of staff in fire fighting and prior liaison with local fire station are necessary.

41.4.2.14 Quality Control for Equipment

The first is acceptance testing which will form the baseline for assessment of future performance also. There should be additional periodic testing and preventive maintenance. A data-recording log book is essential. Appropriate calibration instruments and radiation sources should be available. The major quality control tests (as discussed in ICRP 52) are summarized below:

Radiation sources: Gamma radiation sources of appropriate energy, activity and shape, calibrated against a national or other standard should be available. Unsealed sources in solution are used in phantoms and flat field sources for quality control of equipment.

Dose calibrator (activity meter): Tests include background; shield leakage; linearity; accuracy and precision for various radionuclides and geometries (e.g., vials, syringes).

Well counter: Performance of pulse height analyzer, scaler/ratemeter; energy response; energy resolution; sensitivity; precision; linearity.

Probe: In addition to tests for well counter performance, tests on output devices, collimators; matching of performance of individual probes in a multiprobe system.

Photographic process: Daily checks on settings of formatter and video hard copy of gamma camera; daily film processing checks by densitometric measurements on films exposed to a sensitometer.

Rectilinear scanner: Additional checks include those on mechanical parameters like scanning speed, line spacing, display functions (background subtraction, contrast enhancement, antiscalloping, colour and photorecording); collimator characteristics.

Gamma camera: Calibration of analyzer energy peak and window controls; energy and spatial resolutions; field uniformity; sensitivity; linearity; shield leakage; intrinsic and extrinsic performance (with and without collimator); routine inspection of collimators, mechanical and electric subsystems.

Single photon emission computed tomography (SPECT): Checks here are more important than for planar gamma camera. Important parameters are camera uniformity and alignment of geometric axis of rotation with the electronic axis of the camera.

Positron emission tomography (PET): The high energy (0.51 MeV) of annihilation gamma rays will necessitate more shielding of imaging room. In view of short half-lives of most positron emitters, more efficient systems for radiopharmaceutical preparation and quality control are necessary.

41.5 RECOMMENDATIONS FOR DIAGNOSTIC USES

Separate areas should be available for the different types of activities. Area design must aid logical work flow:

Storage → radiopharmacy → administration → investigation

A separate patient waiting area with toilet facilities is recommended. It will rarely be necessary to establish a 'controlled area' around a patient in view of relatively low external radiation levels. For typical activities of ^{99m}Tc pharmaceuticals (50-500 MBq) the dose rates at 1 metre from the adult patient immediately after administration would be only around 0.2-2 μGy/h.

41.6 RECOMMENDATIONS FOR THERAPEUTIC USES

All forms of therapy involve substantial activities and doses to workers and the public require serious consideration. Where high activity gamma emitters are used, declaration of a 'controlled area' and retention of the patient in hospital are required. Design of the facility will depend on types of radionuclide and activity to be administered.

Laboratories should be close to wards. Wards for therapy patients would normally be a 'controlled area'. Therapy patients should be returned to the ward immediately after administration. There should be separate wards with adequate shielding, and separate toilets for therapy patients. Where smaller levels are administered (e.g., radioiodine for thyrotoxicosis), less stringent arrangements like keeping the patient in the corner of a ward (with mobile shield if required) may be acceptable. Radiation hazard warning notices should be prominently displayed. Persons entering the area should wear protective clothing.

Immediately after administration the following details should be entered in the patient's medical record: type and activity of radionuclide, dose and time administered, and dose rate measured at 1 m from the patient. An area within 1.5 m of the patient may need to be considered as a 'controlled area'. Patients should be discouraged from leaving the area. All items that have come into contact with the patient should be placed in a separate container. Disposable crockery/cutlery should be used. Only essential nursing should be provided to the patient. Attendants should have personal dosimeters and should wear disposable plastic gloves, and, on leaving the area, wash their hands and be monitored.

41.7 RECOMMENDATIONS FOR IN VITRO USES

In vitro uses involve a low degree of hazard, and the area will be generally classified as a 'supervised area', except in case iodination is carried out. Individual monitoring is not required except on an intermittent basis to confirm safe working conditions.

41.8 MISCELLANEOUS

41.8.1 Surgery

Surgical staff who have to handle a patient given a therapy dose (in case surgery cannot be delayed until the activity comes down to an acceptable level) should seek the advice of the Radiation Safety Officer. Concerned workers should wear personal dosimeters. If possible, organs containing high activity can be shielded during surgery to minimize worker exposure. On completion of surgery, the operating theatre, dressings and other materials should be monitored.

41.8.2 Autopsy, Cremation, Embalming

Advice of the Radiation Safety Officer should be taken. ICRP 57 gives details of maximum activities of radionuclides for disposal of corpses without special precautions. For example, in the case of ^{131}I, the limits for post-mortem/embalming are 10 MBq and for burial/cremation 400 MBq. (Based on Wrixon *et al.*, (1979); Wrixon and Linsley (1982); NRPB (1988)). Heavy autopsy gloves will reduce finger doses from beta emitters. Tissues removed for examination may have significant activity, in which case histological preparation and examination should be delayed appropriately. Autopsy rooms should be monitored on completion of the examination.

41.9 TECHNICAL ASPECTS OF PATIENT PROTECTION IN DIAGNOSTIC APPLICATIONS

The radiopharmaceutical should be appropriately chosen to optimize the diagnostic information in relation to the radiation dose, particularly for children. During imaging, the nuclear medicine physician should view the image as it is being acquired to ensure that necessary information is being acquired. It may be necessary to immobilize or sedate children.

A substantial reduction in patient dose can often be achieved by simple measures. Most radiopharmaceuticals are excreted via the urinary tract and the dose to bladder and gonads can be reduced by increasing fluid intake and frequent voiding, especially in the immediate interval following the examination. Blocking agents such as KI or $KClO_4$ are useful for ^{99m}Tc- or radioiodine-containing pharmaceuticals. Except in the case of thyroid imaging they can be given before the administration of the

radiopharmaceutical. They can even be given after the data have been acquired and still achieve a reduction in thyroid dose. Laxatives can increase elimination rate of tracers introduced or secreted into the GI tract (e.g., Ga citrate). Diuretics help to reduce kidney dose in renography and renal scintigraphy, while cholecystokinin (fatty meal) will reduce gall-bladder dose in hepatobiliary scintigraphy.

41.9.1 Women of Reproductive Capacity

In women of child-bearing age the possibility of pregnancy should be taken into account. The patients must be interviewed to assess the likelihood of pregnancy. If the menstrual period is irregular, a pregnancy test may be indicated before proceeding. Advisory notices should be posted in the department:

IF YOU THINK YOU MIGHT BE PREGNANT PLEASE NOTIFY STAFF.

41.9.2 Avoidance of Pregnancy after a Diagnostic Procedure

Since in today's nuclear medicine practice no diagnostic test gives doses to expose a subsequent embryo significantly, there is no reason to wait in case the woman wants to get pregnant.

41.9.3 Pregnant Women

It is important that the diagnostic procedure is justified in this case. Placental localization should preferably be done by ultrasound. Some radionuclides can cross the placenta and be taken up by fetal tissues, e.g., ^{131}I iodide or ^{99m}Tc pertechnetate can concentrate in fetal thyroid during the second and third trimesters of pregnancy. Radiocolloids retained in the maternal reticulo-endothelial system and radiopharmaceuticals that are eliminated by kidney and bladder can irradiate the fetus; hence frequent voiding should be ensured. The radiopharmaceutical can be administered when the bladder is partially filled, rather than immediately after voiding.

In the case of exposures at a time when pregnancy was unrecognized, the risk to the fetus is so small that a termination of pregnancy is rarely justified.

41.9.4 Breast-feeding Women

Consideration is to be given to postponing the diagnostic test if possible. To minimize exposures to the breast-fed child, advisory notices like "IF YOU ARE BREAST FEEDING PLEASE NOTIFY THE STAFF " should be posted in the department.

Nursing should be stopped for a minimum of the following periods following the administration of respective radionuclides noted against each period (Ahlgren *et al.* 1985; Coakley and Mountford, 1985):

3 weeks: All ^{125}I and ^{131}I radiopharmaceuticals (except hippuran), ^{22}Na, ^{67}Ga, ^{201}Tl and ^{75}Se-methionine.

12 hours: ^{123}I, ^{125}I, ^{131}I hippuran; most ^{99m}Tc pharmaceuticals.

4 hours: ^{99m}Tc RBC, phosphonate, DTPA.

41.9.5 Children

Particular care should be exercised in deciding on *in vivo* tests on children. For example, ^{67}Ga will irradiate epihyseal regions of growing bone (Gelfand *et al.*, 1983).

41.9.6 Protection of the Family

There is usually very little hazard to the patient's family. It is prudent to minimize prolonged intimate contact of the patient with the family during the first few hours after the test.

41.9.7 Misadministration

This is a rare occurrence in practice. A major type of mistake is the administration of a therapeutic dose when a diagnostic test was intended. (This is discussed later.) Errors may arise due to inadequate labelling, uncritical processing of nuclear medicine requisitions, and incorrect patient identification. After a misadministration, prompt attention should be given to the care of the patient and steps should be taken to mitigate the exposure.

41.10 TECHNICAL ASPECTS OF PATIENT PROTECTION IN THERAPEUTIC APPLICATIONS

Radiation therapy with unsealed sources is used for treatment of both benign and malignant diseases. Such uses may increase in the near future with new developments in immunotherapy.

The physician should be aware of the relative risks and benefits of all therapeutic methods. Radiation measurements and internal dosimetry are the responsibility of the medical physicist.

41.10.1 Treatment of Malignant Diseases

In radionuclide therapy, dose to some normal healthy tissues may approach or exceed the threshold for deterministic effects. Especially for children risks *vs.* benefits should be carefully evaluated. To aid better dosimetry and treatment planning, it is often helpful to administer a test dose to obtain information on biodistribution and effective half-lives in various organs.

41.10.2 Treatment of Benign Diseases

The age of the patient is an important consideration. For thyrotoxicosis therapy, it is important to make individual uptake and retention measurements initially to decide on the optimum therapeutic dose.

41.10.3 Late Effects of Radiation Therapy

There have been reports of increased leukaemia incidence after ^{32}P treatment for polycythemia vera and after ^{131}I therapy for thyroid cancers, but epidemiological evidence indicates negligible risks. Hereditary effects have not been established. (Most therapy patients are beyond childbearing age.)

Non-stochastic (deterministic) effects after radionuclide therapy include hypothyroidism after ^{131}I therapy for thyrotoxicosis, radiation pneumonitis and bone marrow depression after ^{131}I therapy for thyroid carcinoma, and bone marrow depression after ^{32}P therapy.

41.10.4 Pregnant Women

As a rule pregnant women should not undergo radionuclide therapy unless for life saving; in that event consideration should be given to terminating the pregnancy.

41.10.5 Women of Reproductive Capacity

Possibility of an undiagnosed pregnancy should be kept in mind. There have been reports of hypothyroidism in children born to mothers who had undergone ^{131}I therapy for thyrotoxicosis during pregnancy. Women should be advised on the interval that should elapse after therapy before attempts to become pregnant, so that the dose to the conceptus does not exceed 1 mGy; the interval may be 6 months for ^{131}I therapy for thyrotoxicosis.

41.10.6 Protection of the Family

Patients should not be released from hospital until the time when irradiation of a member of the family will not give rise to doses beyond 5 mGy. Even after discharge, the patient should avoid intimate contact with family members for an appropriate period. Breast feeding may have to be postponed.

41.10.7 Incidental Exposure of One Patient by Another

The risks are minimal. However, individual rooms for therapy patients, separate toilets, and frequent removal of radioactive wastes from the ward are desirable.

41.10.8 Therapeutic Misadministration

The maladministration of a therapeutic dose is of greater consequence than that of a diagnostic amount. When such a maladministration is recognized, all means to minimize the adverse effects should be taken immediately. Steps to hasten elimination of activity from the body include emesis, gastric lavage, laxatives or enemas for orally administered agents, hydration, diuresis and chelation therapy for intravenous administrations, catheterization to remove urine in the case of patients who cannot void spontaneously, and use of blocking agents like KI or $KClO_4$ for ^{99m}Tc- or radioiodine-labelled pharmaceuticals.

41.11 EDUCATION AND TRAINING

Education at the postgraduate level is valuable for physicians in training to appreciate the potentialities of and indications for nuclear medicine procedures. Education in patient protection should similarly form part of the educational programmes of the physicians. Concepts of efficacy and radiation protection should be interwoven into clinical presentations.

Appropriate specialized education and training are necessary for nuclear medicine physicians, medical physicists, technologists, radiopharmacists and nurses.

All nuclear medicine personnel should make every effort to give the patient a balanced view of radiation risks, so that a diagnostic examination that is clinically indicated will be acceptable to the patient.

41.12 RADIATION DOSES TO PATIENTS FROM RADIOPHARMACEUTICALS

41.12.1 Introduction

ICRP 53, 'Radiation dose to patients from radiopharmaceuticals' (1987) gives comprehensive data on the subject. It includes new radiopharmaceuticals which had come into use since ICRP publication 17, 'Protection of the patient in radionuclide investigations' (1971) which gave dose estimates for 92 compounds. ICRP 53 lists 290 sets of dose tabulations for 162 types of radiopharmaceuticals incorporating 74 radionuclides of 36 elements. It presents biokinetic models and biokinetic data as well as doses for individual radiopharmaceuticals. In addition to the organ doses (expressed in mGy/MBq), the effective doses E (in mSv/MBq), including the range of variation to be expected in some pathological states, are given for adults, and 15-, 10-, 5-, and 1-y old children. (These effective doses have been computed on the basis of the tissue weighting factors given in ICRP 26.) A little less than half of the radiopharmaceuticals contain radionuclides in ionic form, whereas the rest are labelled organic molecules or complexes, or are present in cells labelled with the radiopharmaceutical. The latter usually have a metabolic fate different from that of the ionic form.

As indicated earlier, ICRP 62 contains an addendum to ICRP 53 giving detailed biokinetic and dosimetric data for 6 new radiopharmaceuticals. The following changes have been introduced in the methodology of dosimetric computation:

(a) The effective doses have been calculated using the tissue weighting factors recommended by ICRP 60;

(b) An age-related bladder voiding model is used:

Age (y)	Newborn	1	5	10	15	Adult
Voiding period (h)	2.0	2.0	2.0	3.0	3.5	3.5

(c) Lower large intestine (LLI) is equated with colon for dosimetric calculations, while the upper large intestine (ULI) has been placed among 'remainder organs'.

The addendum to ICRP 53 also gives a table of recalculated effective doses for the case of normal adults from the radiopharmaceuticals listed in ICRP 53, using the revised tissue weighting factors given in ICRP 60.

41.12.2 General Considerations of Biokinetics and Dosimetry

Good quantitative biokinetic data on man are scarce. The clinician is often interested only in the initial metabolism and distribution, while for dosimetry more detailed information is necessary. ICRP 53 makes a plea for securing the maximum possible information on biokinetics from any investigation; this should be encouraged by scientific societies, editors/ referees of scientific journals and regulatory authorities.

For dosimetry, knowledge of the time-activity curve in different organs and tissues is needed. In most cases, a single exponential model for uptake/elimination is postulated (and in a few cases 2- or 3-exponentials). Intravenous administration with immediate uptake is assumed in most cases. In children, the same model as for the adult is used; this would lead to an overestimate of the dose due to the shorter biological half-life in children. Uniform distribution in the organ/tissue is assumed. The error in such an estimate would result in an error of less than 10% (except for Auger electron emitters incorporated in cell nuclei, where the risk may be underestimated).

Methods for calculating the doses are given in a brief form.

$$D(T \leftarrow S) = \tilde{A}_s \, . \, S(T \leftarrow S)$$

where D is the absorbed dose in target organ T from radioactivity in source organ S, $\tilde{A}_s$ the time-integrated or cumulative activity in source

organ S (equal to the total number of nuclear transformations in S), and S $(T \leftarrow S)$ the absorbed dose in T per unit cumulated activity in S.

$$\tilde{A}_s = \int_0^\infty A_s(u)\, du$$

where $A_S(u)$ is the activity at time u in the source organ.

Uncertainties in dose estimates arise due to uncertainties in the S-value and the cumulated activity. Dose estimates will not generally deviate from actual values by more than a factor of 3. The deviation is even less for short-lived radionuclides like ^{99m}Tc. The effective dose is less sensitive to variations in the distribution pattern than are organ doses and may vary by a factor of 2. (Roedler, 1981; Kaul *et al.*, 1984).

Under the section 'Biokinetic models' the following aspects are discussed: The models basically conform to those given in ICRP publication 30 with occasional variations. Organ and tissue masses are tabulated for different ages. Then follow descriptions of biokinetic models for various cases: Blood flow and blood volume, GI tract, kidney-bladder, glomerular filtration, lung, bone-seekers, colloids taken up in liver, spleen and red bone marrow, liver and biliary excretion, CSF, and short-lived positron emitters. Absorbed doses have been calculated in all cases for adrenals, bone surfaces, breast, GI tract (separately for stomach wall, small intestine wall, upper large intestine wall, lower large intestine wall), kidneys, liver, lungs, ovaries, pancreas, red bone marrow, spleen, testes, thyroid, bladder wall, uterus and 'other tissues' (e.g., muscle). Additional computations have also been made in cases where a particular organ or tissue receives significantly higher doses than the average to the rest of the body, or for organs or tissues of special interest to the investigation (brain, gall bladder wall, heart, salivary glands, spinal cord). Calculations have been specially made where specific S-values are not available (e.g., embryo, fetus, breast, gall bladder, salivary glands, and lymph nodes). The dose to fetus can be taken as that to the maternal uterus provided that there is no placental transfer of the radiopharmaceutical. For each radiopharmaceutical, detailed information is presented on the biokinetic model and biokinetic data, the latter incorporating values of F_S, the fractional distribution to organ/tissue S, 'a', the fraction of F_S taken up or eliminated with the corresponding half-life, and $\tilde{A}_S/A_0$, the cumulated activity in organ/tissue S per unit of administered activity.

In the case of radioiodine isotopes administered as iodide, values are given in ICRP 53 for a variety of uptake values from 0% to 55%. The tabulations include cases of intrathecal lumbar/cisternal injections; oral administration (in addition to the intravenous route) including non-absorb-

able markers in liquid and solid form; inhalation of gases with a single breathhold of some seconds as well as for continuous breathing; inhalation of aerosols with either a fast or a slow clearance from lungs; and colloids. Among the cases of abnormal physiology considered are abnormal renal function including unilateral renal blockage; parenchymal liver disease; occlusion of cystic duct and common bile duct; and congenital biliary atresia in newborn. Effective doses are given for the common radionuclide impurities found in radiopharmaceutical preparations.

41.12.3 Summary of Dose Tabulations

Table 41.2 (taken from addendum to ICRP 53 given in ICRP 62) lists the effective doses per unit administered activity (mSv/MBq) for normal adults; it includes radiopharmaceuticals listed in ICRP 53 as well as the newer ones listed in the addendum; these values are based on the revised ICRP 60 tissue weighting factors.

Table 41.3 (taken from ICRP 52 which in turn has used data from ICRP 53) presents absorbed doses to the 3 most highly irradiated organs, doses to the conceptus, and effective doses in the case of normal adults for major types of diagnostic investigations; the effective dose values, however, are the recalculated values given in addendum to ICRP 53.

The rest of this chapter is based on the author's analysis of the tabulations in ICRP 53 and its addendum.

41.12.4 Analysis of Effective Dose (E) Tabulations

The largest varieties of radiopharmaceuticals pertain to the following radionuclides: ^{99m}Tc, ^{131}I, ^{125}I, ^{51}Cr, ^{123}I, ^{111}In and ^{113m}In. Out of the total of 206 dose tabulations given in addendum to ICRP 53, the percentages that fall under different broad categories are as follows:

Dose range, mSv/MBq	% of Total
10-24	1.9
1-10	14.6
0.1-1	31.6
0.01-0.1	28.2
0.001-0.01	16.5
0.0001-0.001	7.3

Over 80% of the investigation types give E values less than 1 mSv/MBq.

(a) Investigations giving doses in the range 10-24 mSv/MBq are:

^{59}Fe (intravenous); ^{123,125,131}I sodium iodide in cases of high thyroid uptakes (for the same uptake, ^{131}I gives the highest dose followed in turn by ^{125}I and ^{123}I);

(b) Investigations giving 1-10 mSv/MBq are:

^{22}Na; ^{32}P; ^{45,47}Ca; ^{46}Sc; ^{52,55,59}Fe; ^{57,58}Co-labelled vitamin B_{12}; ^{65}Zn; ^{75}Se; ^{84,86}Rb; ^{89}Sr; some ^{131}I formulations; ^{198}Au colloid; and ^{203}Hg neohydrin;

(c) ^{99m}Tc radiopharmaceuticals give E values in the range 0.005-0.02 mSv/MBq;

(d) ^{111}In radiopharmaceuticals give doses in the range 0.02-0.4 mSv/MBq, while ^{113m}In radiopharmaceuticals give 0.01-0.03 mSv/MBq;

(e) ^{51}Cr radiopharmaceuticals give 0.002-0.18 mSv/MBq;

(f) ^{3}H inulin, and inhalation of ^{13}N, ^{15}O, or their compounds, ^{81m}Kr, ^{123,133}Xe gases give doses in the range of 10^{-4} mSv/MBq.

E values for oral administration in some cases are not very different from those for intravenous administration. For radioisotopes of iron, the oral route gives doses significantly smaller than for the intravenous route. For ^{51}Cr-EDTA, the intravenous route gives only 7% of that for oral administration. In the case of non-absorbable markers, the E values for fluids are equal to or about 5% smaller than those for solids. For intrathecal administration, the E values for lumbar injections are 5% to 60% greater than for cisternal injections.

As between normal and abnormal physiology, it is difficult to generalize about the variation in E values. For abnormal renal function the ratio of E values of abnormal to normal varies between 0.5 and 2. For diffuse parenchymal liver disease the E values for abnormal physiology are higher by 25-40% depending on the severity of the disease. For occlusion of the cystic duct and common bile duct the E values are 25% and 10-60% smaller respectively than for normals. For unilateral renal block the E values for the abnormal cases could be 4-35 times more.

In the case of radionuclide impurities, it is not again possible to generalize on the ratio of E values between MBq of impurity and MBq of the main radionuclide. The values vary from 0.009 to 3100.

41.12.5 Variation of *E* values with Age

ICRP 53 gives organ doses and *E* values for various ages expressed as mGy/MBq and mSv/MBq respectively. The doses, with an exception or two, show an increasing trend with decreasing age. This is mainly because the organ and total body weights decrease with decreasing age. Hence, if the source and target organs are the same, for unit cumulated activity, the dose would be inversely proportional to the mass of the organ for non-penetrating radiations.

However, the normal practice would be to administer a specific activity per unit body weight (expressed as, say, MBq/kg body weight). It would therefore be logical to normalize the dose values for any age as per the following formula:

$$\text{Normalized dose at age } y = \text{Adult dose} \times \frac{\text{Body wt. at age } y}{\text{Adult body wt.}}$$

We can also derive the normalized dose ratio (NR) by the following formula:

$$\text{NR} = \frac{\text{Dose at age } y}{\text{Adult dose}} \times \frac{\text{Body wt. at age } y}{\text{Adult body wt.}}$$

The total body weights for adult, 15-, 10-, 5- and 1-year olds are given in ICRP 53 as 70.0, 56.8, 33.2, 19.8 and 9.72 kg respectively.

A detailed analysis shows that NR values are, by and large, independent of the age. This means that if the same activity per unit body weight is administered, *E* is independent of age. For all ages, over 80% of all NR values lie within ± 30%. There is a general tendency for NR to decrease with decreasing age. The reason for this trend is the greater metabolic turnover rate with decreasing age. This trend is particularly noticeable for some radiopharmaceuticals where NR can come down from 1.00 for the adult to as low as 0.4-0.6 for the 1-year old; it includes tritiated water, radiocobalt-labelled vitamin B_{12}, some technetium and indium-labelled pharmaceuticals, and all cesium isotopes.

There are also some exceptions to the above trend (which includes ^{99m}Tc pertechnetate). For radioisotopes of iodine administered as iodide,

there is a general increase in NR from 20-40% for lower ages as compared to adult values, but there is some anomalous behaviour at some ages.

41.12.6 Organs Getting High Doses

The percentage of cases (out of the total dose tabulations given in ICRP 53) where a specific organ gets doses greater than twice the effective dose and/or greater than 1 mGy/MBq (for the case of the adult) are as follows:

Bladder wall - 22% of the tabulations; liver, spleen, upper large intestine wall, lower large intestine wall, kidneys - around 15% each; lungs, thyroid, red marrow - around 10% each.

The types of investigations which give organ doses above 10 mGy/MBq and the organs getting such doses are:

^{32}P and ^{45}Ca -- bone surface and red marrow;
^{57}Co and ^{58}Co vitamin B_{12} -- liver, adrenals, pancreas;
^{55}Fe and ^{59}Fe -- heart, spleen, lungs;
^{75}Se selenite -- kidneys;
^{84}Rb, ^{86}Rb -- liver, kidneys;
^{89}Sr -- bone surface, red marrow;
^{124}I, ^{125}I, ^{131}I iodide - thyroid (40-800 mGy/MBq depending on the isotope and uptake);
^{198}Au colloid - spleen.

41.12.7 Variation of Organ Doses with Age

We have already established that the normalized effective dose ratio is reasonably independent of age. The ratio of organ dose to E would therefore be similarly independent of age provided that the ratio of organ weight to total body weight does not significantly depend on age. An examination of the data given in ICRP 53 shows that this can be broadly taken as true for the majority of organs correct to + 20%. (Exceptions are: breast, uterus, ovaries, and testes, where there is a sudden growth in the organs around the age 15. Muscle and thyroid also show a somewhat similar, but smaller, trend in extent. On the other hand, adrenals, kidneys and trabecular bone show a relatively larger organ weight per unit body weight with decreasing age.)

A detailed examination shows that the ratio of the organ dose to the effective dose is indeed independent of age to within 30-40%. Certain

trends are noticeable. For bone surfaces and red marrow there is an increase in this ratio with decreasing age, while for adrenals and brain, the reverse is the case. (In the case of thyroid, it is the thyroid dose which almost completely determines the effective dose; the variation of thyroid dose with age is similar to that of *E*.)

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Table 41.1: Derived levels for surface contamination
(from Wrixon *et al.* (1979) and Wrixon and Linsley (1982), quoted in ICRP 57)

Surface	Radionuclide class (Bq cm^{-2})		
	A	B	C
Surface and equipment in controlled area (averaged over 1000 cm^2)	30	300	3000
Surfaces of the body (averaged over 100 cm^2)	3*	30	300
Supervised and public areas, personal clothing, hospital bedding (averaged over 300 cm^2)	3	30	300

* Use a tenth of this value for alpha emitters.
Note: The above levels do not apply to volatile compounds which can readily penetrate the skin.

Table 41.2: Effective doses per unit administered activity for normal adults
(from Addendum to ICRP 53, ICRP 62)

Radio-nuclide	Substance	*E* (mSv/MBq)
^{3}H	Water	1.5E-02
^{3}H	Inulin	9.4E-04
^{3}H	Neutral fat and free fatty acids	2.2E-01
^{11}C	Carbon monoxide (single inhalation with 20 s breath-hold)	4.8E-03
^{11}C	Carbon monoxide (continuous inhalation for 1 h)	3.2E-03
^{11}C	Carbon dioxide (single inhalation with 20 s breath-hold)	1.6E-03
^{11}C	Carbon dioxide (continuous inhalation for 1 h)	1.0E-03
^{11}C	COHb-labelled erythrocytes	5.0E-03
^{11}C	Spiperone	5.3E-03
^{14}C	Inulin	8.2E-03
^{14}C	Neutral fat and free fatty acids	2.1E+00
^{13}N	Nitrogen gas (single inhalation with 20 s breath-hold)	3.8E-04
^{13}N	Nitrogen gas (continuous inhalation for 1 h)	4.3E-04
^{13}N	Nitrogen gas in solution	4.1E-04
^{13}N	Ammonia	2.0E-03
^{13}N	l-glutamate	3.9E-03
^{15}O	Carbon monoxide (single inhalation with 20 s breath-hold)	8.0E-04
^{15}O	Carbon monoxide (continuous inhalation for 1 h)	5.5E-04
^{15}O	Carbon dioxide (single inhalation with 20 s breath-hold)	5.1E-04
^{15}O	Carbon dioxide (continuous inhalation for 1 h)	3.8E-04
^{15}O	Oxygen gas (single inhalation with 20 s breath-hold)	3.7E-04
^{15}O	Oxygen gas (continuous inhalation for 1 h)	4.0E-04
^{18}F	Fluoride	2.4E-02
^{18}F	2-fluoro-2-deoxy-d-glucose (FDG)	2.0E-02
^{22}Na	Sodium (intravenous or oral administration)	2.6E+00
^{24}Na	Sodium	3.2E-01
^{24}Na	Sodium (oral administration)	3.6E-01
^{28}Mg	Magnesium	7.2E-01
^{32}P	Phosphate	2.4E+00
^{33}P	Phosphate	6.6E-01
^{35}S	Sulphate	9.0E-02
^{34m}Cl	Chloride	1.4E-02
^{36}Cl	Chloride	6.7E-01
^{38}Cl	Chloride	1.4E-02
^{38}K	Potassium (ultrashort-lived)	1.9E-02
^{42}K	Potassium	2.8E-01
^{42}K	Potassium (oral administration)	3.4E-01
^{43}K	Potassium	2.0E-01
^{43}K	Potassium (oral administration)	2.2E-01

(contd ...)

Radio-nuclide	Substance	*E* (mSv/MBq)
^{45}Ca	Calcium	3.1E+00
^{45}Ca	Calcium (oral administration)	1.8E+00
^{47}Ca	Calcium	1.2E+00
^{47}Ca	Calcium (oral administration)	1.8E+00
^{46}Sc	Sc-labelled non-absorbable markers (fluids)	1.6E+00
^{46}Sc	Sc-labelled non-absorbable markers (solids)	1.7E+00
^{47}Sc	Sc-labelled non-absorbable markers (fluids)	4.7E-01
^{47}Sc	Sc-labelled non-absorbable markers (solids)	7.6E-01
^{51}Cr	Chromium (III) chloride	6.8E-02
^{51}Cr	Chromium EDTA	2.1E-03
^{51}Cr	Chromium EDTA (oral administration)	4.4E-02
^{51}Cr	Cr-labelled platelets (thrombocytes)	1.4E-01
^{51}Cr	Cr-labelled erythrocytes	1.7E-01
^{51}Cr	Cr-labelled denatured erythrocytes	1.8E-01
^{51}Cr	Cr-labelled white blood cells (leukocytes)	1.2E-01
^{51}Cr	Cr-labelled non-absorbable markers (fluids)	4.3E-02
^{51}Cr	Cr-labelled non-absorbable markers (solids)	4.5E-02
^{52}Fe	Iron	1.1E+00
^{52}Fe	Iron (oral administration)	7.1E-01
^{55}Fe	Iron	4.0E+00
^{55}Fe	Iron (oral administration)	4.2E-01
^{59}Fe	Iron	1.0E+01
^{59}Fe	Iron (oral administration)	2.0E+00
^{57}Co	Co-labelled bleomycin	4.7E-02
^{57}Co	Vitamin B12 (intravenous injection with no carrier)	4.4E+00
^{58}Co	Vitamin B12 (intravenous injection with no carrier)	8.2E+00
^{57}Co	Vitamin B12 (intravenous injection with carrier)	4.6E-01
^{58}Co	Vitamin B12 (intravenous injection with carrier)	8.9E-01
^{57}Co	Vitamin B12 (oral administration with flushing)	2.1E+00
^{58}Co	Vitamin B12 (oral administration with flushing)	4.0E+0(
^{57}Co	Vitamin B12 (oral administration without flushing)	3.1E+0(
^{58}Co	Vitamin B12 (oral administration without flushing)	5.9E+0(
^{64}Cu	Copper	3.6E-02
^{67}Cu	Copper	1.5E-01
^{62}Zn	Zinc	3.5E-01
^{65}Zn	Zinc	8.4E+00
^{69m}Zn	Zinc	1.4E-01
^{66}Ga	Gallium citrate	3.2E-01
^{67}Ga	Gallium citrate	1.1E-01
^{68}Ga	Gallium citrate	2.0E-02

(contd ...)

Radio-nuclide	Substance	*E* (mSv/MBq)
^{68}Ga	Elthylene diamine tetraacetic acid (EDTA)	4.0E-02
^{72}Ga	Gallium citrate	3.4E-01
^{72}As	Arsenate, arsenite	3.6E-01
^{74}As	Arsenate, arsenite	5.1E-01
^{76}As	Arsenate, arsenite	2.8E-01
^{75}Se	Selenite	2.6E+00
^{75}Se	Selenomethylcholesterol	1.5E+00
^{75}Se	l-Selenomethionine	2.5E+00
^{75}Se	Selenium-labelled bile acid (SeHCAT)	6.6E-01
^{76}Br	Bromide	2.8E-01
^{77}Br	Bromide	7.7E-02
^{82}Br	Bromide	4.0E-01
^{77}Br	Bromospiperone	8.5E-02
^{81m}Kr	Krypton	9.5E-04
^{81}Rb	Rubidium	2.8E-02
^{82}Rb	Rubidium	3.4E-03
^{84}Rb	Rubidium	2.8E+00
^{86}Rb	Rubidium	3.0E+00
^{81}Rb	Rb-labelled denatured erythrocytes	1.4E-01
^{85}Sr	Strontium	7.9E-01
^{87m}Sr	Strontium	6.4E-03
^{89}Sr	Strontium	3.1E+00
^{99m}Tc	Tc-labelled albumin (HSA)	6.1E-03
^{99m}Tc	Tc-labelled citrate complex	6.1E-03
^{99m}Tc	Tc-labelled large colloids	9.2E-03
^{99m}Tc	Te-labelled small colloids	9.7E-03
^{99m}Tc	Tc-DMSA	8.7E-03
^{99m}Tc	Tc-DTPA	5.2E-03
^{99m}Tc	Tc-labelled plasmin	7.3E-03
^{99m}Tc	Tc-gluconate, glucoheptonate	5.4E-03
^{99m}Tc	Tc-penicillamine	7.3E-03
^{99m}Tc	Pertechnetate	1.2E-02
^{99m}Tc	Pertechnetate (blocking agent given)	4.7E-03
^{99m}Tc	Pertechnetate (oral administration, no blocking)	1.4E-02
^{99m}Tc	Tc-labelled IDA derivatives	1.5E-02
^{99m}Tc	Tc-labelled fibrinogen	6.2E-03
^{99m}Tc	Tc-labelled erythrocytes	6.6E-03
^{99m}Tc	Tc-labelled denatured erythrocytes	1.9E-02
^{99m}Tc	Tc-labelled phosphates and phosphonates	5.8E-03
^{99m}Tc	Tc-labelled aerosols (substances with fast clearance)	6.1E-03

(contd ...)

Radio-nuclide	Substance	*E* (mSv/MBq)
^{99m}Tc	Tc-labelled aerosols (substances with slow clearance)	1.4E-02
^{99m}Tc	Tc-labelled heparin	5.5E-03
^{99m}Tc	Tc-labelled macroaggregated albumin	1.1E-02
^{99m}Tc	Tc-labelled non-absorbable markers (fluids)	2.0E-02
^{99m}Tc	Tc-labelled non-absorbable markers (solids)	2.2E-02
^{99m}Tc	Tc-labelled albumin microspheres	1.0E-02
^{99m}Tc	Tc-labelled platelets (thrombocytes)	1.2E-02
^{99m}Tc	Tc-labelled white blood cells (leukocytes)	1.1E-02
^{99m}Tc	Hexamethylpropylene amineoxine (HMPAO)	9.3E-03
^{99m}Tc	Mercaptoacetyltriglycerine (MAG3)	7.3E-03
^{99m}Tc	Methyl oxy-butyl-isonitrile (MIBI)	8.5E-03
^{111}In	Indium	2.1E-01
^{113m}In	Indium	1.0E-02
^{113m}In	Indium hydroxide (colloidal)	1.1E-02
^{111}In	In-DTPA	2.1E-02
^{113m}In	In-DTPA	1.1E-02
^{111}In	In-aerosols (substances with fast clearance)	2.5E-02
^{111}In	In-aerosols (substances with slow clearance)	2.4E-01
^{113m}In	In-aerosols (substances with fast clearance)	1.6E-02
^{113m}In	In-aerosols (substances with slow clearance)	2.5E-02
^{111}In	In-labelled non-absorbable markers (fluids)	3.1E-01
^{111}In	In-labelled non-absorbable markers (solids)	3.2E-01
^{113m}In	In-labelled non-absorbable markers (fluids)	2.0E-02
^{113m}In	In-labelled non-absorbable markers (solids)	2.9E-02
^{111}In	In-labelled platelets (thrombocytes)	3.9E-01
^{111}In	In-labelled white blood cells (leukocytes)	3.6E-01
^{111}In	In-labelled bleomycin	1.0E-01
^{123}I	Iodide (thyroid blocked, uptake 0%)	1.1E-02
^{123}I	Iodide (thyroid uptake 35%)	2.2E-01
^{124}I	Iodide (thyroid blocked, uptake 0%)	9.5E-02
^{124}I	Iodide (thyroid uptake 35%)	1.5E+01
^{125}I	Iodide (thyroid blocked, uptake 0%)	9.1E-03
^{125}I	Iodide (thyroid uptake 35%)	1.4E+01
^{131}I	Iodide (thyroid blocked, uptake 0%)	6.1E-02
^{131}I	Iodide (thyroid uptake 35%)	2.4E+01
^{123}I	Iodoamphetamine (IMP)	2.7E-02
^{123}I	Iodine-labelled fibrinogen	2.0E-02
^{125}I	Iodine labelled fibrinogen	8.0E-02
^{131}I	Iodine-labelled fibrinogen	4.2E-01
^{123}I	Iodine-labelled albumin (HSA)	2.0E-02

(contd ...)

Radio-nuclide	Substance	*E* (mSv/MBq)
^{125}I	Iodine-labelled albumin (HSA)	2.2E-01
^{131}I	Iodine-labelled albumin (HSA)	6.4E-01
^{131}I	Iodine-labelled macroaggregated albumin (MAA)	4.5E-01
^{125}I	Iodine-labelled non-absorbable markers (fluids)	1.7E-01
^{125}I	Iodine-labelled non-absorbable markers (solids)	1.7E-01
^{131}I	Iodine-labelled non-absorbable markers (fluids)	1.2E+00
^{131}I	Iodine-labelled non-absorbable markers (solids)	1.2E+00
^{123}I	Iodine-labelled microaggregated albumin (MIAA)	1.8E-02
^{131}I	Iodine-labelled microaggregated albumin (MIAA)	2.2E-01
^{123}I	Hippuran	1.2E-02
^{125}I	Hippuran	7.7E-03
^{131}I	Hippuran	5.3E-02
^{131}I	Iodo-antipyrine	6.7E-02
^{125}I	Iodo-antipyrine	1.0E-02
^{125}I	Iothalamate	7.2E-03
^{131}I	Iodomethyl-19-norcholesterol (NP 59)	1.8E+00
^{125}I	Iodinated polyvinylpyrrolidone (PVP)	6.5E-01
^{131}I	Iodinated polyvinylpyrrolidone (PVP)	6.0E-01
^{125}I	Thyroxine (T4)	1.0E-01
^{131}I	Thyroxine (T4)	4.4E-01
^{125}I	Triiodothyronine (T3)	4.7E-02
^{131}I	Triiodothyronine (T3)	3.0E-01
^{125}I	Reverse triiodothyronine (rT3)	3.7E-02
^{131}I	Reverse triiodothyronine (rT3)	2.5E-01
^{125}I	Diiodothyronine	3.6E-02
^{131}I	Diiodothyronine	2.5E-01
^{123}I	Metaiodobenzylguanidine (MIBG)	1.4E-02
^{131}I	Metaiodobenzylguanidine (MIBG)	1.4E-01
^{123}I	Sodium Rose Bengal	5.9E-02
^{131}I	Sodium Rose Bengal	1.1E+00
^{127}Xe	Xenon-gas (single inhalation or i.v.inj., 30 s breath-hold)	1.3E-04
^{133}Xe	Xenon-gas (single inhalation or i.v.inj., 30 s breath-hold)	1.8E-04
^{127}Xe	Xenon-gas (rebreathing for 5 min)	7.1E-04
^{127}Xe	Xenon-gas (rebreathing for 10 min)	1.1E-03
^{133}Xe	Xenon-gas (rebreathing for 5 min)	7.3E-04
^{133}Xe	Xenon-gas (rebreathing for 10 min)	1.1E-03
^{129}Cs	Caesium	4.9E-02
^{130}Cs	Caesium	3.4E-03
^{131}Cs	Caesium	5.0E-02
^{133m}Cs	Caesium	6.7E-03

(contd ...)

Radio-nuclide	Substance	*E* (mSv/MBq)
^{131}Ba	Barium	5.0E-01
^{133m}Ba	Barium	4.7E-01
^{135m}Ba	Barium	3.4E-01
^{131}Ba	Ba-labelled non-absorbable markers (fluids)	4.9E-01
^{131}Ba	Ba-labelled non-absorbable markers (solids)	5.1E-01
^{140}La	La-DTPA	1.5E-01
^{169}Yb	Yb-DTPA	3.6E-02
^{198}Au	Gold colloid	1.1E+00
^{197}Hg	Mercury chloride	1.4E-01
^{197}Hg	Bromo-mercuri-hydroxypropane (BMHP)	1.4E-01
^{197}Hg	Chlormerodrin (neohydrin)	8.7E-02
^{203}Hg	Chlormerodrin (neohydrin)	1.1E+00
^{201}Tl	Thallium	2.3E-01

Table 41.3: Absorbed doses to most highly exposed organs and effective dose per unit administered activity in normal adults for major diagnostic investigation types

(from ICRP 52; effective dose values are from addendum to ICRP 53, ICRP 62)

Organ 1 (mGy/MBq)	Organ 2 (mGy/MBq)	Organ 3 (mGy/MBq)	Conceptus (mGy/MBq)	Eff. dose (mSv/MBq)
		BONE		
		^{99m}Tc phosphate/phosphonate		
Bone surface 0.063	Bladder 0.050	Bone marrow 0.0096	0.0061	0.0058
		RENAL		
		^{51}Cr EDTA		
Bladder 0.023	Uterus 0.0028	Kidney 0.0018	0.0028	0.0021
		^{123}I hippuran		
Bladder 0.20	Uterus 0.017	LLI 0.0073	0.017	0.012
		^{131}I hippuran		
Bladder 0.96	Uterus 0.035	Kidney 0.030	0.035	0.053
		^{99m}Tc DTPA		
Bladder 0.065	Uterus 0.0079	Kidney 0.0044	0.0079	0.0052
		^{99m}Tc DMSA		
Kidney 0.17	Bladder 0.019	Adrenal, spleen 0.013	0.0046	0.0097
		THYROID		
		^{99m}Tc pertechnetate (no blocking)		
ULI 0.062	Stomach 0.029	Thyroid 0.023	0.0081	0.014
		^{131}I iodide (35% uptake)		
Thyroid 500	Stomach 0.46	Bladder 0.40	0.050	24

(contd ...)

Organ 1 (mGy/MBq)	Organ 2 (mGy/MBq)	Organ 3 (mGy/MBq)	Conceptus (mGy/MBq)	Eff. dose (mSv/MBq)
		^{123}I iodide (35% uptake)		
Thyroid 4.5	Stomach 0.068	Bladder 0.060	0.014	0.22
		LIVER (+ GALL BLADDER)		
		^{99m}Tc colloid (large)		
Spleen 0.077	Liver 0.074	Pancreas 0.012	0.0019	0.0092
		^{99m}Tc millimicrospheres		
Spleen 0.077	Liver 0.074	Bone marrow 0.015	0.0018	0.0097
		^{99m}Tc HIDA		
Gall bladder 0.11	ULI 0.092	LLI 0.062	0.013	0.015
		^{57}Co Vitamin B_{12} (no carrier)		
Liver 51	Adrenals, pancreas 5.4	Kidney 5.0	1.8	4.4
		BRAIN		
		^{99m}Tc pertechnetate (blocked thyroid)		
Bladder 0.032	Uterus 0.0063	Kidney, ovary 0.0047	0.0066	0.0047
		^{99m}Tc DTPA (lumbar)		
Spinal cord 0.046	Bone marrow 0.029	Kidney, bladder 0.017	0.0045	0.010
		^{99m}Tc gluconate/glucoheptonate		
Bladder 0.056	Kidney 0.049	Uterus 0.0077	0.0077	0.0054
		^{18}FDG		
Bladder 0.17	Heart 0.065	Brain 0.026	0.020	0.020

(contd ...)

Organ 1 (mGy/MBq)	Organ 2 (mGy/MBq)	Organ 3 (mGy/MBq)	Conceptus (mGy/MBq)	Eff. dose (mSv/MBq)
		LUNG		
		^{99m}Tc MAA		
Lung 0.067	Liver 0.016	Bladder 0.010	0.0024	0.011
		^{99m}Tc aerosol (fast clearance)		
Bladder 0.047	Lung 0.017	Uterus 0.0059	0.0059	0.0061
		^{99m}Tc aerosol (slow clearance)		
Lung 0.093	Bladder 0.013	Breast 0.0064	0.0017	0.014
		^{133}Xe gas, 5 min (re-breathing)		
Lung 0.0011	Bone marrow 0.00084	Breast 0.00083	0.00074	0.00073
		^{133}Xe gas, 30 s (one breath)		
Lung 0.00077	Bone surfaces, bone marrow, breast 0.00012	SI, large intestine, liver, pancreas, spleen, uterus 0.00011	0.00011	0.00018
		^{81m}Kr gas		
Lung 2.1×10^{-4}	Breast 4.6×10^{-6}	Pancreas 3.5×10^{-6}	1.3×10^{-7}	0.00095
		HEART		
		^{201}Tl (thallous ion)		
Testes 0.56	Kidney 0.54	LLI 0.36	0.050	0.23

(contd ...)

Organ 1 (mGy/MBq)	Organ 2 (mGy/MBq)	Organ 3 (mGy/MBq)	Conceptus (mGy/MBq)	Eff. dose (mSv/MBq)
		^{99m}Tc RBC		
Heart 0.023	Spleen 0.015	Lung 0.014	0.0047	0.0066
		ABSCESS		
		^{111}In white cells		
Spleen 5.5	Liver 0.71	Bone marrow 0.69	0.12	0.36
		^{67}Ga citrate		
Bone surface 0.59	LLI 0.20	Bone marrow 0.19	0.079	0.11
		THROMBI		
		^{125}I fibrinogen (thyroid totally blocked)		
Heart 0.32	Spleen 0.24	Lung 0.23	0.055	0.080
		^{111}In platelets		
Spleen 7.5	Liver 0.73	Pancreas 0.66	0.095	0.39
		^{51}Cr platelets		
Spleen 2.6	Liver 0.30	Bone marrow 0.19	0.028	0.14
		PANCREAS		
		^{75}Se methionine		
Liver 6.2	Kidney 5.3	Spleen 3.9	2.6	2.5

(contd ...)

Organ 1 (mGy/MBq)	Organ 2 (mGy/MBq)	Organ 3 (mGy/MBq)	Conceptus (mGy/MBq)	Eff. dose (mSv/MBq)
		ADRENALS		
		^{75}Se methyl cholesterol		
Adrenals 5.1	Liver 2.0	Pancreas, uterus, SI, bone marrow 1.8	1.8	1.5
		^{131}I MIBG		
Liver 0.84	Bladder 0.59	Spleen 0.49	0.077	0.14
		SPLEEN		
		^{51}Cr RBC denatured		
Spleen 5.6	Pancreas 0.30	Liver 0.17	0.013	0.18
		GIT		
		^{99m}Tc pertechnetate (oral, no blocking agent)		
ULI 0.074	Stomach 0.050	SI 0.030	0.0087	0.014

Note: SI - small intestine; ULI - upper large intestine; LLI - lower large intestine

Part VI

Exposures to Radon, Thoron and Their Daughter Products

CHAPTER 42: SECTION 1

INTRODUCTION TO THE RADON EXPOSURE PROBLEM

42(1).1 BACKGROUND

Radiogenic lung cancer is the oldest type of radiation-induced malignancy known (ICRP 50, 1987). It was recorded in the 15th and 16th centuries among miners in the Schneeberg region. The so-called 'Schneeberger Krankheit' was diagnosed as lung cancer in 1879. Possible association with radon was suggested around 60 years ago when high levels of radon were discovered in these mines. The real cause, viz. inhalation of short-lived radon daughters, was recognized only in the 1950s when the first attempts at lung dosimetry were made.

It is only over the last 15 years or so that we have come to realize that every member of the human population is receiving substantial doses from the naturally present and ubiquitous radon and daughters. In fact, half of the total effective dose from natural background radiation comes from this source. Both the individual and collective doses from radon and its progeny are higher than from almost any other source. Further, the contribution from this source is highly variable, depending on a variety of factors. In many countries, there are some individuals who receive doses from this natural source which are substantially higher than those that would be permitted in occupational exposure.

It is therefore not surprising that ICRP as well as other scientific bodies are devoting considerable attention to radon exposures, both to occupational workers and to members of the public.

ICRP publication 24, 'Radiation protection in uranium and other mines' (1977) (which has been superseded by ICRP 47), ICRP publication 32, 'Limits for inhalation of radon daughters by workers' (1981), ICRP publication 47, 'Radiation protection of workers in mines' (1985) and ICRP publication 50, 'Lung cancer risk from indoor exposures to radon daughters' (1987) are devoted entirely to this subject. In addition, ICRP publication 39, 'Principles for limiting exposures of the public to natural sources

of radiation' (1984) gave guidelines on principles for limiting exposures of members of the public from radon, with special reference to regions where such levels are high, distinguishing between existing situations which can only be influenced by remedial action, and future situations which can be brought under administrative control. ICRP publication 60 (1991) has also devoted attention to the subject, and has mentioned that revised occupational levels are under review.

42(1).2 RADON IN THE ENVIRONMENT

Radon is produced by the decay of radium which is a member of the ^{238}U decay series. Uranium occurs to the extent of 3 ppm in the earth's crust, and so radon occurs everywhere. Radon exhaled from ore bodies migrates through rock and is exhaled from the earth's surface or mine surfaces continuously. Radon exhaled into the free atmosphere is rapidly dispersed and diluted by vertical convection and turbulence. Considerably higher levels can occur if the radon is released in confined air spaces such as underground mines and homes. In such areas the radon level increases with decreasing ventilation rate. The radon level in the free atmosphere is about one-thousandth of that in the soil air.

Radon, ^{222}Rn, has a half-life of 3.8 days and decays by emission through a chain of short-lived solid daughter products till it reaches the long-lived 22-y half-life ^{210}Pb (RaD), which undergoes further decays to end up in stable ^{206}Pb. Radon and some of its short-lived daughter products (3.1 min ^{218}Po or RaA, and 0.16 ms ^{214}Po or RaC') are alpha emitters. After formation, the short-lived daughters exist briefly in atomic form, before becoming attached to aerosol particles, particularly in the sub-micron range, or solid surfaces. In aerosol form they tend to remain suspended, although they are subject to electrostatic, inertial and diffusive forces by which they can be removed. Inside rocks and soil the daughters will be in radioactive equilibrium with the parent.

Thoron, ^{220}Rn, is similar to radon, and is formed by the decay of ^{224}Ra in the ^{232}Th series. While the masses of uranium and thorium per unit mass of rock are similar, the relative importance of thoron is less than that of radon, since its half-life is only 55 s, and also because the specific activity of thorium in ore bodies is less than that of uranium. Thoron, like radon, is an alpha emitter; in its chain of decay, the short-lived daughter products, viz. 0.15 s ^{216}Po or ThA, 60 min ^{212}Bi or ThC, and 300 ns ^{212}Po or ThC', are alpha emitters. The end product of the series is stable ^{208}Pb. Figure 42(1).1 gives the radon and thoron decay series.

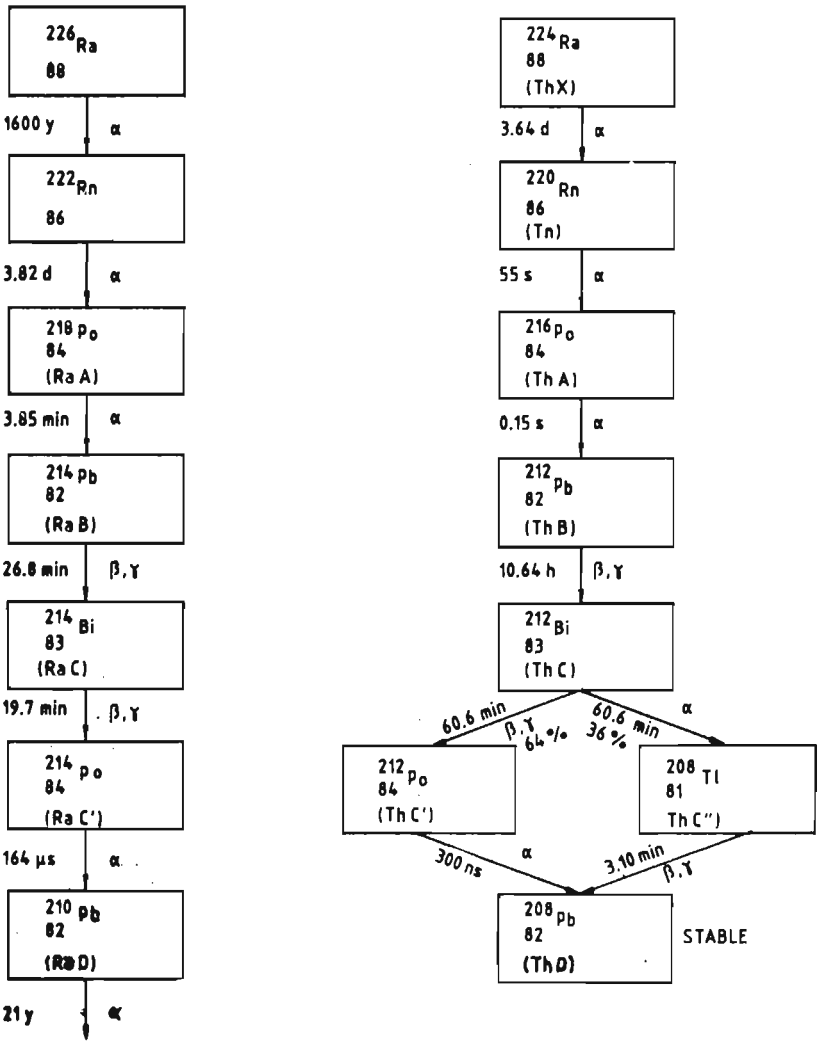

Figure 42(1).1. Radon and thoron decay series.

42(1).3 RADON IN MINES

Uranium miners are exposed to radon, thoron, and their short-lived decay products, ore dust, external gamma and beta radiations. Even in non-uranium mines there could be substantial levels of radon.

Radon produced by decay of radium in ore bodies migrates through rock and emanates continuously from mine surfaces into air spaces, is then carried along in ventilation currents and produces solid daughter products. Because of its long half-life compared with the residence time of air in a mine, radon and daughter concentrations increase in proportion to the rate of emanation and travel time along mine passages. Due to various factors, the concentration and relative proportion of the daughter product mixture vary considerably within the mine.

Appendix 42(3).1 summarizes the operational radiation protection aspects in uranium mines. Radon levels in indoor and outdoor air in the normal human environment are dealt with in Chapter 42, Section 4.

CHAPTER 42: SECTION 2

SPECIAL QUANTITIES AND UNITS USED IN RADON EXPOSURES

Due to historical reasons, several special quantities and units have been developed in relation to radon exposures in mines and this terminology has also been carried over in discussions on all radon exposures in general.

42(2).1 POTENTIAL ALPHA ENERGY (ε_p)

The potential alpha energy, ε_p, of an atom of radon or thoron is the total alpha energy emitted during the decay of this atom along its decay chain of short-lived daughters in equilibrium with it down to ^{210}Pb or ^{208}Pb respectively. The total potential alpha energy per Bq of activity is the potential alpha energy of the atom divided by the decay constant, i.e. $\varepsilon_p / \lambda_r$, where the decay constant λ_r is expressed in s^{-1}.

42(2).2 POTENTIAL ALPHA ENERGY CONCENTRATION IN AIR (c_p)

The potential alpha energy concentration in air of any mixture of short-lived radon or thoron daughters, c_p, is the sum of the potential alpha energy of all daughter atoms present per unit volume of air. If $c_{act,i}$ is the activity concentration of a daughter nuclide i in air, then

$$c_p = \sum_i c_{p,i} = \sum_i c_{act,i} \cdot \varepsilon_{p,i} / \lambda_r$$

summed over all short-lived daughters up to ^{210}Pb and ^{208}Pb respectively. It is expressed in $J.m^{-3}$.

$1\ J.m^{-3} = 6.24 \times 10^{12}\ MeV.m^{-3}$.

The special unit Working Level (WL) is often used for this quantity. 1 WL corresponds to a potential alpha energy concentration of 1.3×10^5 MeV/l. It corresponds approximately to the potential alpha energy con-

centration of short-lived daughters in air in equilibrium with a radon activity concentration of 100 pCi/l (or 3.7 Bq/l). (For thoron daughters, 1 WL corresponds to a thoron concentration of 7.43 pCi/l or 0.275 Bq/l.)

Table 42(2).1 gives values of the potential alpha energy per atom and per Bq, as well as the potential alpha energy concentrations in air for the short-lived daughters of radon and thoron.

42(2).3 EQUILIBRIUM-EQUIVALENT CONCENTRATION (EEC) IN AIR

The EEC of a non-equilibrium mixture of radon (or thoron) daughters in air is that activity concentration of radon (or thoron) in equilibrium with its daughters which has the same potential alpha energy concentration as the actual non-equilibrium mixture.

$$EEC_{Rn} = 1.81 \times 10^{8} . c_p$$

$$EEC_{Tn} = 1.32 \times 10^{7} . c_p$$

where EEC_{Rn} and EEC_{Tn} are the equilibrium-equivalent concentrations of radon and thoron respectively in $Bq.m^{-3}$, and c_p is the potential alpha energy concentration of radon or thoron daughters in $J.m^{-3}$.

42(2).4 EQUILIBRIUM FACTOR (F) IN AIR

It is the ratio of the EEC to the actual activity concentration c_{act} of the mother nuclide in air.

$$F_{Rn\ daughters} = EEC_{Rn} / c_{act.\ Rn}$$

$$F_{Tn\ daughters} = EEC_{Tn} / c_{act.\ Tn}$$

42(2).5 RADON DAUGHTER EXPOSURE (E)

The radon daughter exposure E of an individual is the time integral of the potential alpha energy concentration c_p of the daughter mixture in air, or the corresponding EEC of radon, to which the individual is exposed over a given time period T.

$$E_p(T) = \int_0^T c_p(t)\, dt$$

= potential alpha energy exposure

$$E_{act}(T) = \int_0^T EEC(t)\, dt$$

= equilibrium-equivalent activity exposure.

The SI unit of E_p is J.h.m^{-3} .

The radon daughter exposure of miners is often expressed in the unit Working Level Month (WLM). 1 WLM corresponds to an exposure at 1 WL for 160 h, a period appropriate to conditions of occupational exposure.

1 WLM = 160 WL.h. = 3.5 x 10^{-3} J.h. m^{-3}

The corresponding unit of activity exposure E_{act} is Bq. h. m^{-3}.

1 WLM = 6.3 x 10^5 Bq.h.m^{-3} for radon daughters

1 WLM = 4.63 x 10^4 Bq.h.m^{-3} for thoron daughters.

42(2).6 POTENTIAL ALPHA ENERGY AND ACTIVITY INTAKE BY INHALATION (I_P , I_{ACT})

The potential alpha energy intake by an individual of radon daughters, I_p, is the inhaled potential alpha energy of the daughter mixture over a definite period of time.

$$I_p = v.E_p,$$

where v is the mean breathing rate. If v is taken as 1.2 m^3/h during working hours (Reference Man value), 1 WLM corresponds to 4.2 x 10^{-3}J.

Activity intake by inhalation (I_{act}) is the inhaled activity during a definite time.

$I_p = (E_p/\lambda_r).I_{act}$, where λ_r is the radioactive decay constant of the nuclide.

Table 42(2).1: Potential alpha energy per atom and per Bq, potential alpha energy concentration in air per $Bq.m^{-3}$ for radon and thoron
(based on ICRP 32)

Radionuclide	Potential alpha energy		Potential alpha energy concentration per $Bq.m^{-3}$ ($MeV.l^{-1}$)
	Per atom (ε_p) (MeV)	Per Bq (ε_p/λ_r) (MeV)	
^{222}Ra (Rn)	19.2	9.15×10^{6}	-
^{218}Po (RaA)	13.7	3,620	3.62
^{214}Pb (RaB)	7.69	17,800	17.8
^{214}Bi (RaC)	7.69	13,100	13.1
^{214}Po (RaC')	7.69	2.0×10^{-3}	2.0×10^{-6}
^{220}Rn (Tn)	20.9	1,660	-
^{216}Po (ThA)	14.6	3.32	3.32×10^{-3}
^{212}Pb (ThB)	7.80	4.31×10^{5}	431
^{212}Bi (ThC)	7.80	4.09×10^{4}	40.9
^{212}Po (ThC')	8.78	3.85×10^{-6}	3.85×10^{-9}

CHAPTER 42: SECTION 3

EPIDEMIOLOGY, DOSIMETRY AND RADIATION-INDUCED LUNG CANCER RISK

42(3).1 INTRODUCTION

ICRP has used two types of basic approach to the problem of evaluation of lung cancer risks from exposure to radon, thoron and their daughter products, viz. the epidemiological and the dosimetric approaches.

42(3).2 EPIDEMIOLOGICAL APPROACH

In countries with a high mean life expectancy of 70-80 years, the integral natural incidence of lung cancer is in the range of 400-800 and 80-200 cases per million per year for males and females respectively (WHO, 1983). An excess lung cancer risk has been observed among several groups of underground miners exposed to radon and its daughters. The largest groups which have been followed up for 30 years include uranium miners in Colorado (USA) (Hornung and Meinhardt, 1987), Saskatchewan (Canada) (Howe *et al.*, 1986), Czechoslovakia (Sevc *et al.*, 1988), Ontario (Canada) (Muller *et al.*, 1985), and France, as well as fluorspar miners in Newfoundland (Morrison *et al.*, 1988). Metal ore (mainly iron ore) miners in Malmberget (Sweden) (Radford and Renard, 1984) and other places have also been studied. Table 42(3).1 gives some details.

Epidemiological studies in relation to lung cancer have also been carried out on large populations consisting of atomic bomb survivors, ankylosing spondylitic patients, and those living in high natural background areas.

From an examination of the epidemiological data, particularly for large groups, ICRP 32 concluded that there is a linear non-threshold proportion between exposure and excess lung cancer up to around 500 WLM. Epidemiological findings as well as animal experiments indicate a decreased slope of the exposure-risk function at high exposure levels, which

is due to deterministic effects resulting from cell killing. For the atomic bomb survivors who were exposed mainly to low LET radiation at a high dose rate, a slightly lower risk than for miners is obtained.

Below 50 WLM the excess was not significant at the 5% level (although a significant excess has been found in more recent Czech studies (Sevc *et al.*, 1988)). Lifetime exposures of 10-20 WLM (which are of interest in exposure to natural radon levels) are a factor of 2-5 below the statistical levels above which an excess lung cancer frequency among the uranium miners has been detected.

The appearance rate of radiation-induced lung cancer as a function of time is similar to the age-dependent distribution of the normal lung cancer rate in a comparable non-exposed population. Consequently, estimation of the attributable lifetime risk on the basis of a relative risk projection model seems to be more appropriate than an absolute risk model. After reviewing the studies made by different agencies, ICRP 60 states that it appears that the excess risk varies with time since exposure; exposures more distant in time have a smaller impact on the age-specific excess risk than more recent exposures. For children below 20 years, the relative risk is probably somewhat higher than for adults.

Regarding the influence of smoking, to-date the epidemiological evidence allows no firm, quantitative conclusion on the combined carcinogenic effects of inhaled radon progeny and smoking. About 70% of the miners were smokers (around 20 cigarettes a day). Some of the larger studies (including the Colorado group) suggest a multiplicative or promoting effect of smoking. This is supported by animal experiments. The inhalation of tobacco smoke influences target regions in the bronchial epithelium which receive the highest doses from inhaled radon daughters. The BEIR IV Committee (NAS, 1988) chose a multiplicative interaction in its risk projection which leads to the conclusion that the lifetime cancer risk for heavy smokers from radon exposure might be 6-10 times higher than for non-smokers. ICRP 50 concluded that the attributable lifetime risk for non-smokers might be a factor of 4 lower than for the population-averaged risk coefficients. The conclusion is that a more than additive (approximately multiplicative) model is indicated for the risk from the combined influence of smoking and radon exposure.

42(3).2.1 Epidemiological Studies on Populations in High Background Radon Level Areas

The studies include populations in Sweden (Edling, 1983; Edling *et al.*, 1984; Pershagen *et al.*, 1984), 60,000 residents in Finland (radon level 4 times the mean for the country) (Castren *et al.*, 1985), Norway (Stranden, 1986), and Guangdong province of China (Hofmann *et al.*, 1985; Weihul *et al.*, 1985). Except for the Norway study which suggested that 10-30% of lung cancers in the Norwegian population might be due to exposure to indoor radon daughters, none of the other studies enable a reliable estimate of the possible contribution of the natural radon daughter exposure to the observed lung cancer frequency in populations.

The epidemiological approach to risk evaluation is fairly straightforward and gives an upper estimate for the risk, as the miners are actually exposed to external gamma radiation, a beta component of dose from the mine surface, inhalation of ore dust, and chemical carcinogens in the mine atmosphere, in addition to radon. The gamma dose rate inside the mine can be taken to be $50C$ μGy/h, where C is the % concentration of U_3O_8; typical dose rates are 5-15 μGy/h.

From the value of excess lung cancer risk probability (per J inhaled alpha energy per year or per exposure of 1 WLM per year), values of the annual limit on intake (ALI), annual limit on exposure (ALE) and derived air concentration (DAC) are worked out, for a mean breathing rate of 1.2 m^3 per hour, using a quality factor of 20 for alpha rays, ICRP 26 effective dose limit of 50 mSv, and the ICRP total risk coefficient of 1.65×10^{-2} per Sv.

42(3).3 DOSIMETRIC APPROACH

The critical cells at risk are those in the basal cell layer of the bronchial epithelium (mostly those of the segmental-subsegmental bronchi) and the pulmonary epithelium (including the alveoli and the non-ciliated terminal bronchioles). There is some uncertainty in our knowledge of the exact depth distribution of the proliferating epithelial cells beneath the mucus sheet in the different bronchial airways. The important types of radiation-induced lung cancer are anaplastic oat cell cancers with a short latency period, and, to a smaller extent, epidermoid cancers, both originating from the bronchial epithelium. Adenocarcinomas have also been observed at a small frequency.

Tissues other than lung receive a negligible dose in the case of radon and daughters. (In the case of thoron daughters, a considerable fraction of the deposited activity from the longer-lived ^{212}Pb will be transferred to other tissues, mainly the RBCs, kidney and bone.)

ICRP 32 discusses 3 models, viz. the Jacobi-Eisfeld (1981), James-Birchall (James *et al.*, 1980, 1981) and the ICRP 30 model (with clearance class D). For each of these, two major concepts have been utilized, viz. the *mean lung dose*(MLD) and the *regional lung dose* (RLD) concepts. In the MLD approach the mean dose to the total lung (considered as a composite organ with the tissue weighting factor of 0.12 given in ICRP 26) is computed. However, for inhaled radon daughters, the dose to the bronchial epithelium is considerably higher than the mean dose to the pulmonary region or the total lung. In the RLD approach, the doses to the bronchial basal cell layer in the tracheo-bronchial (TB) region and pulmonary (P) region are calculated separately. Here, out of the ICRP 26 tissue weighting factor of 0.12 for the lung, each of these two regions is assigned a weighting factor of 0.06.

Both the Jacobi-Eisfeld and James-Birchall models take into account the observed large variation in depth of stem cells throughout the bronchial tree. Both models agree that, normally, under mine conditions, the dose distribution over the bronchial generations from inhaled radon daughters is more uniform than previously assumed, with a broad maximum in the range from the lobar bronchi down to the upper bronchioles. The dose to the basal cell layer of different sizes of bronchial airways varies by a factor of less than 5. The dose to the bronchial target tissue is 4-10 times higher than the mean pulmonary dose. (This is in accord with the histological findings, among radon exposed miners, that the carcinomas originate in the bronchial epithelium.) The dependence of the dose on the parameters characterizing the deposition, translocation and retention of daughters in the lung is relatively small. ICRP 30 overestimates the deposition probability of radon daughters in the TB and P regions.

A proportion of the daughter product nuclei remain unattached; this unattached fraction, f, is an important determinant of the dose to the target cells in the respiratory tract, because of the efficient deposition of the unattached daughters to the airways. The bronchial dose increases nearly linearly with the value of the unattached fraction, f, which is usually less than 0.05. A value of 0.05 is assumed in the computations. The bronchial dose also depends on the breathing pattern and the activity median diameter (AMD) of the carrier aerosols of the attached daughter

atoms. An increase in ventilation rate increases the bronchial dose. For children, the risk factor may be twice that for adults.

As mentioned earlier, the dose to the lung comes mainly from radon daughters. The contribution from radon itself (dissolved in lung tissue and contained in the lung air) is 40-100 times lower than for the daughters and becomes important only when the equilibrium factor F is less than 0.01.

The results for the mean bronchial dose given by different models agree within a factor of 2. The MLD computations give doses 1.5-3 times lower than those calculated by the RLD method. The epidemiological approach gives doses which are 3-6 times larger than those given by the MLD concept. It may be pointed out that in all these calculations the conversion from dose to lifetime cancer risk is based on the absolute risk projection model.

For details of the methods of dosimetric computations reference may be made to ICRP 32 and ICRP 50. The basic steps are as follows: Due to its inert properties, radon is not chemically bound in body tissues and the specific radon activity in any tissue is limited by its saturation solubility which is proportional to the activity concentration of radon in environmental air. The solubility factor (volume saturation ratio of radon concentration in tissue relative to air) in risk-relevant tissues is 0.3-0.5. The specific equilibrium activity from dissolved radon can then be calculated. In the case of lungs, in addition to the radon content of lung tissue, the radon content of the air in the lungs has also to be added. The short-lived daughter atoms produced are assumed to decay in the same tissue as the mother atom. Taking a potential alpha energy of 19.2 MeV per ^{222}Rn atom, the committed effective energy per ^{222}Rn transformation becomes $20 \times 19.2 = 384$ MeV (taking a quality factor of 20 for alpha radiations). The dose equivalent rate to lungs can then be calculated for equilibrium conditions.

Committed effective doses corresponding to unit values of various quantities relating to levels of radon and thoron decay products in air are given in Table 42(3).2A while the corresponding doses for radon and thoron levels in air are given in Table 42(3).2B. Table 42(3).3A gives the derived ALI, ALE and DAC values for radon and thoron daughter products, while Table 42(3).3B gives the corresponding values for radon and thoron in the absence of decay products. Figure 42(3).1 gives the variation of effective dose as a function of the unattached fraction for the RLD and

MLD concepts of the 3 models. Figure 42(3).2 shows the mean doses to the bronchial and pulmonary regions as a function of the breathing rate for an AMD of 0.15 μm and a mean value of 0.03 for the unattached fraction in indoor air; they represent an average of best estimates for adults derived from different dosimetric models. The radon activity concentration $c_{act.,Rn}$ is a quantity which is easier to measure than the potential alpha energy. The conversion factor is given by

$$c_{act.,Rn} = EEC_{Rn}/F,$$

where F is the equilibrium factor. In normally ventilated mines F can be taken as 0.5. Under certain special circumstances (e.g. when air filtration or electrostatic precipitation of decay products is performed), this value of F may be very low, in which case the exposure to radon gas becomes relatively important.

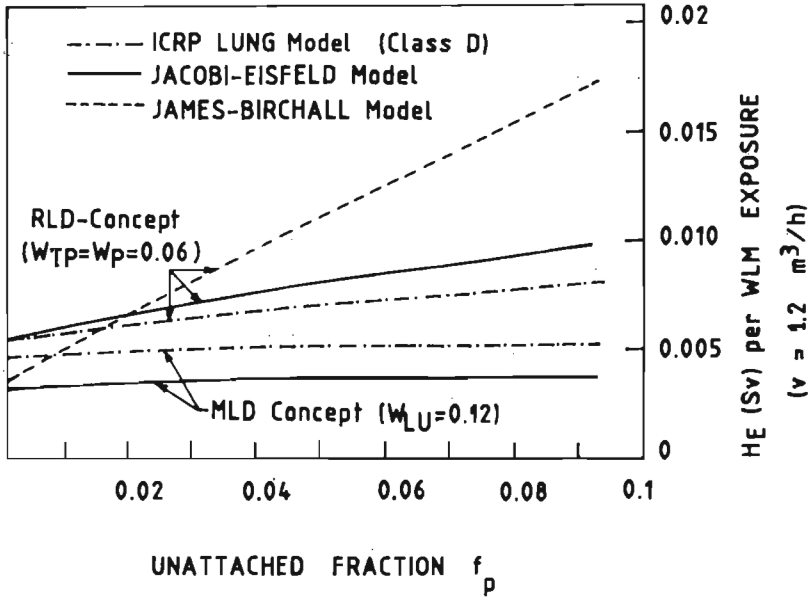

Figure 42(3).1. Effective dose from inhaled ^{222}Rn daughters as a function of the unattached fraction of the potential alpha energy of the daughter mixture: comparison of different dosimetric models and weighting concepts (from ICRP 32).

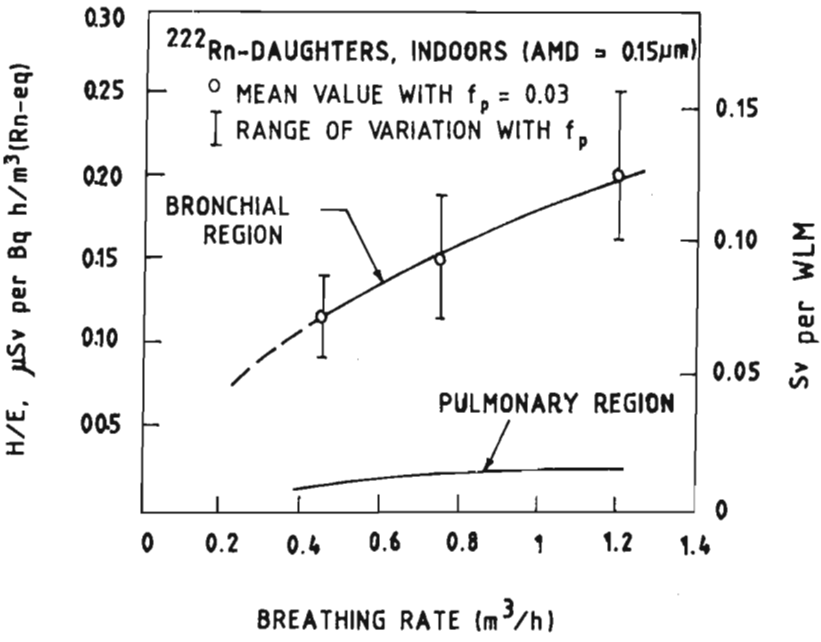

Figure 42(3).2. Mean equivalent dose, *H*, to the bronchial and pulmonary region per unit equilibrium equivalent radon exposure, EEC, to ^{222}Rn daughters in indoor air as a function of breathing rate: best estimates for adults from different models (from ICRP 50).

As discussed earlier in the section on epidemiology of lung cancer (Section 42(3).2), the relative risk projection model is more appropriate than the absolute risk projection model. ICRP 50 has made detailed computations based on both the models. The following risk coefficients are derived.

Risk coefficients derived from the uranium miners data

Absolute risk coefficient: 10 (range 5-15) cases per million person-years per WLM;

Relative excess risk coefficient: 1.0% (range 0.5-1.5%) per WLM.

Risk coefficients derived for a standard population with a mean life expectancy of 70-80 years, averaged over both sexes, including both smokers and non-smokers

Attributable lifetime excess risk:

(a) Absolute risk model:

0.013 for exposure rate of 1 WLM in each year;
2×10^{-8} for exposure rate of 1 Bq.h.m^{-3} in each year;

(b) Relative risk model:

0.5 for exposure rate of 1 WLM per year;
0.8×10^{-6} for exposure rate of 1 Bq.h.m^{-3} per year.

For the absolute lifetime risk coefficient ICRP 60 gives the values: $1\text{-}4 \times 10^{-4}$ per WLM, or $3\text{-}10 \times 10^{-3}$ per mJ inhaled potential alpha energy of radon progeny.

Ore dust

As mentioned earlier, inhalation (and, to a smaller extent, ingestion) of ore dust in the mine atmosphere will give an additional dose. ICRP 47 gives details of the relevant computations. Secular equilibrium of all the radionuclides in the uranium series, an activity median aerodynamic diameter (AMAD) of 1 μm for ore dust, and the most non-transportable form of inhalation class (as per ICRP 30 lung model) are assumed. ALI values of 1.7 kBq (range 1-10 kBq) for the uranium series (based on stochastic limits) and 0.3 kBq (range 0.3-5 kBq) for the thorium series (based on deterministic effects) are derived.

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Table 42(3).1: Epidemiological studies on lung cancer in underground miners
(from ICRP 60)

Group	Number	Mean exposure WLM	Person-years at risk	No. of lung cancer deaths	
				Observed	Expected
Colorado USA (1951-82)	3,347	882	71,642	256	59.1
Ontario Canada (1955-81)	11,076	37	217,810	87	57.9
Saskatchewan Canada (1950-80)	6,847	22	114,170	65	28.7
Czechoslovakia (1948-80)	4,043	226	83,836	484	98
Malmberget Sweden (1951-76)	1,292	98	27,937	51	14.9

Table 42(3).2A: Committed effective doses corresponding to unit values of various quantitites relating to levels of radon and thoron decay products in air

(from ICRP 47)

Quantity	Unit	Dosimetric Conversion Coefficient	
		^{222}Rn decay products	^{220}Rn decay products
Equil.-equiv. Rn-activity	Sv/Bq	1.4×10^{-8}	6.2×10^{-8}
Time-integrated equil.-equiv. Rn-conc.	$Sv/(Bq.h.m^{-3})$ or Sv/WLM	1.7×10^{-8} 1.0×10^{-2}	7.6×10^{-8} 3.5×10^{-3}
Equil.-equiv. Rn-conc.	$(Sv.y^{-1})/(Bq.m^{-3})$	3.5×10^{-5}	1.5×10^{-4}

Table 42(3).2B: Committed effective doses corresponding to unit values of various quantities relating to radon and thoron levels in air

(from ICRP 47)

Quantity	Unit	Dosimetric Conversion Coefficient	
		^{222}Rn	^{220}Rn
Activity	Sv/Bq	1.5×10^{-10}	0.9×10^{-10}
Time-integrated activity conc.	$Sv/(Bq.h.m^{-3})$	1.8×10^{-10}	1.1×10^{-10}
Activity conc.	$(Sv.y^{-1})/(Bq.m^{-3})$	3.3×10^{-7}	2.0×10^{-7}

Table 42(3).3A: Recommended annual limits on intake ALI and exposure ALE and derived air concentrations DAC for radon and thoron decay products

(from ICRP 47)

Type of limit	Quantity & unit	Decay products	
		^{222}Rn	^{220}Rn
ALI_p	Potential alpha energy, J	0.02	0.06
	Equil.-equiv. Rn-activity, Bq	3.6×10^6	8.0×10^5
ALE_p	Time integ. of potential alpha energy conc.		
	$J.h.m^{-3}$	0.017	0.050
	or		
	WLM	4.8	14
	Time integ. of equil.-equiv. radon conc.		
	$Bq.h.m^{-3}$	3.0×10^6	6.6×10^5
DAC*	Potential alpha energy conc.		
	$J.m^{-3}$	8.3×10^{-6}	2.5×10^{-5}
	or		
	WL	0.4	1.2
	Equil.-equiv. radon conc.		
	$Bq.m^{-3}$	1500	330

* Based on a mean breathing rate of 1.2 $m^3.h^{-1}$ during a working period of 2000 h per year.

Table 42(3).3B: Recommended ALE, ALI and DAC values for radon and thoron in the absence of decay products
(from ICRP 47)

Type of limit	Unit	^{222}Rn	$^{220}Rn + {}^{216}Po$
ALE	$Bq.h.m^{-3}$	3×10^8	5×10^8
ALI	Bq	3.6×10^8	6.0×10^8
DAC	$Bq.m^{-3}$	1.5×10^5	2.5×10^5

APPENDIX 42(3).1

OPERATIONAL RADIATION PROTECTION IN URANIUM MINES

The following paragraphs give a very brief summary of the detailed discussions given in ICRP publication 47 (1985).

Exposure control is primarily by mechanical ventilation combined with other techniques. The choice of mining method is important. For high uranium content (> 1%) mines, remotely operated tools may have to be considered.

Application of sealant to rock surfaces to reduce radon emanation is not economically effective. Abondoned workings may build up high levels of radon. Water seeping from rocks (which releases dissolved radon into the mine air) should be conducted away by pipes. Ventilation systems with conventional air cleaning provisions should be operated continuously with maximal air velocities compatible with safety and comfort; recirculation should be avoided. Mined ore should not be broken into sizes smaller than necessary for efficient loading and hauling (to reduce surface area for radon seepage). Access to areas not currently worked should be controlled and high radon level areas (> 100 kBq/m^3) marked by a warning sign.

Personnel should wear protective clothing and respirators in high level areas. Routine monitoring for external dose rates (which are fairly constant with time), potential alpha energy concentrations of radon daughters, and concentration of ore dust should be carried out. Personal air monitors are the best; these should also be supplemented by area monitoring for air concentration and gamma radiation levels. The system should detect deviations from reference levels as soon as possible.

CHAPTER 42: SECTION 4

INDOOR AND OUTDOOR RADON EXPOSURES

The material presented in this part is based on ICRP 50.

42(4).1 RADON SOURCES IN HOUSES

The sources of radon in houses are: (i) radon exhalation from building materials; (ii) radon influx from underlying soil; and (iii) radon infiltration from outdoor air due to ventilation. Of less importance under normal circumstances is radon released from water supplies and from natural gas used for heating and kitchen appliances.

The radon entry from a given source is expressed in terms of the rate of supply of radon activity to the indoor area per unit indoor air volume (Table 42.(4).1). The table also gives the mean indoor concentration of radon contributed from various sources.

Only a small fraction of the total radon produced inside the building materials diffuses to wall surfaces and is released into indoor air. Exceptionally high entry rates are associated with concrete based on alum shales, such as in Sweden, with values of 500-1000 $Bq/m^3.h$ (Swedjemark and Mjönes, 1984). In houses built with turf, phosphate slags, or phosphogypsum, the rates are several hundred $Bq/m^3.h$.

^{232}Th content of normal building materials is 10-100 Bq/kg, comparable with their ^{226}Ra activity content. Because of its short half-life, only the thoron produced near the surface can diffuse into indoor air. For concrete and brick houses the average rate of entry is 100-500 $Bq/m^3.h$, which is 10-100 times higher than for radon (Folkerts *et al.*, 1984).

The mean radon concentration in soil air is 1000 times higher than the radon concentration in the free atmosphere near ground level. Radon diffuses indoors mainly by convective flow. Indoor concentration varies markedly depending upon the soil permeability and cracks and openings in the understructures of houses. In view of the short half-life of thoron, its entry from soil is probably lower than for radon.

Concentration of radon in the atmospheric air at ground level shows strong local and temporal variations. Average values are 1-10 $Bq.m^{-3}$ (Gesell, 1983; NCRP, 1984; UNSCEAR, 1982). The contribution of the radon from outdoor air to indoor air depends on the openings of windows and doors, meteorological conditions (increasing with wind speed and temperature gradient between indoor and outdoor air). An average ventilation rate of 0.7 h^{-1} may be assumed for houses. In houses, indoor concentration may vary from a few to more than 10,000 Bq/m^3. (The latter implies an annual effective dose of more than 10 times the ICRP dose limit for occupational workers.) On the average, indoor levels are 2-20 times higher than the mean outdoor concentration. Mean thoron entry is probably lower by a factor of 3.

For ground storey houses (particularly wooden houses), the soil is likely to be the most important source. The radon concentration is nearly inversely proportional to the ventilation rate. Summing the contributions from all the sources to the indoor air, a mean entry rate of 6-60 $Bq/m^3 \cdot h$ can be arrived at (Nero *et al.*, 1985).

For thoron the major source is probably the release from building materials. The mean indoor thoron concentration would be 2-20 Bq/m^3 (a factor of 5 lower than for radon).

Due to ventilation and deposition no equilibrium between radon and daughters is reached in indoor air. The equilibrium factor is in the range of 0.3-0.6 and lower than in outdoor air. It increases with decreasing ventilation rate and increasing aerosol concentration in indoor air. Mean values of 0.45 and 0.7 for the equilibrium factors can be taken for indoor and outdoor air respectively. For thoron daughters, the equilibrium factor is 0.02-0.1 in indoor air. The average ^{212}Pb concentration in indoor air will be 0.04-2 Bq. m^{-3} (Porstendörfer, 1984; UNSCEAR, 1982).

42(4).1.1 Results of Surveys in Houses

The measured frequency distribution of indoor radon levels follows a log-normal distribution. The relative frequency distribution of radon daughter levels can be assumed to be similar to that of radon. In houses with high radon levels, the main source of indoor radon is entry from the underlying soil (except for houses built with alum shale where buildings contribute the most). High mean values are found in the Nordic countries (Finland, Norway, Sweden). There is a relatively large diurnal variation in radon daughter concentration in indoor and outdoor air. The annual average

outdoor night concentration seems to be a factor of 2-5 higher than that around noon; in indoor air this variation is less pronounced (Gesell, 1983; UNSCEAR, 1982).

Assuming a mean EEC_{Rn} level of 15 Bq/m^3 and F = 0.45, it may be computed that the percentage of dwellings exceeding specified levels would be as follows:

50 Bq/m^3 - 5%; 100 Bq/m^3 - 1%; 200 Bq/m^3 - 0.1%.

The few measurements of thoron levels indicate a value of 0.1-1 Bq m^{-3} for EEC_{thoron} (mainly attributable to ^{212}Pb). A mean value of 0.5 Bq m^{-3} is assumed in ICRP 50. This is a factor of 2-3 higher than the mean level in outdoor air. EEC_{thoron}/EEC_{radon} in indoor air has a mean value of 0.03; the ratio of potential alpha energy concentration between thoron and radon daughters is about 0.4.

42(4).2 EXPOSURE LEVELS INDOORS AND OUTDOORS, AND RISK COEFFICIENTS

Three components of exposure have been taken into account, viz. indoor exposure at home, indoor exposure elsewhere, and outdoor exposure. Mean indoor residence fraction of 0.85 (0.65 at home, 0.20 in other buildings) and outdoor residence fraction of 0.15 are assumed. Mean daily breathing volumes of 10, 5 and 4 m^3 are assumed for the adult Reference Man during residence at home, indoors elsewhere, and outdoors respectively. Using these values and taking the diurnal variation of radon concentration also into account, ICRP 50 has worked out mean annual exposures to radon and thoron daughters for the following reference values of the mean equilibrium-equivalent concentrations in indoor and outdoor air:

Indoor air

Radon daughters	15 Bq/m^3
Thoron daughters	0.5 Bq/m^3

Outdoor air

Radon daughters	4 Bq/m^3
Thoron daughters	0.2 Bq/m^3

The transfer of risk estimates from the occupational setting of the mines to the indoor environment requires several assumptions, primarily concerning the different distributions by age and sex of the populations, and the differences with respect to physical characteristics of the inhaled air (the unattached fraction, equilibrium factor and particle size distribution). The overall influence of these factors that modify lung cancer risk is apparently smaller than the uncertainties of the dosimetry and the limitations of the primary epidemiological data.

The attributable relative risk to the reference population comes out to 11.6% of the total lung cancer risk (10.4% from radon daughters and 1.2% from thoron daughters). The values for the excess frequency of lung cancer cases per million persons per year are: Males – 79; females – 16; average for both sexes – 47; average for both sexes for non-smokers –11. The contribution from radon daughters to these totals is about 90%.

The mean loss of lifetime per lung tumour induced by inhalation of radon daughters is estimated to be around 15 years. At the assumed reference values of the indoor and outdoor radon levels, the mean loss of life expectancy per capita is computed to be 16 days; compared to the mean life expectancy of around 73 years, this corresponds to a mean relative loss of life expectancy of 0.06%.

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Table 42(4).1: Volume specific entry rate and concentration of radon in indoor air from different sources
(based on ICRP 50)

Source	Mean specific entry rate ($Bq/m^3.h$)	Mean indoor concentration* (Bq/m^3)
Building materials		
Brick or concrete houses	2-20	3-30
Wooden houses	< 1	⩽ 1
Soil	1-40	2-60
Outdoor air	2- 5	3- 7
Other sources (water, natural gas)	⩽ 0.1	⩽ 0.1
All sources	6-60	10-100

* Assumed mean ventilation rate of 0.7 h^{-1} (normal range 0.3 to 1.5 h^{-1})

CHAPTER 42: SECTION 5

LIMITING EXPOSURE OF THE PUBLIC TO RADON AND DAUGHTERS IN DWELLINGS

42.(5).1 INTRODUCTION

As discussed in ICRP 39 (see Chapter 34), there is a clear difference between the *existing* exposure situations, where any action would have to be remedial, and *future* situations, which can be subject to limitation and control at the stage of decision and planning.

42.(5).2 EXISTING EXPOSURE SITUATIONS

ICRP feels that it would not be helpful to suggest a generally applicable value of an action level for existing situations. For owner-occupied buildings, general guidance may be adequate, leaving the final decision to be made by the occupier. But in the case of rented buildings, it may be desirable to establish firm national action levels (which define a significant, but not unmanageable, number of houses in need of remedial work). ICRP 39 suggests that if the remedial action considered is fairly simple, an action level for equilibrium equivalent radon concentration in the region of 200 Bq/m^3 (annual effective dose 20 mSv) might be considered. For severe and disrupting action, a value several times larger might be more appropriate.

For practical reasons, ICRP recommends that *investigation levels* be established by national authorities to separate exposures that require investigation from those that do not. In the present case, these can be set on the basis of activity in the type of building material and in the ground, ventilation, and type of construction. Water in deep wells is usually markedly more active than surface water.

42.(5).3 FUTURE EXPOSURE SITUATIONS

The problem of new buildings has some similarity to that of existing buildings because the concentration of radon cannot be determined with

confidence until the building has been occupied for a year or so, in which case it becomes an existing building. Future exposures should be justified in the sense that the source of such exposures does more good than harm and the protection arrangements should minimize the detriment. However, the dose limits should *not* be applied to these situations. Instead, the exposure of the most highly exposed individuals should be limited by the application of an *upper bound* (established by the competent authority) in the optimization assessment. ICRP 60 mentions that it has initiated a further review of current experience with a view to issuing revised recommendations in due course. Meanwhile the guidance in ICRP 39 should still be used. ICRP 39 believes that a reasonable upper bound for the equilibrium equivalent concentration is of the order of 100 Bq/m^3, and that, in many countries, a value of this magnitude would prevent radon from becoming a dominant source of risk in dwellings. This upper bound may influence building standards for construction, manufacture, ventilation, etc.

Part VII

Optimization and Decision-Making in Radiological Protection

CHAPTER 43

OPTIMIZATION AND DECISION MAKING IN RADIOLOGICAL PROTECTION

43.1 INTRODUCTION

The stipulation of ICRP that 'all doses be kept as low as reasonably achievable, economic and social factors being taken into account' makes it necessary to consider what scale of resources should be provided for reducing the radiation risk to individuals and society, and how the resources can best be allocated. Optimization techniques help to decide as to when sufficient effort has been applied to the reduction of radiation detriment associated with a practice. If the next step of reducing the detriment can be achieved only with a deployment of resources that are seriously out of line with the consequent reduction, it is not in society's interest to take that step, provided that all individuals are adequately protected. The protection can then be said to have been optimized. The methods for arriving at such decisions range from simple common sense to complex techniques like cost-benefit analysis and multi-attribute utility analysis.

As mentioned in Appendix 8.2 on the evolution of ICRP recommendations, ICRP 9 stipulated that 'all doses be kept as low as readily achievable, economic and social considerations being taken into account'. ICRP 22 changed the adverb 'readily' to 'reasonably', and also introduced the term 'collective dose'. It also first introduced the cost-benefit analysis notation which has since become familiar.

Judgements involved in optimizing protection are not purely quantitative -- they involve preferences between detriments of different kinds and between financial costs and health effects – but they can be supported by quantitative techniques. Optimization procedures are 'decision-aiding', clarifying the factors involved, quantifying them where this is reasonable, and systematizing trade-offs between the various factors. Resources devoted to radiation protection have to be compared with those devoted to other needs of society. Optimization should also apply to plans designed to prevent the consequences of accidents as well as to choices between different options for post-accident management.

ICRP publication 37, 'Cost-benefit analysis in the optimization of radiation protection' (1983) focussed attention on the cost-benefit analysis technique for optimization. Less quantifiable social factors will also have to be accommodated in a satisfactory technique of optimization. ICRP publication 55, 'Optimization and decision-making in radiological protection' (1989) deals with the subject on a broader basis.

43.2 OPTIMIZATION OF PROTECTION

Once it moves beyond the intuitive level, a structured approach to optimization should provide the structure and framework within which to apply judgement, systematize the judgement, and help to achieve coherent and consistent objective decisions. Figure 43.1 (which is based on three figures given in ICRP 55) illustrates the steps in the structured approach, applicable broadly to both design and operational optimization.

A brief explanation of the terms used in the approach may be worthwhile. *Protection option* is a specified design or set of operational procedures aimed at reducing doses or the probability of doses being received. Those options which do not meet the design requirement or are impracticable will be eliminated. The *performance of the option* is based on the results of applying a specific design option or set of operational procedures, expressed in terms such as the collective dose, process efficiency, etc. The *cost of the option* includes the direct financial and resource costs of the operation as well as other costs, e.g. training. A *factor* is an identifiable measure of either the cost or the performance of an option. The factors can be broadly divided into those concerned with and those not concerned with radiological protection. Radiological protection factors include financial cost, collective dose, maximum dose, number of persons receiving high doses, training, discomfort from protective clothing, etc. Further parameters relevant to the radiological protection factor are the historical trends of individual and collective doses, short-term and long-term risks (especially for waste management), number of incidents and accidents that have occurred, etc. Some of these factors like cost of production and collective dose are quantifiable. Other factors not connected with radiological protection include costs incurred solely to increase efficiency, for aesthetic or public relations reasons, etc. Even though these factors may profoundly influence the eventual decision, they are not part of the optimization of radiation protection. An *attribute* is equivalent to a factor, used in the technical phrase *multi-attribute utility analysis*. *Criterion* is a qualitative or quantitative measure of what is

acceptable or desirable for one or more of the factors (e.g. individual dose limits, or a specified value for the unit collective dose).

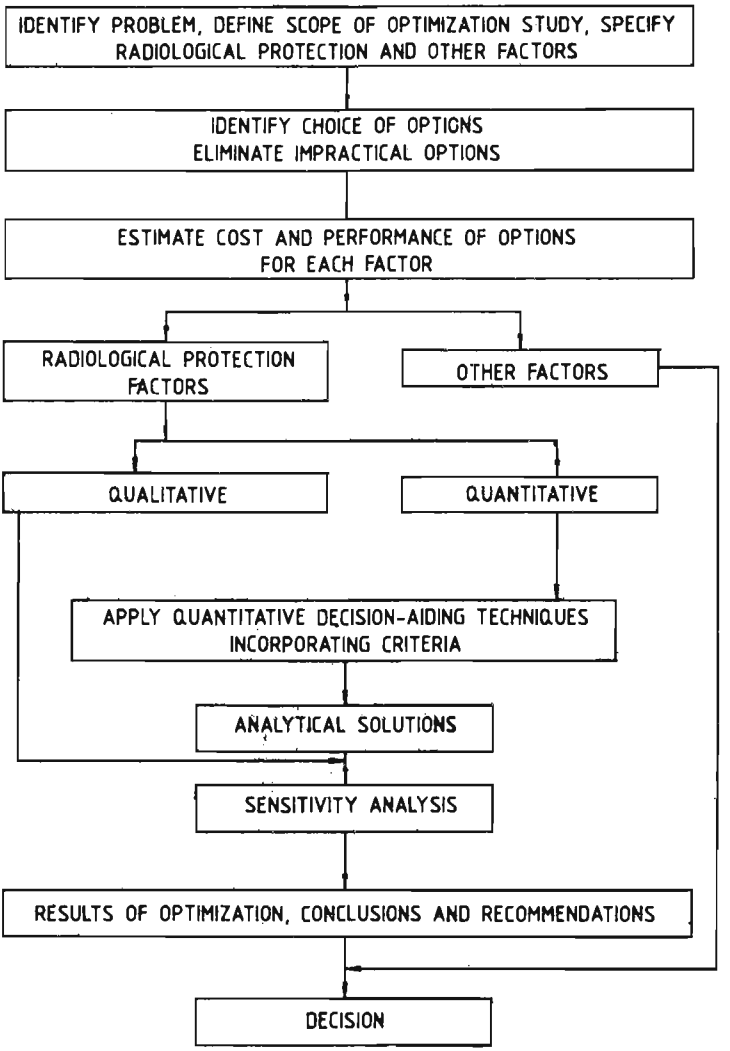

Figure 43.1. Structured approach to optimization of protection for decision-making (based on ICRP 55).

Figure 43.1 shows the major steps in the optimization procedure to be: the initial recognition that there is a need for an optimization study and

the definition of the scope of the study; the identification and quantification of the factors to be considered during optimization, and the separation of the factors into those relevant to radiological protection and the others; the analysis (qualitative or quantitative) of the performance of the options with respect to each of the factors against the criteria, preferably explicitly stated; the generation of a recommended optimum for the analysis; arriving at the 'analytical solution'; the conclusions as to what actions need to be taken, and the final decision which may take into account factors that are outside radiological protection. Modelling is an important part of estimating performance (e.g. environmental modelling to predict doses to members of the public).

In design, the need for a study is generally obvious, and the focus is on possible design options that have different implications for protection, effort including cost, operational aspects, reliability of the options, and other factors. The cost of protection and collective dose (which are quantifiable) are always included in the analytical procedure. The collective dose is the product of the average dose rate, exposure time and number of people exposed; optimization involves changing one or more of them.

In operations, decisions often have to be made on an hour-to-hour basis and so optimization should be a continuous process. The stimulus may be a management initiative or a regulatory requirement, triggered by, for example, high doses in the workforce. The resulting study will often be based on an analysis of the current situation. The choice of options will involve aspects such as changes in working procedures, maintenance requirements, training and education, management structures, updating of instrumentation systems, and may include design changes. In addition to the accumulated experience of those carrying out the study, experience of a number of studies will help to codify the experience into qualitative guidelines.

The technique of cost-benefit analysis is a principal decision aid in such circumstances. The simplest form in which it has been applied is to assign a value to unit collective dose as an expression of the radiation-induced health detriment. But it has its own limitations. For example, has a given collective dose to the workers the same significance as the same collective dose to the public? Can a given collective dose made up of a very large number of small doses be equated with the same dose made up of a small number of high doses? The more general decision-aiding techniques introduced in ICRP 55 would be helpful in making explicit allowance for different kinds of weighting between different components

of the radiation detriment and enable the innate preference of decision makers to be expressed overtly.

Certain other considerations are relevant. The justification of a practice is that it should do more good than harm. If there is a range of choices for a practice, the justification principle can eliminate the practices not producing a net benefit. But it does not require the benefit to be the greatest obtainable. Optimization is concerned with the improvement in the protection arrangements within each alternative justified practice.

Assessment of doses for comparison with dose limits are generally based on pessimistic assumptions, while optimization should be based on realistic assumptions. Where a source upper bound has been stipulated, it will act as a constraint in the optimization. Authorized limits should be selected after an optimization study, and constrained by the source upper bound or the dose limit; the authorized limit can be somewhat higher than the result of optimization study to provide some operational flexibility. Optimization studies are also relevant in deciding on intervention and action levels.

It is desirable that the results of generic or specific optimization studies be expressed in terms of design targets or standards to make them more useful to design engineers. Optimization studies on operations, maintenance and inspection should result in revised standardized procedures.

One of the reasons for exempting a source from regulatory control is that the available control procedures cannot reasonably achieve a reduction in dose comparable with the resources involved in the control; this is equivalent to an optimization approach.

In optimization studies, we normally start with a 'base case', e.g. the cheapest option in a design study, or the current set of practices in the case of existing operations. Knowledge of previous optimization studies of a generic nature will give useful general guidance.

In operations, the qualitative element is likely to be large. The scope of the study may involve review of the entire system or of one specific aspect. Although plant design imposes some constraints, there will still be some flexibility in operating procedures which can be reviewed periodically. Experience has shown the utility of reviews in practically every field of application of radiation. When protection options are narrow, such as in the case of a specific component of the plant, the determination of the collective doses and protection costs for the options may be sufficient

for comparison and a simple cost-benefit analysis will be appropriate. Where large uncertainties exist, a technique able to handle the uncertainty, such as a multi-criteria outranking analysis, may have to be resorted to. The outcome of the reviews should be passed on to the designers.

There are several uncertainties associated with the analysis including: (i) imperfect knowledge of the performance of options and parameters used in the assessment of dose estimates; (ii) adequacy of the model; (iii) uncertainties due to future developments; (iv) intrinsic uncertainties resulting from the statistical treatment of variables; and (v) value to be assigned to unit collective dose.

Sensitivity analysis involving the above uncertainties gives important insight into the stability of the results with regard to variations in data and assumptions; in particular, it identifies aspects which have the greatest influence on the results. Sensitivity analysis is more difficult in operational optimization.

The important assumptions and judgements on which the conclusions of the optimization study are based should be clearly stated so that they can be scrutinized independently. The degree of credibility of inputs (e.g. dose estimates, model reliability) is an important aspect.

Recommendations from optimization procedures are only one input to a wider consideration leading to a decision. If the study is simple and unlikely to be affected by factors outside the scope of radiological protection, the recommendations will be accepted. But if other factors (e.g. aesthetic reasons, likelihood of public acceptance) are involved to a significant extent, management may take a decision different from the recommendations of the optimization study. In such cases the reasons for the decisions should be recorded.

43.3 QUANTITATIVE DECISION-AIDING TECHNIQUES

This is a specialized subject and hence only a very brief indication of the contents of ICRP 37 and 55 will be given here.

While ICRP 37 confined itself to the cost-benefit technique, ICRP 55 discusses 4 techniques, viz. cost-effectiveness analysis, cost-benefit analysis including differential cost-benefit analysis, multi-attribute utility analysis, and multi-criteria outranking analysis.

Cost-effectiveness is not appropriate on its own for optimization but can be used to eliminate non-viable options if they are similar in character to the viable options.

It is the specification of the radiological protection factors and of the criteria to be used in the analysis that determines the outcome; all techniques would lead to the same analytical solution and optimum choice. If only two factors are relevant (e.g. cost and collective dose), a simple technique like cost-benefit analysis will give a solution that directly indicates the option. Otherwise, a more complex technique has to be used.

43.3.1 Cost-effectiveness Analysis

Each option has a level of protection cost and a corresponding collective dose. The simplest way to express the relation between options is to plot the two variables against each other (Fig. 43.2). The options can be discrete (represented by points joined by straight lines) or continuous (given by a smooth curve). We see that some of the options have a neighboring collective dose at a lower cost; these options (marked by a cross in the circle in the figure) can be eliminated. We can then impose a constraint either on the collective dose (for a fixed protection cost) or on protection cost (for a limited collective dose), and can select the option which minimized one or the other. Neither of these cost-effectiveness procedures corresponds to an optimization since they do not involve a trade-off between protection cost and the collective dose.

43.3.2 Cost-benefit Analysis

This is an old, straightforward technique, originating from the economic theory of welfare, which has probably been used the most widely in optimization of radiation protection. (Out of the 13 case studies quoted in the annotated bibliography in ICRP 55, 11 have used this technique.) The factors influencing the decision are commonly expressed in monetary terms. The cost (including the social cost) of achieving a selected level of protection X, and the cost of the detriment Y (generally represented by the collective dose S) are the options for optimization.

In the notation of ICRP 22, the net benefit B of a practice is given by

$$B = V - (P + X + Y)$$

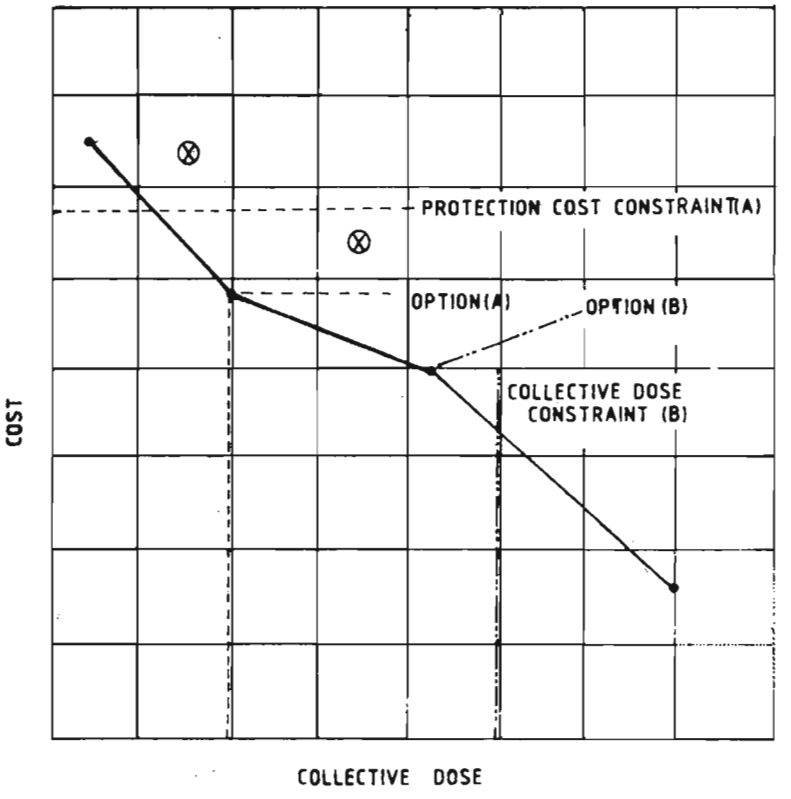

Figure 43.2. Cost-effectiveness analysis (based on ICRP 55).

where V is the gross benefit and P the production cost. 'Justification' requires that B should be greater than zero. Optimization of the net benefit involves 'differential cost-benefit analysis'. V and P can be considered constant for a given practice, and hence the total cost, $X + Y$, should be minimized. Taking the collective dose as the independent variable, it is seen that optimization is achieved at a value of S equal to S_0, where

$$(dX/dS)_{S_0} = -(dy/dS)_{S_0}$$

At this optimum value of S, the increase in benefit from a unit reduction in dose is balanced by the increased cost in its achievement. When all doses are sufficiently low (well below the deterministic threshold doses) the detriment can be taken to be proportional to S. This situation is illustrated in Fig. 43.3.

The value assigned to the unit collective dose, α, is highly variable, ranging from \$ 1000 to \$ 100,000 per man-Sv.

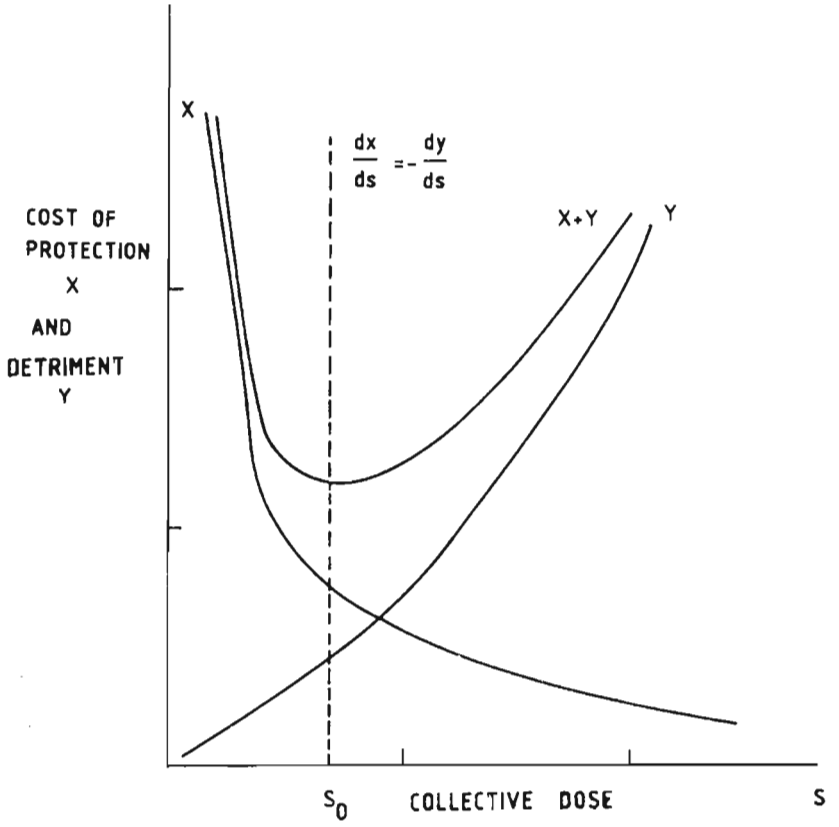

Figure 43.3. Differential cost-benefit analysis (based on ICRP 22).

The cost-benefit analysis can be extended to take into additional account other factors like the individual dose distributions (ICRP 37, 42). (For example, we may be concerned with the group which receives doses which are a substantial fraction of the dose limit.) In this case we introduce an additional term β.

$$Y = \alpha S + \sum_j \beta_j S_j$$

where S_j is the collective dose originating from a per caput dose H_j delivered to the N_j individuals of the jth group, and β is the additional value assigned

to unit collective dose in the jth group. As the individual dose increases, β can be assigned higher monetary values. Optimization can be done with this modified value for the detriment cost *Y*.

43.3.3 Multi-Attribute Utility Analysis

This broadly applicable approach has evolved from several disciplines including physiology, engineering, and management. It can overcome the problem of factors difficult to quantify in monetary terms (Merkhofer and Keeney, 1987). A scoring scheme (or a multi-attribute utility function) is used for the relevant factors. An option with a higher score is preferred.

The protection factors to be included in the optimization study are identified, and the consequences of each protection option quantified in terms of these factors. A 'utility function' u_j is developed giving the relative desirability of the possible outcomes for the factor j, ranging from 1 for the best outcome or lowest adverse consequence for each factor (e.g. lowest cost, lowest minimum *S*) to zero for the worst consequence. (The utility function need not be linear.) From this single utility function u_j, expressing the various utilities of the n factors associated with each protection option i, a multi-attribute function U_i or 'figure of merit' or 'total utility' is obtained for each option, as

$$U_i = \sum_{j=1}^{n} k_j \, u_j$$

where k_j is a scaling constant expressing the weight assigned to each factor j. These are normalized so that $\Sigma k_j = 1$. The higher the figure of merit, the better the overall ranking of the option. The optimum option is one which maximizes U.

Cost-benefit analysis is a particular form of additive multi-attribute analysis, where all the single utility functions are linear and the k_j are dimensionless constants corresponding to the monetary values assigned to each unit of consequence.

43.3.4 Multi-Criteria Outranking Analysis

In the previous analysis the performance of each option was expressed in terms of a single overall figure of merit (whether it is a total cost-benefit analysis or a utility function in multi-attribute utility analysis). The outranking technique initially compares each option to every other

option in order to evaluate whether option i outranks (is preferred to) option m. This comparison by pairs is based on two indicators, viz.

(a) An 'advantage index' $Ad_{i,m}$ that expresses the amount by which option i is preferred to option m; its value ranges from 0 to 1.

(b) An 'exclusion criterion' $Ec_{i,m}$ that expresses the degree to which the disadvantages of option i as compared with option m are significant for the factors where i is not preferred or equal to m. This index is equal to 1 when the drawbacks associated with choice i are substantial and equal to 0 otherwise.

If $Ad_{i,m}$ is high and $Ec_{i,m}$ low, option i outranks option m. The outranking relationships are worked out for each pair of options. We would then be left with a limited number of options which are not outranked by others. By imposition of additional exclusion criteria the final analytical solution can be pinpointed.

43.4 QUANTIFICATION

The simplest evaluation of the cost of an installation, design, or the protection efforts, the 'crude cost estimate', is to add the initial cost to the sum of the annual operating costs over the expected years of operation. (This cannot distinguish between options with high capital costs and low operating costs or *vice versa*.) More refined methods are 'present worth evaluation' and 'annualized cost estimates' which, in addition, make allowance for 'discounting' of costs incurred in the future with costs incurred at the start. Indirect costs (like training programmes and management supervision) are difficult to quantify.

We have already discussed the quantification of the cost of detriment in terms of the collective dose, and how individual dose distributions can also be included as an additional component of the detriment. Another factor is the time distribution of doses. The social importance of doses at different periods of time may have to be given different weights. It often happens (e.g. in waste disposal) that the dose commitment beyond a certain cutoff time (the 'tail') may not be very different for different options.

In potential exposure situations the probability of receiving the dose as well as the probability of harm from the dose have to be assessed. ICRP 46 (1985) has developed the methodology of probabilistic risk assessment in relation to solid waste disposal, as discussed in Chapter 36.

43.5 APPLICATION OF THE PROCEDURE

For major installations, comprehensive studies on optimization will usually be conducted by the licensee and will tend to be specific to the installation. In other instances, e.g. industrial radiography, practices are widespread and fairly uniform in working procedures and equipment. In these cases the competent authority is the most effective one to organize generic studies leading to standardization of procedures.

It is during the design stage that the main features which determine the level of protection are fixed. Everyone in the design organization should be involved in the optimization of design. Design engineers should have appropriate qualifications and training in the basic concepts of radiation protection and be helped by regulatory guides.

ICRP has not previously provided specific recommendations on quantitative optimization techniques for operational radiation protection which is less amenable than design optimization. But the subject has equal importance here also. The radiological protection officer would be the best suited to carry out such studies and inform the management, which will then more easily be persuaded to allocate the necessary resources for radiation protection.

Although the responsibility for protection remains with the management, the competent authority can assist by establishing relevant basic criteria such as the value of unit collective dose for optimization, guiding designers and managers on the basic methodologies as well as how to apply them on a case-by-case basis, organizing generic assessments on optimization, enforcing suitable regulations and auditing performance of activities.

ICRP 37 has given a number of appendices illustrating the application of cost-benefit analysis for optimization in various fields, including design of shielding, ventilation for a plant for production of ^{131}I-labelled pharmaceuticals, radon contamination problems, backfitting of a ventilation system in a mine, design of a simple air-cleaning system, control of gaseous releases from a boiling water reactor, nuclear medicine operations, and domestic use of natural gas which may contain radioactive contaminants. ICRP 55 has chosen for detailed illustration an example of design of a ventilation system in a uranium mine, based on work by Lombard *et al.* (1984). The radiological protection factors chosen are occupational collective dose, protection cost, distribution of individual occupational doses, and discomfort associated with the ventilation factor.

Protection of the public has not been considered. The application of each one of the 4 quantitative techniques for optimization discussed earlier has been illustrated in ICRP 55.

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CHAPTER 44

QUANTITATIVE BASES FOR DEVELOPING A UNIFIED INDEX OF HARM

44.1 INTRODUCTION

ICRP 26 (1977) emphasized the importance of ensuring that the likely risks from occupational exposure to radiation should at least not exceed those observed in other occupations recognized as having high standards of safety. For comparing the relative safety of different industries, the aim should be to aggregate the total detriment from different types of harmful effect (e.g. accidental deaths, injuries and diseases) according to an acceptable method.

ICRP publication 27, 'Problems involved in developing an index of harm'(1977), first addressed itself to this task in a preliminary way. This publication discussed the difficulties in making an appropriate comparison of radiation and other effects, and suggested a quantitative 'index of harm', which could conceivably take into account the aggregate of the lengths of time lost from normal health and activity as a result of accidental deaths, occupational injuries and illnesses, appropriately weighted according to the perception of the hardship caused by equal periods of time lost in different ways. Subsequently, a more broad-based assessment of harm has been done in ICRP publication 45, 'Quantitative bases for developing an index of harm' (1985), taking into account new data on occupational accident risks and including a consideration of radiation-induced non-fatal cancers, non-stochastic effects and hereditary detriment. The index of harm is expressed as the years of healthy life lost per 1000 worker-years at risk.

44.2 OCCUPATIONAL INJURIES

These are normally recorded in three groups, viz. (a) fatal; (b) temporary (involving limited periods off work); and (c) causing permanent incapacity of greater or lesser degree.

44.2.1 Fatal Accidents

Occupational accident death rates differ by more than two orders of magnitude. Based on data from the International Labour Organization (ILO, 1980), the weighted mean death rates (per million worker-years, or 2×10^9 working hours) come out to be: Manufacturing: 75; railway workers: 230; construction: 230; and mining and quarrying : 450. The ranking is broadly the same in different countries. Rates are highest (5000 or over) for navy frogmen and steeplejacks (Metropolitan Life Insurance Company, 1974). During wars, the risk to armed forces personnel is one order of magnitude higher than the value for frogmen (Pochin, 1975). Rates in mining, quarrying, and forestry are several times higher than the mean rate for all industries. Risks in construction are above the mean. Manufacturing processes as a whole have rates in the region of half the national average (but show a variation by an order of magnitude among themselves). In the same industry, the rates vary by a factor of 5 according to the job. The rates are regularly lower by an order of magnitude for women. Occupations mainly involving office work have low rates of fatal injury. Table 44.1 gives, in some detail, UK data for the range of fatal accident rates in different manufacturing industries (UK Health and Safety Executive, 1975 and subsequent reports). For India, the median annual fatality rate in manufacturing industries was 120 per million-years around 1980.

It is interesting to note that, for a given occupation, there is a relation between the size of the workforce and the fatal accident rate. It may be substantially higher in undertakings with a small workforce. For a given industry, the rate reaches a maximum for a given employment size and falls on either side of it (US Department of Labour, 1983). Further, the death rate during travel to and from work, although variable, can be quite a substantial fraction (30 to 60%) of the total death rate at work in some countries. (This is attributed to around 90% of the workers travelling to work by automobile.) The fatal accident rates have been normally decreasing by a few per cent of their value annually, halving every 20-40 years. An average loss of life expectancy of 35 years per accidental death has been worked out by ICRP 45 for a mixed population; this is nearly equal to the mean age of the workforce.

44.2.2 Accidents at Work Causing Temporary Disability

The frequency of non-fatal accidents is usually in the range of 100-1000 times that of fatal accidents. This ratio varies systematically with

the risk of the occupation, being less in those occupations in which the accident fatality is high. This implies that accidents are more frequently fatal in occupations in which the accident rate is high. The total time loss from such accidents is comparable with the total loss of life expectancy due to fatal accidents in the same period. (The mean and median values of the ratio are 1.2 and 0.9 respectively.) The mean period of disability differs only moderately between different industries or different countries. Relative to a mean loss of life expectancy of 35 years from an accidental death, in occupations with fatality rate greater than 200 per million-year, the total loss of time from temporary incapacities is less than the loss of life expectancy from a fatal one. For the safer industries, however, this ratio is 3-4 times.

44.2.3 Accidents Causing Permanent Disability

Since the degree of impairment may vary widely, the absolute number of disabilities in a year gives no indication of the loss of health or working capacity involved. In several countries, the amount of partial permanent disability is assessed as a fraction of that from complete permanent disability.

The ratio of accidents causing permanent disability (partial or total) to temporary disability varies from 2 to 15%. The time loss detriment varies from 0.1 to 6.8 (relative to loss of life expectancy from fatal accidents taken as 1.0) for permanent disability and 0.2 to 2.1 for temporary disability. The mean duration of detriment from a permanent disability will be nearly equal to the mean loss of life expectancy from a fatal accident. The arithmetic mean of the ratio between the time loss due to permanent disabilities and that from fatal accidents, each being determined by the life expectancy at the age of the accident, and the former taking account also of the weighting applied to each disability (relative to that for complete disability), came out to 6.9 in 56 industrial groups; there was a large dispersion among different industries.

44.2.4 Time Loss from Injuries of Different Severity

On the average, the total length of healthy life impaired during temporary incapacities is about equal to that lost owing to fatal accidents. The length of life impaired by permanent incapacity is uncertain. When expressed in equivalent years of *total* incapacity for work, it may be much greater than that lost owing to accidental deaths. Each year of life

impaired by temporary incapacity may be considered to involve less detriment than a year of lost life expectancy due to death. In that case, the total detriment would be dominated by that from fatal accidents. The amount of partial disability (as a percentage of total permanent disability) would be 10-25%.

ICRP 45 concludes that the typical detriment from all types of occupational injury is equivalent to 50-100 years loss of life expectancy per accidental death as compared to the 35 year average loss of life expectancy due to the fatal accident itself. If so, in an industry with 100 accidental deaths annually per million workers, the contribution to an index of harm of 3.5 due to fatalities would typically be raised to 5-10 to include harm due to non-fatal and fatal injuries.

44.2.5 Injury Rates in Occupations Involving Radiation Exposure

Fatal injury rates in uranium mines are 400-1700 per million per year. (The figure for non-coal mines is 500-1400 per million per year.) In most other phases of the nuclear fuel cycle, the fatality rate is low, around 20-30 per million per year. The non-fatal injury rate is around 20 per 1000 per year.

44.3 INDUSTRIAL DISEASES

Figures quoted by the International Labour Organization (ILO, 1970) for the annual mortality rate from pneumoconiosis in mines vary from 140 to 5400 per million at risk for different mines and countries, being high for coal workers (especially underground coal miners) and metal miners, while the figures for silicosis are 200-5800. The large differences are partly due to the type of material being mined, and the procedures for dust control, but mostly due to differences in recording procedures. Data on incidence are much more uncertain.

The continuing detection of raised incidence of malignant and other diseases in different occupations (Doll and Peto, 1981) indicates that the total occupational mortality and morbidity from industrial diseases may be substantially higher than is at present recorded in national statistics. The more recently detected causes of disease include esophagal cancer in vulcanizers, and bladder cancer in 'rodent operators' (rat catchers). There is evidence that several percent of all cancers may be of occupational origin. Increased cancer mortality has been reported in several apparently healthy manufacturing processes as of footwear, furniture or printed material.

Table 44.2 summarizes the cancer mortality rates attributed to occupational exposure to chemical agents.

With the exception of mining and some chemical industries, the total detriment from occupational illnesses is only around 5% of that from industrial accidents. The average incidence of diseases is lower in women. It seems reasonable to assume that, in most occupations, occupational diseases are likely to add only 5-10% to the periods of time lost due to deaths or occupational injuries and to make a correspondingly smaller contribution to the total harm.

44.4 EFFECTS OF EXPOSURE TO RADIATION

ICRP 27 estimated that occupational exposures were received at an approximately constant rate with age, so that the mean age of all exposures was close to the mean age of the workforce. This has been confirmed by detailed studies by ICRP 45. Since the information regarding induction of cancers in ICRP 60 is more comprehensive and recent than in ICRP 45, this aspect is not dealt with here.

44.4.1 Induction of Inherited Abnormalities

UNSCEAR (1982) has estimated the radiation-induced harm that is expressed in liveborn in terms of period of life lost or impaired by different forms of inherited abnormality. The analysis includes mean age of development of impairment, mean age of death from the abnormality, and weighting given to minor abnormalities also. As with somatic effects, it is not easy to give relative weights to equal periods of different kinds of inherited disability. Can a year of lost health or life in an exposed individual be equated to a year lost in his descendants?

UNSCEAR has arrived at the figures given in Table 44.3 for the years of life impaired or lost. It can be seen that, for genetically induced abnormalities, an average of 25 years of a normal 70 year span is unimpaired, an equal period is impaired, and 20 years lost by premature death.

It may be seen that per person-gray of genetically significant dose (GSD) the detriment is 0.05 years impaired and 0.05 years of lost life in first generation offspring or a total of 0.10 years impaired or lost. The corresponding total to all generations is 0.63 years per person-gray of GSD.

44.4.2 Genetically Significant Fraction of Occupational Doses

US data for 1980 (Kumazawa *et al.*, 1984) showed that the genetically significant fraction of the collective dose was 29% (same for males and females) for all occupations put together. The fraction of females in radiation work was on the average 46% of the total, ranging from 72% of the total in the category of medicine to 7% for the nuclear fuel cycle. The mean age at conception was 25 years for females and 28 years for males. Corresponding data for other countries are scanty. The UN Demographic Yearbook for 1981 (UN, 1983) gives an average paternal age at conception of 30.6 $\pm$ 2.9 years and maternal age of 25.9 $\pm$ 1.7 years as the average for 41 countries.

44.4.3 Detriment from Somatic and Genetic Risks

For an annual whole body exposure at a constant rate from age 20 for a 45 year working life at 2 mSv per year, the time-loss detriment, from illnesses and loss of life expectancy together due to all cancers, comes to 22×10^{-3} years as the average for males and females. For hereditary effects, the total time-loss detriment, from illness or incapacity and loss of life expectancy together, would be 11×10^{-3} years as an average for males and females. (This ignores the probability of lower induction rate of hereditary abnormalities per unit dose in females than in males.)

44.4.4 Deterministic Effects

For uniform external irradiation, deterministic effects would not be induced in any organ at the ICRP stochastic dose limits for effective doses. For internal exposures, individual organs may be selectively irradiated. After a detailed examination ICRP 45 concludes that the combination of stochastic and deterministic dose limits (at the earlier ICRP 26 values) will ensure that even for sensitive organs like the bone marrow or gonads the deterministic thresholds will not be exceeded. (As we have seen, ICRP 60 has given special attention to deterministic effects in the lens of the eye and the skin and prescribed annual equivalent dose limits separately for these two organs.)

44.4.5 Radiation Induction of Effects during Pregnancies

Broadly following ICRP 27, but with some modifications, ICRP 45 has taken into account 4 parameters which will influence the total harm. These are: (i) proportion of a female working population likely to be

pregnant in any one year, (ii) the risk of harm per unit dose from exposure prior to implantation, (iii) risk rates for developmental abnormalities, and (iv) risk rates due to induction of cancers during development. Table 44.4 gives the detriment due to exposure during pregnancies per year of exposure at 2 mSv per year.

44.5 COMPARISON BETWEEN RADIATION AND OTHER SOURCES OF OCCUPATIONAL RISKS

Variations in doses (and hence risks) between different sections of the nuclear industry are comparable with variations in risk observed within other industries (UNSCEAR, 1982). The highest category gets 3-4 times the mean dose for all categories in a particular group. There are also systematic trends in the variations with time of the mean annual doses; these are due to changes in work practices, workload per worker, etc. Overall, the rates with which many annual doses are decreasing are comparable with many of the rates with which risks of fatal injuries are falling in many occupations, around - 2.5% per year. However, in some types of radiation work such as in the nuclear fuel cycle (especially power reactors), there has been an increase in the average annual doses of + 1.0% per year, while transportation has shown an increase of + 3.4% per year in USA (Kumazawa *et al.*, 1984). Radon daughter concentrations in US uranium mines have been falling fairly rapidly (15% per year during 1940-1960) (Svent, 1981). Recent data from UK and USA (Hughes and Roberts, 1984; Kumazawa *et al.*, 1984) show that the range of occupational doses was 0.4-4 mSv per year (but non-coal mines in UK showed 26 mSv per year.) The mean for all occupational workers was 1.4 mSv/year in UK and 1.1 mSv/year in USA. High rates are recorded for uranium (30 mSv/year) and other hard rock (25 mSv/year) mines.

The broad picture, therefore, is that in the majority of occupations the great majority of workers is exposed to a few mSv effective dose per year. These are the occupations and workers for which the associated risk of fatal or non-fatal injuries is likely to be low. A representative estimate is a dose rate of 2 mSv/year associated with a fatal accident rate of 25×10^{-6} per year. The annual detriment in this case would be equivalent to that of a risk without radiation but with a fatal accident rate of $35\text{-}50 \times 10^{-6}$ per year, depending upon the weight attached to the occurrence of permanent disabilities relative to that of fatal accidents. The total risk lies within the range of those observed in the safer manufacturing industries. In both cases the index of harm works out to 2.5.

However, in a small number of occupations involving radiation (as in mines, staffed mainly by male workers) the dose rates may be a few tens of mSv per year; these are high injury risk occupations with a few hundred accidental deaths per million workers at risk per year. For this category the effective annual dose can be taken as 30 mSv/year and the fatal accident rate 400 x 10^{-6} per year. The estimated component of risk from radiation exposure is smaller than that from accidents, but would raise the total estimate of risk to that of occupations without radiation exposure but with an accident fatality rate of 500-600 x 10^{-6} per year, comparable with other high risk occupations in industries. The corresponding index of harm would be 30.

ICRP 45 draws attention to the limitations of the present type of approach. No simple numerical index of harm can be regarded as complete or compelling. The use of total periods of health or life lost appears to allow a rather fuller assessment of total harm than can be obtained from mortality rate alone.

ICRP 45 has an appendix on the 'Estimation of life loss detriments to the general public'. The conclusion is that, as in the case of occupational exposure, it should be easier to develop indices of harm for radiation exposures of particular populations than for populations of the public in general, just as it is more realistic to assess the risk of specified industrial groups than those of industry in general.

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Table 44.1: Fatal accident rates in UK manufacturing industries

(based on ICRP 45)

Industry	Fatality rate per million years at risk
Clothing and footwear; instrument engineering; electrical engineering	< 10
Textiles; vehicles; paper, printing and publishing; food, drink and tobacco; leather goods; mechanical engineering; timber, furniture;	10-50
Chemical and allied industries; bricks, pottery, glass, cement	50-100
Shipbuilding and marine engineering; metal manufacture; coal and petroleum products	100-150

Table 44.2: Cancer mortality rates attributed to occupational exposure to chemical agents

(based on ICRP 45)

Occupation	Form of cancer	Annual mortality (per 10^6 persons at risk)	Reference
Shoe manufacturing (process and finishing rooms)	Nasal	130	a
Printing	Lung, bronchus	200	b
Work with cutting oils	Scrotum	400	c
Wood machining	Nasal	700	d
Coal carbonizing	Lung (and bronchitis)	2,800	e
Rubber mill working	Bladder	6,500	f
Mustard gas manufacture	Bronchus	10,400	g
Cadmium	Prostate (incidence)	14,000	h
Nickel (pre-1925)	Nasal sinuses	6,600	i
	Lung	15,500	i
Beta-naphthylamine	Bladder	24,000	j

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Table 44.3: Years of life impaired or lost owing to inherited abnormalities (per genetically significant exposure of 1 person-gray)
(source: UNSCEAR (1982) quoted in ICRP 45)

Source of abnormality or disease	Average detriment per inherited abnormality (years of life)			Detriment (years of life)	
	Unimpaired	Impaired	Lost	Impaired	Lost
Spontaneous genetic	20	22	28		
Radiation genetic:					
First generation	25	24	21	0.055	0.045
Equilibrium	26	24	20	0.34	0.29

Table 44.4: Detriment due to exposure during pregnancies per year of exposure at 2 mSv per year
(based on ICRP 45)

Exposure period	Effect	Life loss detriment 10^{-4} y
Preimplantation	Non-implantation	1.7
During organogenesis	Developmental defects	5.8
Throughout pregnancy	Cancer, fatal	1.6
Cancer, curable		< 0.1
Inherited defects		0.6
	Total	9.8

CHAPTER 45

BASES FOR JUDGING THE SIGNIFICANCE OF THE EFFECTS OF RADIATION

45.1 INTRODUCTION

Annex C of ICRP 60 discusses the risk concept and ways of expressing quantities describing various aspects of a potentially hazardous situation. The main emphasis is on the probability of serious or lethal radiation effects, particularly death from cancer, and ways of indicating the severity of such effects.

The risk expression (attributable probability of death and its distribution over age after exposure) will depend on demographic data such as background cancer incidence and total mortality rates by age. Calculations have been made by ICRP 60 for a hypothetical population. In view of the large uncertainties of the primary risk coefficients, the influence of varying demographic assumptions is negligible.

45.2 THE MEANING AND EXPRESSION OF 'RISK'

'Risk' has several meanings, including the common loose meaning in everyday language, i.e. the threat of an undesirable event, including both the probability and the character of the event. In reactor safety, 'risk' usually means the mathematical expectation of the magnitude of the undesirable consequence, i.e. the product of the probability and the consequence of the event. If risk is expressed as a probability, it is dimensionless, but has some dimension if it means the mathematical expectation of consequence. ICRP uses risk as a concept rather than as a quantity. 'Risk assessment' is therefore not necessarily synonymous with 'probability assessment', but may include other aspects like the nature and severity of consequences. Preferences in risk comparison would have to be made on the basis of a multi-attribute analysis.

ICRP 60 is mainly concerned with two quantifiable risk quantities, viz. , P_i , the *probability* of each harmful effect i, (e.g. lethal cancer, curable cancer, severe hereditary disease); and W_i , the *consequence* if

the effect occurs. (It can be described in various ways, indicating the severity of the effect and its distribution in time).

The *mathematical expectation* of consequence, identical to the average consequence, is

$$\widetilde{W} = \sum_i P_i \cdot W_i$$

when averaging is relevant, a quantity which is sometimes used in the effort to express the magnitude of the 'risk' by a single measure. In the collective case (of a large population) the mathematical expectation is not far from a likely result unless the individual probability (p) of harm is very small. If the possible consequence for each individual is w = 1 case of harm, the expectation will be

$$\widetilde{W} = N\,\tilde{w} = n\,(p/w) = Np$$

Weighted for the severity of harm, it has been used by ICRP 26 under the name 'radiation health detriment'.

For the individual, the mathematical expectation $w = p.\tilde{w}$ is not an 'expected' result, because the only possible outcomes are 0 or w measures of harm. For example, $p = 10^{-6}$ may be the probability of losing, on an average, 20 years of life because of cancer. The expectation of loss of life is then 2×10^{-5} years or 10 min. However, the real loss of life is either 0 (almost certain) or about 20 years (with a very small possibility) but never 10 min.

The probability of death is the major factor in the multi-attribute concept of risk and is given major importance by ICRP 60. Other attributes such as illness, risk to fetus, economic loss, anxiety, and other societal impacts are not fully and precisely known and simplified approaches have been made to take them into account. Hereditary risk is fairly well understood and is given some detailed attention.

45.3 CONVENTIONS ON ACCEPTABLE RISKS

All human activities carry some risks. Some activities are accepted by most persons, even though risks have not been reduced 'as far as reasonably achievable'. The corresponding risks, e.g. traffic risks, are not necessarily acceptable. We accept certain levels of risk in order to enjoy the benefits of a modern society, provided that the risks are not unnecessary or easily avoided.

What is the upper limit of risk to an individual which would not be acceptable even if it could not reasonably be further reduced? A study by the Royal Society of UK (1983) concluded that a continuing annual occupational probability of death of 1 in 100 would be unacceptable. But for a 1 in 1000 risk, the situation was less clear, and could not probably be called totally unacceptable, provided that the individual at risk knew the situation, its benefits *vs.* risks and understood that everything reasonable had been done to reduce the 'risk'.

A retrospective study has been made (Travis *et al.*, 1987 a,b) of how cancer risk estimates for the public had been made by US federal agencies in the regulation of 132 chemical carcinogens. Among the risk measures examined were the individual attributable lifetime probability of death. The conclusion was that all substances with this probability above 4×10^{-3} appeared to have been regulated regardless of cost. At lower individual probabilities, substances with regulatory costs above $ 2 million per life saved were not regulated.

ICRP 26 dose limits implied that an annual occupational death probability of 10^{-3} to the most exposed individual and 10^{-5} to a member of the public would be at the border of being unacceptable.

45.4 THE RISK OF DEATH

The total lifetime probability of death, which is 100%, cannot be increased by any additional risk, which can only change the distribution of the probable causes of death. Any increment in risk from a new source is an increment to the death probability rate at any given age, provided that the person is alive at that age (i.e. a conditional probability rate).

Let $G_0(u)$ be the 'total conditional death probability rate' at age u from all natural causes for an average person (i.e. given that the individual is alive at age u). $G_0(u)$ is usually described by the Gompertz-Makeham expression

$$G_0(u) = Ae^{B.u} + C$$

where A, B and C are parameters that can be derived from demographic tables. It should be remembered that there is a distinction between the probability in a year (which can never exceed 100%), and the probability per year, i.e., the probability rate (which will exceed 100% at very high ages). Figure 45.1 illustrates a typical example of this curve.

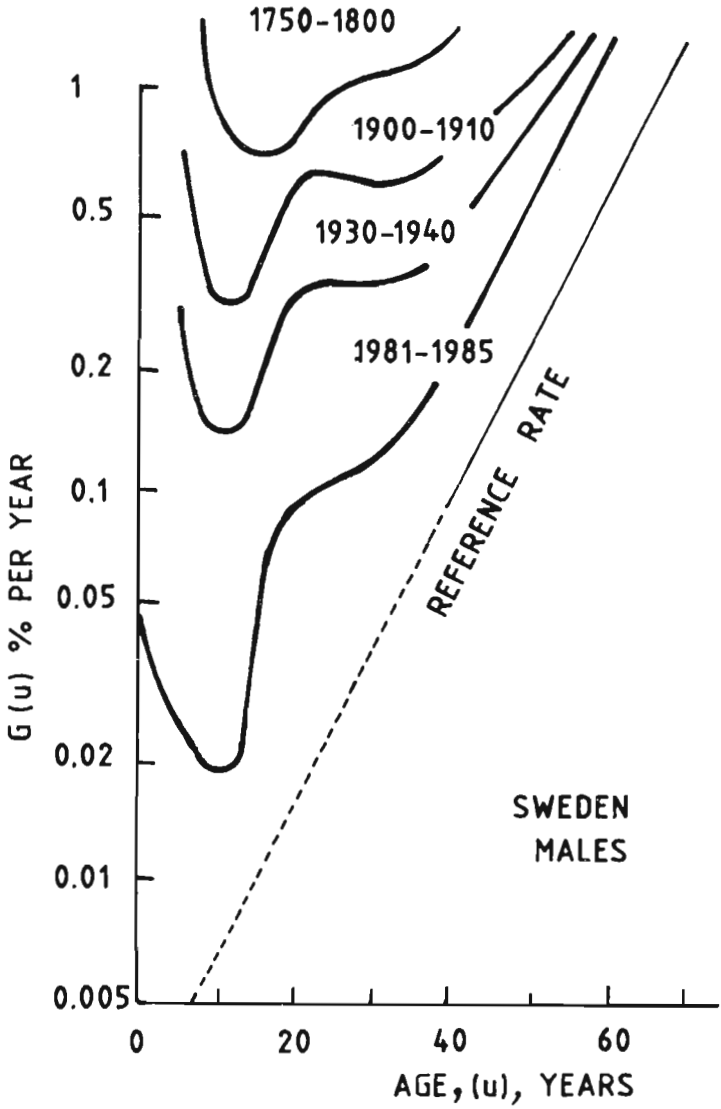

Figure 45.1. Gompertz-Makeham curves (the age-specific mortality rate) for Swedish males from 1750-1800 to 1981-1985 (from ICRP 60).

45.4.1 The Conditional Death Probability Rate (*dp/du*)

To the background probability rate discussed above is added a radiation source-related incremental 'conditional death probability rate', *dp/du*

(caused by a given source or practice over the rest of the life of the exposed individual), i.e.

$$G(u) = G_0(u) + dp/du$$

It can be calculated from the postulated dose-response curve, once the dose rate is known as a function of age. This rate which is conditional (because it will only be expressed if the individual is alive at age u for which it is defined) should be kept low for protection purposes.

The ratio $(dp/du)/G_0(u)$ is the relative value of the conditional death probability rate. The aim of protection is to keep this value $<<1$ for all ages of concern. In practical situations the maximum value of this ratio occurs at ages below 60 years for both the additive and multiplicative projection models.

45.4.2 The Unconditional Death Probability Rate (*dr/du*)

The conditional death probability rate dp/du due to a given risk source cannot be used for calculation of the total attributable lifetime probability of death, because the expression has no well-defined integration limit. The 'unconditional incremental death probability rate' (also called 'expressed' or *a priori* death probability rate) dr/du, can be calculated from the conditional probability rate dp/du taking account of the probability of reaching age u by considering the likelihood of dying from other causes as well as from radiation. It is the product of the conditional incremental probability rate dp/du and the survival probability modified by the incremental risk $S(T,u)$. For a single exposure, T is the age at time of exposure. For prolonged exposure, T has been taken by ICRP 60 as the age at the onset of the exposure period.

The *Attributable Lifetime Probability of Death* (R) is given by

$$R = \int_T^\infty (dr/du) \, . \, du$$

45.4.3 The Probability Density of the Age at Death

The attributable lifetime probability of death, R (being the probability of dying of cancer due to one particular cause rather than dying from another), gives no indication of when death will occur. For this purpose, we define the 'probability density of the age of death', which can be derived from the variation of dr/du with age, normalized so that the area

under the curve is not unity but the attributable lifetime probability of death.

45.4.4 Mean Loss of Life if Radiation Death Occurs (Y)

This can be calculated from the value of dr/du at any age and the normal remaining life expectancy as a function of age. The pair of values R and Y is the minimum information needed to express the incremental risk.

45.4.5 Reduction of Lifespan Expectancy (ΔL)

ΔL is simply equal to $R.Y$. If $R << 1$, this expectation value is not very informative in the individual case; if R is small, the most likely loss of lifetime is zero, and there is the small probability R of losing the life-period Y. The expectation value of ΔL will then never occur. In the collective case, however, the situation is different. In a cohort of $N > 1/R$ individuals, a lifetime expectation of $N.\ \Delta L$ (e.g. in man-years) is a very likely outcome.

45.4.6 Probabilistic 'Aging'

The shift in the age-specific death probability rate may be described as equivalent to 'aging' in the sense that the increased probability rate equals that at a higher age. This 'aging' with regard to death probability rate is of the same order of magnitude as the expectation of loss of life-time, but varies with age also because dp/du varies with age.

45.5 THE BACKGROUND CONDITIONAL DEATH PROBABILITY RATE $G_0(U)$

The lowest conditional annual death probability rate from all causes usually occurs around the age of 10 years (1-2 per 10,000 in industrialized countries and over 1 in 1000 in developing countries). In many countries a small peak around the age 20 years apparently reveals juvenile risk taking. At ages above 30-40 years, the death probability rate doubles every 7 years (i.e. increases by 10% per year) and is of the order of 1 in 100 at age 60 years. In most countries the age-specific mortality rate has decreased substantially over the last century as a result of advances in medicine and public health.

As mentioned earlier, the control of risk from a new source is a problem beyond the realm of radiation protection alone. ICRP 60 prefers a multi-attribute approach to the choice of dose limits. For this purpose it is necessary to examine the overall risk picture that would be the consequence of various options of dose limits.

45.6 PRIMARY RISK COEFFICIENTS: INCREMENT OF DEATH PROBABILITY RATE AFTER A SINGLE EXPOSURE AND FOR PROLONGED EXPOSURES

An increased cancer death probability (dp/du) will only occur after the latent period; this will be at a later age, when the risk of death from other causes is also higher. For a single exposure, in the additive model,

$$dp/du = r_a.D,$$

where r_a is a constant and D the dose.

In the multiplicative model,

$$dp/du = r_m.D.B(u)$$

where r_m is a constant and $B(u)$ the background rate of cancer deaths. We see that the calculation of dp/du involves a knowledge of the background cancer incidence as a function of age. These data can reasonably well be approximated by the following expression:

$$B(u) = a.u^b + c.$$

The values of a, b and c quoted in ICRP 60 are in the range of a = 3-4 x 10^{-10}, b = 3-5 and $c = 15 \times 10^{-6}$.

Figure 45.2 depicts the variation of dp/du with age for the additive and multiplicative projection models. The excess cancer incidence is taken to begin after a latent period in both cases. For the additive model the excess incidence is taken to last over a 'plateau' of time.

ICRP 60 gives a number of tables and figures concerned with the calculations of the various parameters for both the additive and multiplicative models.

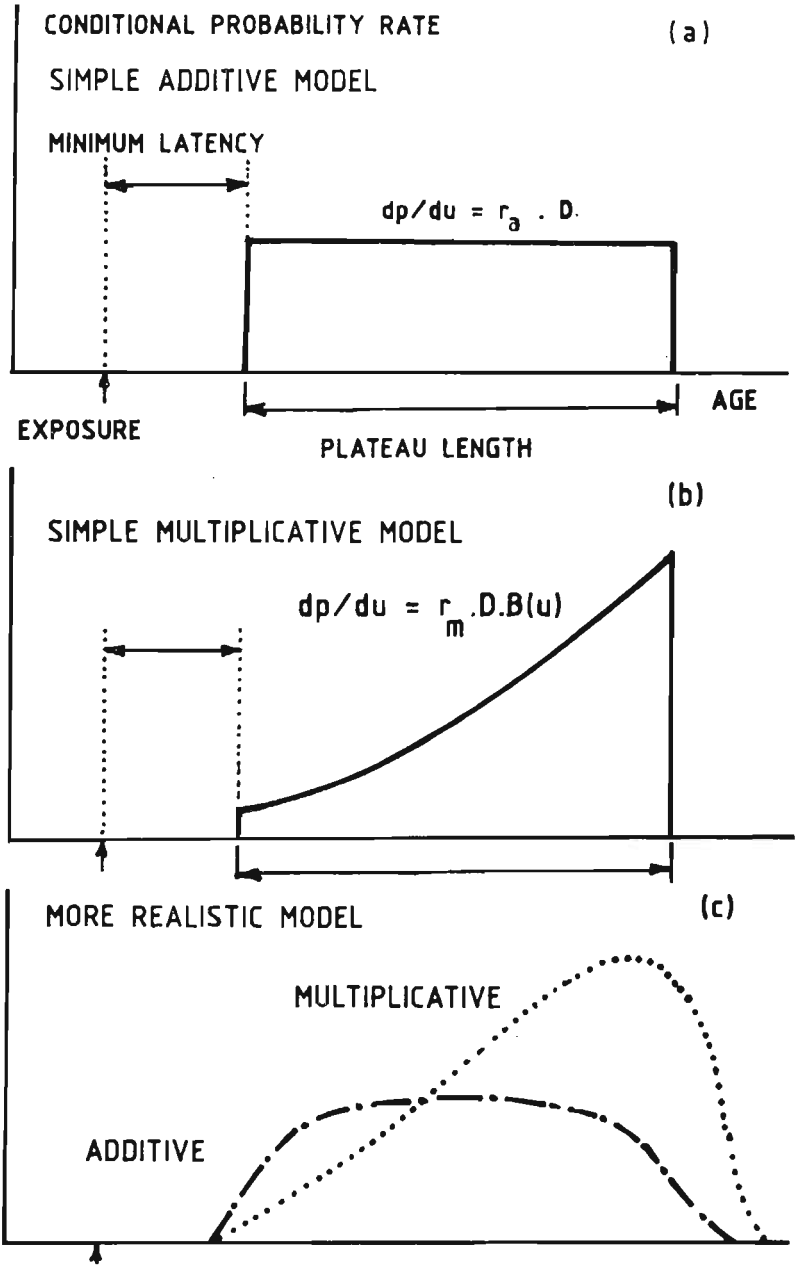

Figure 45.2. **Variation of the conditional death probability rate dp/du with age for the additive and multiplicative risk projection models (from ICRP 60).**

The tables include:

(a) Primary risk coefficients for annual cancer death for leukaemia and other cancers for different ages at exposure for males and females (UNSCEAR 1988 figures);

(b) Conditional death probability rate, *dp/du*, total and attributable to various annual doses (1-5 mSv) from birth over lifetime for males and females, along with $G_0(u)$ values at each age for the Swedish population;

(c) Same computations as in (b) repeated for various annual doses (10-50 mSv) from age 18 to 65; and

(d) and (e) Calculations similar to those in (b) and (c) for the unconditional death probability, *dr/du*.

The figures include:

(a) and (b) Variation with age of *dp/du* and *dr/du* after a single dose at age 5 and 35 respectively for males;

(c) Attributable lifetime risk for a single dose at various ages at the time of exposure for males and females;

(d) Variation with age (extreme values) of the specific mortality rate (approximately the conditional death probability) for 18 industrialized countries for males and females;

(e) Changes in the total conditional death probability rate as a function of age for the Swedish population after an exposure of 5 mSv/y from birth over lifetime; and

(f) Same as in (e) for exposure of 50 mSv/y from age 18 to 65 years.

Figures 45.3 to 45.6 have been reproduced from ICRP 60 for illustrative purposes. The main conclusions are:

(a) The mean attributable lifetime probability of death per unit (single) dose over all ages in a normal population is 2% per Sv as average for both sexes (1.8% for males, 2.1% for females) on the additive model, and 5.2% per Sv (4.8% for males, 5.6% for females) in the multiplicative model (DDREF = 2);

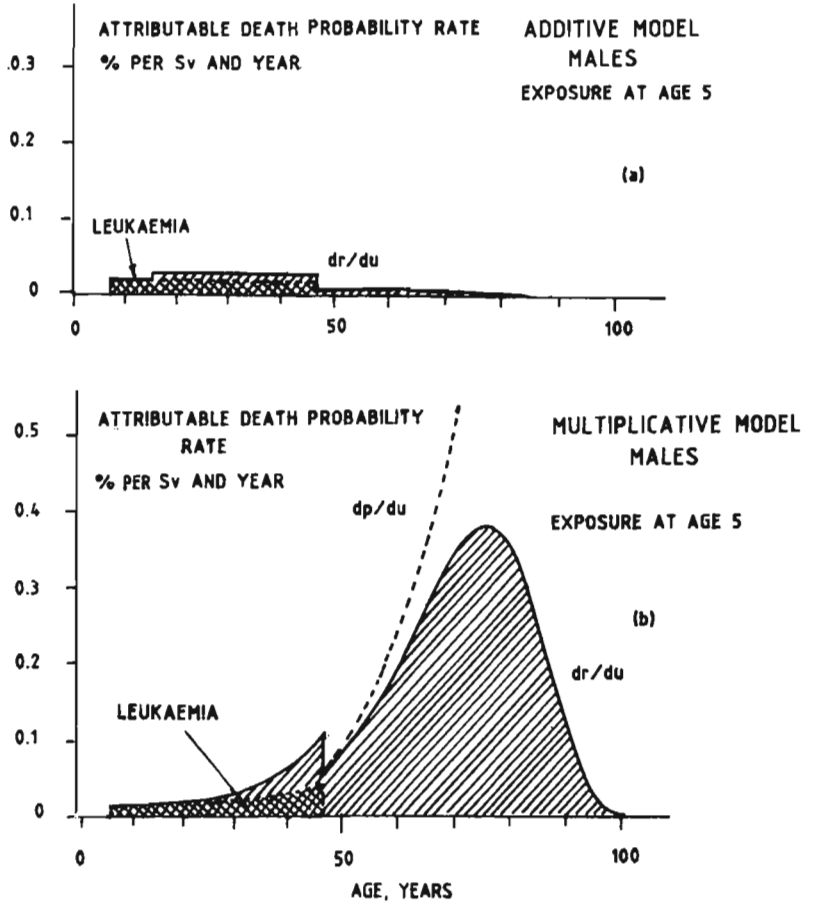

Figure 45.3. Variation with age of the attributable death probability rates *dp/du* (conditional) and *dr/du* (unconditional) after a single small dose at age 5 years (from ICRP 60).

(b) Under the same conditions of exposure, the values of *dp/du* become higher for the multiplicative model than for the additive model for ages above 50 in the case of exposures from birth, and for ages above 60 years at exposure from 18 to 65 years;

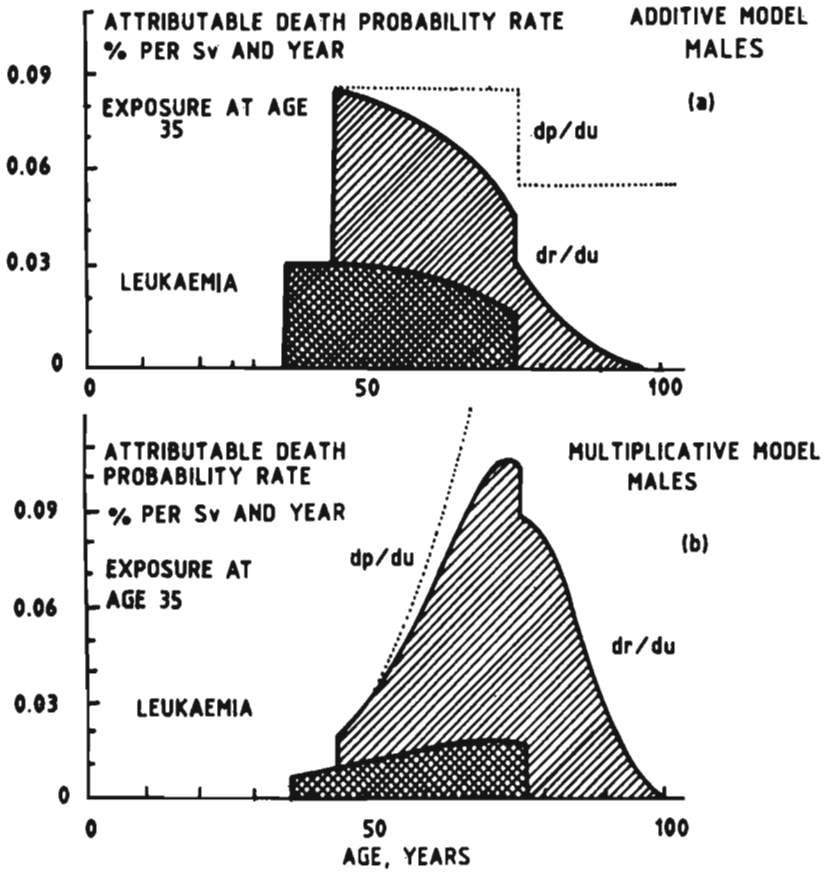

Figure 45.4. Variation with age of the attributable death probability rates after a single small dose at age 35 years (from ICRP 60).

(c) Since the total background risk increases with age somewhat more rapidly than the cancer background, the relative increments of the death probability rate in relation to the background rate do not increase at high ages. For exposure at 1 mSv/y from age zero, the maximum increases are 1.4% and 0.9% in the additive and multiplicative models respectively. For 50 mSv/y for exposures during ages 18-65, the corresponding figures are 40% and 17% respectively; and

(d) The largest increases in relative risk occur earlier in life for the additive model.

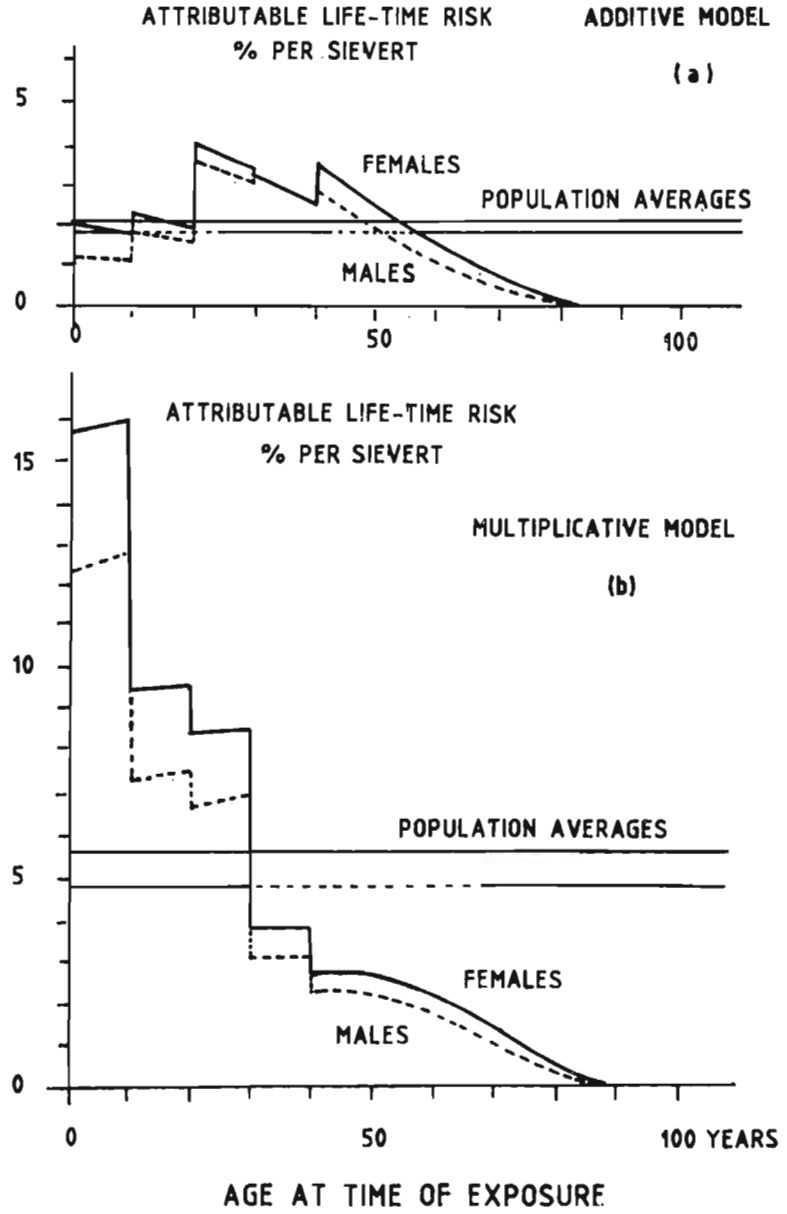

Figure 45.5. The attributable lifetime risk from a single small dose at various ages at the time of exposure (from ICRP 60).

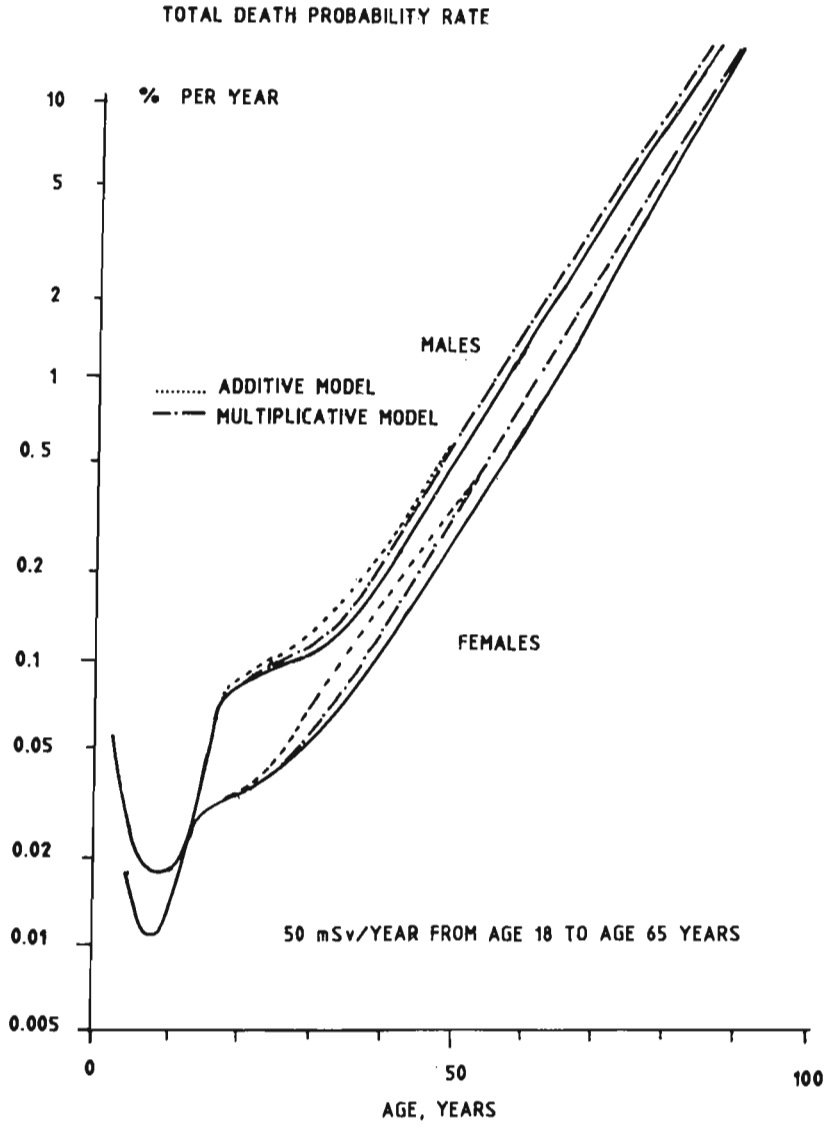

Figure 45.6. **Change in the total conditional death probability rate (reference: the Swedish population, 1986) after an exposure of 50 mSv per year from ages 18 to 65 years (from ICRP 60).**

45.7 SUMMARY OF THE RISK DESCRIPTION

Figure 45.7 depicts the unconditional death probability rate (the attributable probability density of age of death, normalized for lifetime risk) for females for two exposure situations: (a) exposure from birth over lifetime; and (b) exposure from age 18 to 65 years. The curves can also be seen to represent the normalized probability density of the age of death from radiation-induced cancer. The area under the curve represents the lifetime probability of dying from radiation-induced cancer. The values are given in Table 45.1.

It is seen that there are differences in the time distribution of the radiation-induced cancer deaths. With the multiplicative model, the deaths, on the average, will occur significantly later.

Figure 45.8 supplements Fig. 45.7, in that the unconditional attributable death probability rates are compared with the total conditional death probability rates related to age 18 for females.

Tables C-4a and C-4b of Annex C of ICRP 60 list values for ten parameters which may be used to express the radiation risk. Numerical values for these parameters have been tabulated for two important types of exposure, viz. (i) exposure from age zero over lifetime for annual doses in the range 1-5 mSv, and (ii) exposure from age 18 to age 65 years for annual doses in the range 3-50 mSv. The values are for whole body exposure and averaged for both sexes with a DDREF value of 2. Computations have been made for both the additive and multiplicative projection models. An examination of these tables shows that for 7 out of the 10 parameters the numerical values of the quantity to describe the risk is proportional to the dose, while for the other 3 these values are independent of the dose. Hence these two types have been separately given in Tables 45.1A and 45.1B. The numerical values are given for an annual dose of 1 mSv (the dose limit for exposure of members of the public) for exposure from age zero over lifetime, and an annual dose of 20 mSv (dose limit for occupational exposure) for exposure from age 18 to age 65 years. The corresponding values for any other annual dose can be worked out by simple proportion.

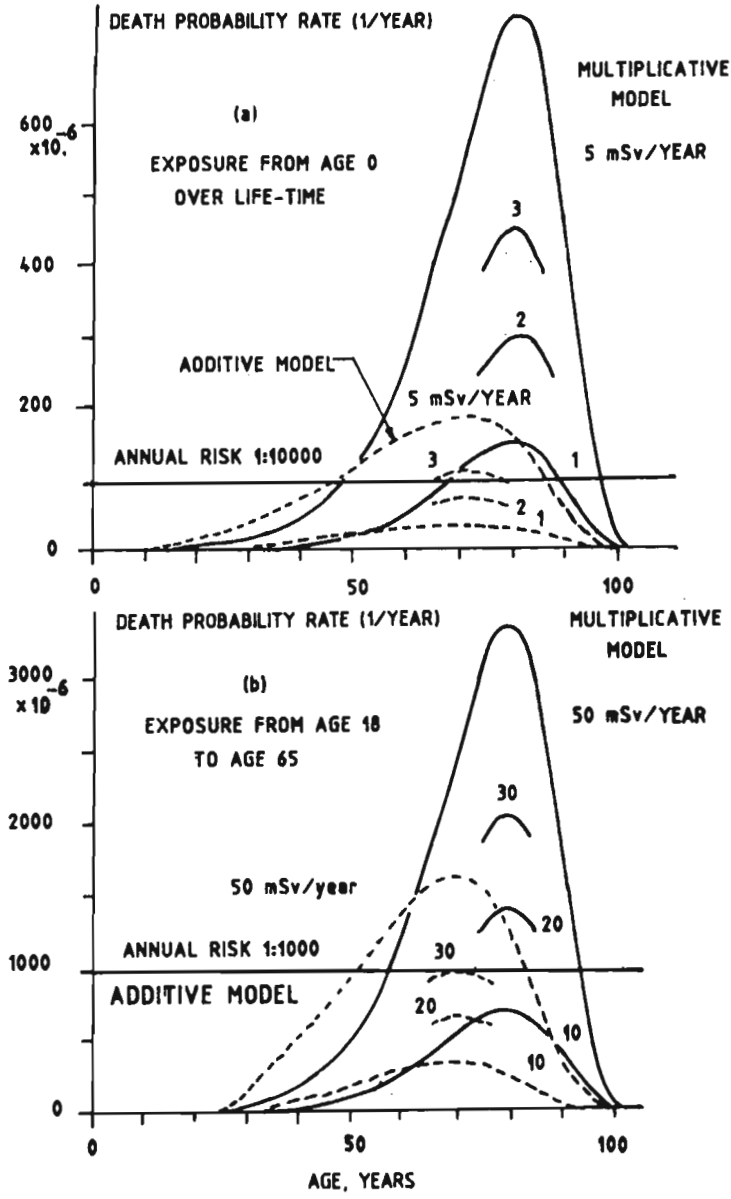

Figure 45.7. **The unconditional death probability rate (the attributable probability density of the age of death normalized for lifetime risk) for two exposure situations: (a) exposure from birth over lifetime, and (b) exposure from ages 18 to 65 years (from ICRP 60).**

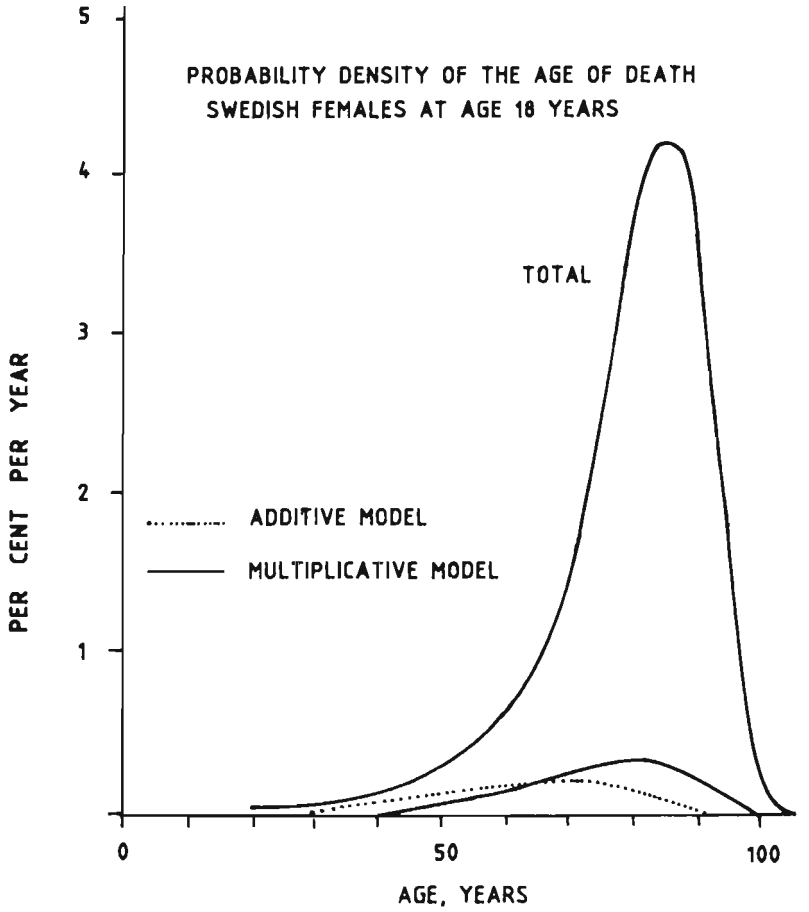

Figure 45.8 **Comparison between the probability density of the age of death from all causes and the probability density of the age of attributable radiation death (normalized to make the attributable lifetime risk of death directly comparable with 100% total lifetime risk) for females exposed at 50 mSv per year from ages 18 to 65 years (from ICRP 60).**

The most informative presentation is by the combination of the attributable lifetime probability of death and the mean loss of lifetime in the case of death from radiation-induced cancer. In the collective case, for a population exceeding the inverse value of the per caput attributable lifetime probability of death, the detriment represented by the expected number of cancer deaths or by the collective loss of man-years is informative, although not relevant for individual risk limitation. The comprehensive

data given in Tables C-4a and C-4b, Annex C, ICRP 60 have been used, together with other information, for the multi-attribute approach to selection of dose limits in the main text of ICRP 60.

45.8 A MULTI-ATTRIBUTE APPROACH TO THE SELECTION OF DOSE LIMITS

For deterministic effects it is easy to select dose limits. But stochastic effects occur at doses even below the thresholds for deterministic effects. The selection of dose limits is only partly a scientific decision and value judgements on levels of acceptable risk also play a part.

From what has been discussed above, it is clear that radiation 'risk' can be presented in a number of ways. If only the conditional death probability is shown, for example, by the shift in the Gompertz curve (Fig. 45.6), even high annual doses would not seem to change the overall risk situation significantly, and the change would be small in comparison with sex or population differences. However, the same risk expressed as reduction of life expectancy may amount to several years and look less acceptable. How should the probability increment be expressed, in absolute (say, dp/du) terms, or relative $[(dp/du)/G_0(u)]$ terms?

ICRP 60 has drawn attention to the rationale and methodologies adopted by ICRP 26 (1977) in arriving at dose limits for occupational workers and members of the public.

Some comparisons between ICRP 26 and ICRP 60 have been attempted. Table 45.2 (based on Table C-5, Annex C, ICRP 60) shows the conditional incremental cancer death probability rate, averaged over the sexes, at various ages and annual doses for workers exposed from 18 to 65 years and for members of the public exposed from birth, using the multiplicative model. The original ICRP 60 Table C-5 gives the values of the parameters for various annual doses (10-50 mSv for workers and 0.5-5 mSv for the public). As explained in the context of Table 45.1, the annual conditional cancer death probability rate is proportional to the dose. Hence values are given in Table 45.2 only for 50 and 20 mSv annual dose for workers (corresponding to the ICRP 26 and ICRP 60 dose limits) and 1 mSv for the public. It may be noted that ICRP 26 used an annual risk of 1.25×10^{-2} Sv^{-1} for all ages (although a latent period was recognized, it was not taken into account). Risk estimates based on the ICRP 26 risk coefficient are also given in Table 45.2.

The information given in Table 45.2 is not sufficient basis for judging the appropriateness of a new dose limit. As discussed earlier, the nominal probability coefficients are composed of 3 components, viz. fatality coefficient, weighted coefficient for curable cancer, and weighted coefficient for hereditary effects. In ICRP 26 only hereditary effects in the first two generations were taken into account, and curable cancer was not counted in the risk coefficient. (The 1980 Brighton ICRP statement gave a detriment of 10% of the fatality detriment for the curable cancers.) Table 45.3 (based on Table C-6, Annex C, ICRP 60) gives the total detriment values and their components. As in Table 45.2, the values of the various parameters of the detriment are proportional to the dose and hence only a portion of the original ICRP 60 table is reproduced in Table 45.3. With the new estimates, the weighted coefficient for curable cancer is 20% of the fatality coefficient for both workers and the public. The weighted coefficient for hereditary effects is 1.33×10^{-2} Sv^{-1} for the public and 0.80×10^{-2} Sv^{-1} for workers (based on 47 years for the workers and 75 years for the public). The nominal cancer fatality coefficient is 5×10^{-2} Sv^{-1} for the public and 4×10^{-2} Sv^{-1} for workers.

In ICRP 26 it was assumed that a radiation-induced cancer death would, on the average, mean a loss of life of 10-15 years. The present values are 20 years on the additive model and 13-15 years on the multiplicative model.

In ICRP 26 it was assumed that an occupational fatality probability of 10^{-3} might be taken as a reference risk for the dose limit. This was made on the assumption that, in 'safe' non-radiation occupations, the average annual fatality rate was 100 per million workers and that subgroups with high risks might run a risk 10 times the average. It may be seen from Table 45.2 that an annual cancer death probability of 10^{-3} is not exceeded before 65 years for annual doses below 20 mSv, nor before age 75 years for annual doses below 1 mSv. For 50 mSv/year (ICRP 26 dose limit for occupational workers), it is exceeded after 55 years.

There are risks not related to radiation in radiation work, for example, in mines and factories. In all industries there are occupational diseases which may cause untimely death. There may also be differences in the mean loss of life when death occurs; for an accidental death in industries the mean loss of life is as high as 35 years.

The choice of dose limits for members of the public is even more difficult because of the many sources of risk in addition to radiation, to which the public is exposed. With an annual dose limit of 1 mSv, using

the multiplicative model and DDREF = 2, the attributable lifetime fatality probability is 4×10^{-3}. (This limit of 1 mSv is to be applied to the total dose from all regulated practices.)

Natural background radiation must be assumed to cause dose-related risks. The relative magnitude of any radiation risk in relation to the risk from background radiation is described by the ratio of annual effective doses. The fact that a man-made procedure involving radiation causes doses comparable to background does not necessarily imply that the practice is justified; but it does imply that the radiation risk situation of the exposed individual is not significantly changed by the new practice.

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Table 45.1A: Risk evaluation: summary table
(based on ICRP 60)

Quantity to describe 'risk'	Exposure from age zero over lifetime (annual dose 1 mSv)	Exposure from from age 18 to age 65 years (annual dose 20 mSv)
Attributable lifetime probability of cancer death (%)	0.15 0.40	2.31 3.57
Loss of life expectancy (man-years per caput)	(At age 0) 0.03 0.05	(At age 18) 0.46 0.46
Mean annually committed probability of attributable cancer deaths (per million)	(0-70 years) 21 57	(18-65 years) 490 760
Annual extra probability of cancer death at age 70 years (per million)	44 135	780 1300
Maximum relative death probability rate (%)	1.4 0.9	17 7
Maximum risk equivalent aging (years)	0.3 0.2	1.6 0.5
Attributable cancer deaths per million and year in a population at 10% of dose level	2 5	Not given Not given

Average for both sexes; DDREF = 2. Upper numbers relate to additive model and lower numbers to multiplicative model.

Table 45.1B: Risk evaluation: summary table
(based on ICRP 60)

Quantity to describe 'risk'	Exposure from age zero over lifetime	Exposure from age 18 - 65 years
Loss of lifetime if cancer death (years)	22.6 13.4	19.8 12.7
Most probable age at attributable death (years)	68 79	68 78
Age at maximum relative rate (years)	34 42	39 57

Average for both sexes; DDREF = 2. Upper numbers relate to additive model and lower numbers to multiplicative model. These parameters are independent of the actual value of the exposure.

Table 45.2: Comparison of ICRP 60 (multiplicative model; DDREF = 2) and ICRP 26 risk estimates for the annual conditional cancer death probability (per million)
(based on ICRP 60)

Annual dose (mSv)	Age at risk (years)						
	30	40	50	60	65	70	75
Workers							
50	42	190	570	1500	2200	3200	4700
20	17	75	230	590	890	1300	1900
50*	625	625	625	625	625	625	625
Public							
1	1	4	12	30	45	65	95
1*	12	12	12	12	12	12	12

* ICRP 26 values

Table 45.3: Detriment as assessed in ICRP 60 on the basis of multiplicative model at the ICRP 60 and ICRP 26 dose limits compared with the total risk assessed in ICRP 26 at old dose limits
(based on ICRP 60)

Annual effective dose (mSv)	Fatal cancer	Weighted curable cancer*	Weighted heredi-tary*	Aggregate detriment
Workers (exposure from age 18 to age 65 years)				
50	8.6	1.72	1.72	12.0
20	3.6	0.72	0.72	5.0
50 (ICRP 26)	2.9	-	-	-
Public (exposure from birth over lifetime)				
1	0.4	0.08	0.11	0.59
1 (ICRP 26)	0.1	-	-	-

* Weighted for severity and length of life lost.

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